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A TREATISE ON

DIAGNOSTIC METHODS

OF EXAMINATION

BY

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EDITORS' PREFACE.

THE first edition of Professor Sahli's treatise, "Clinical Methods of Investigation," was published in 1894, and was immediately recognized as a master-work in this field. A second edition appeared in 1899, a third in 1902, and a fourth during the present year (1905). The present translation of the last edition was undertaken to render easily available to English-speaking students of medicine a work too little known in this country and England.

The distinguished Swiss Professor has chosen the title "Lehrbuch der klinischen Untersuchungs-Methoden"—a title which inadequately expresses the great scope of the work. Not only are all methods of examination for the purpose of diagnosis exhaustively considered, but the explanation of clinical phenomena is given and discussed from physiological as well as pathological points of view, with a thoroughness which has not been attempted in any clinical work which has yet appeared. Its great value will at once be recognized.

It has seemed wise to publish the translation practically as the original came from the pen of its author. A few notes have been added, especially where methods are described which differ from those commonly employed by American and English clinicians. A brief review of the investigations of American and English observers on the value of the clinical estimation of blood-pressure, with the description of some newly devised or modified instruments for this purpose, has been written by Dr. Theodore C. Janeway.

We are indebted to Dr. C. P. Flint, of this city, for a considerable part of the labor in translation, also to Drs. Lewis F. Frizzell and H. D. Meeker for assistance in revising the manuscript. For various notes and assistance in special chapters of the book, we are indebted to the following-named gentlemen, whose respective initials will be found at the end of the notes they have added: Prof. Joseph Collins, Professor of Nervous and Mental Diseases at the Post-Graduate Medical School; Dr. Charles Norris, Instructor in Bacteriology and Hygiene at Columbia University Medical School; Dr. H. C. Jackson, Assistant Professor of Physiological Chemistry at the University and Bellevue Medical School; Dr. Arnold Knapp, Professor of Ophthalmology at Columbia University Medical School; and Dr. W. Sohier Bryant, Instructor in Diseases of the Ear at the Post-Graduate Medical School.

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PREFACE TO THE FOURTH EDITION.

An idea of the entire contents of the present edition is best obtained by consulting the systematic table of contents on page 13.

The bringing out of new editions is very much like keeping a large building in good condition—no sooner have a certain number of repairs been completed so that the agent congratulates himself on getting a little rest, than he is obliged to begin again. After so many changes had been found necessary in former editions, it was my intention to bring out the present (fourth) edition without much alteration or addition; but this intention I was forced to abandon. As in preceding editions, I have again endeavored to lay equal stress on all the various branches of internal medicine; for I hold that all specialization in internal medicine, aside from those branches which must necessarily remain separate on account of the special surgical technic which they demand, is unjustifiable and destructive of that symmetry which should be an essential feature of medical education.

Aside from numerous minor changes in subject-matter and arrangement, I may mention the most important additions by which I have endeavored to increase the value of the book.

The section on sphygmography contains a full account of the changes made by Jaquet as well as myself in the older Jaquet sphygmograph. These modifications correct the excessive vibration of the instrument and, I may venture to say, render it in every way satisfactory. In the chapter on sphygmomanometry a new clinical pocket-manometer of my own is described. In the section on the venous pulse, mention is made of Volhard's recent work and his method of determining the phases of the venous pulse. Numerous other changes and additions in the chapter on physical diagnosis I shall pass over without mentioning. Much has been added also to the paragraphs relating to the examination of the digestive apparatus. I may mention, for example, Reissner's improvement of Lütke-Martius's method of determining the hydrochloric acid in the gastric juice; Reissner's method of determining the chlorids for the early diagnosis of cancer of the stomach; an improvement on the Hehner-Malay method of determining acidity; an improvement of Mett's digestion-test by Nirenstein and Schiff; a refutation of the objections to my method of butyrometric examination of the stomach, with a few improvements of the method; certain newer methods for determining the "crude motility" of the stomach and for diagnosing pyloric stenosis; a description of a special platform, which is both movable and capable of being heated, and is designed for examining feces for amebæ, etc.; and, finally, a table containing the rarer forms of

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tapeworm occurring in man. Some of the new features in the chapter on the examination of the urine are: the method of separating the urine; Seliwanow's reaction for levulose; Bial's method of testing for pentoses; a discussion of the controversy in regard to the nature of the mucin-like body in the urine; the quantitative determination of urochrome after Klemperer; the determination of urea after Schöndorff; Hopkin, Folin, and Shäffer's method of determining uric acid; detailed directions for titrating the urine with phosphates; and the quantitative determination of indican in the urine. Kjeldahl's method is discussed more in detail than in the preceding editions. Magnus-Levy's investigations in regard to oxybutyric acid in the urine are also mentioned at greater length. The important recently published investigations by Moritz in regard to titration of fluids containing phosphates, particularly urine and gastric juice, are described in an appendix, which also contains a discussion of the question whether the recent attempt to substitute the conception of concentration of the ions of hydrogen for that of the titrimetric acidity is justifiable. Osmotic pressure and the cryoscopy of the urine are discussed at length. In connection with urinary sediments, Liebermann and Posner's methods of staining urinary sediments are described.

In the chapter devoted to the bacteriology of the sputum the micrococcus catarrhalis, as well as the bacillus of plague, anthrax, and glanders, is described. Wanner's investigations in regard to the amount of albumin contained in the sputum are mentioned. The chapter on the examination of the blood has been thoroughly revised so as to harmonize with the results of the most recent investigations. Some of the things are new and mentioned for the first time, such as my hemometer; Breuer and Türk's cell for counting leukocytes; Jenner's method of staining; the volume-quotient of the red blood-cells after Capps; a discussion of rouleau-formation and the net of fibrin in recent blood preparations; erythremia or polycythemia; and, finally, the methods of determining the osmotic pressure and the viscosity of the blood. In the section dealing with the behavior of the blood in infectious diseases, mumps, varicella, and whooping-cough have been added to the list. The chapter on leukemia has been revised in accordance with the more recent books on the subject, and acute myelemia is also discussed in addition to acute lymphatic leukemia. The relation of lymphatic leukemia and the pseudoleukemias to the remaining forms of lymphomatosis is discussed on the basis of Türk's system of lymphomatosis. A new table has been added to the section on hematology. In connection with methods of examination of the upper air passages there will be found a short account of bronchoscopy. Under the head of examination of the esophagus are mentioned Mikulicz's experiments in determining the pressure in the esophagus; Rumpel's experiment; and other matters relating to the diagnosis of diffuse dilatations of the esophagus, a subject which has been much discussed in recent literature. In the chapter on exploratory puncture, the controversy in regard to the nature of the mucin-like body found in the fluid obtained by puncture; the significance

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of the chylous nature of that body; cytodiagnosis (so called); and the question of the osmotic pressure of the puncture fluid are discussed. Among the additions in the section devoted to the examination of the nervous system may be mentioned: the expansion of the paragraph on associated movements; the adoption of an explanation of my own for the intention tremor; the disturbance of speech and the forced laugh in multiple sclerosis; the peculiar electrical reactions described by Placzek in certain cases of chronic peripheral palsy of long standing; the muscular atrophy seen in affections of the central motor neuron; a new scheme of pupillary reaction superseding Bechterew's, which appeared in the last edition and which has shown itself to be inadequate; Schirmer's studies in testing the pupillary reaction; Fragstein and Kempner's pupil-tester for determining hemiopic pupillary rigidity; v. Bezold's modification of Rinné's experiment, with reference to the Zimmermann-Helmholtz controversy in regard to the significations of the auditory ossicles; Weber's and Schwabach's experiments; a chapter on cerebral disturbances of sensation, with special reference to the distinction between cerebral disturbances with an anatomic basis and hysterical hemianesthesia; sensation in cattle; and, finally, a chapter on vertigo. The section on aphasia has been remodeled on the basis of a different theory in regard to the speech tract from that adopted in former editions. It is suggested that the expressions "transcortical," "cortical," and "subcortical," which are really incorrect, be replaced by the terms "transcentral," "central," and "subcentral." There has also been added a chapter on certain disturbances allied to aphasia, such as asymbolia, apraxia, amusia, mental deafness, and mental blindness; also a chapter on spinal hemiplegia. The description of the functions of the bladder and rectum takes account also of the doctrine of sympathetic centres for the bladder and rectum and ejaculation centres, as described in the more recent work of L. R. Müller. The neurological section, as well as other portions of the book, has been enriched by a number of new illustrations. Finally, the appendix on the modern methods of analyzing arhythmia of the pulse after Engelmann, Wenkebach, and Hering also contains a number of changes and additions.

In spite of these very considerable additions the size of the book has not been greatly increased, as some of the older methods, which were mentioned in former editions but have not stood the test of time, have been eliminated. I received suggestions from various quarters to include *x*-ray examination; but I could not bring myself to do so, because I wish to confine myself to those methods the technic of which I am myself sufficiently familiar with to give personal advice based on my own experience; and while I do make use of *x*-ray examination for the diagnosis of internal diseases, I do not attend to the technic myself, and, therefore, do not consider myself a competent radiologist. Another reason for excluding *x*-ray technic in a work like the present, which is intended for the general education of the physician, is that *x*-ray examinations can only be made by a limited number of practitioners, who, in order to do so, must go through a special training. The same may be

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said of cystoscopy. It is true that I practise this method myself, but I have not had sufficient practice and experience to justify the effort to teach it to others.

Finally, I cannot refrain from repeating in this place that this work is not a mere compilation. On the contrary, in almost every subject what I have written is based on my experience; many of the theories advanced and the cases reported are my own, and if they have not been utilized by me for original contributions to the medical journals, it is partly on account of lack of time and partly a personal aversion to hack-writing in medicine. I mention this matter because the present work practically never enjoys the honor of being quoted. I believe an explanation of this is to be found in the tendency which prevails at the present time, and which does not tend to further the symmetrical progress of our science, to make the journals and periodicals the sole depositaries of medical literature, and because it is usually presumed that text-books, aside from the systems which are now so popular and have so many names on their title-pages, are mere compilations and do not contain anything new. It is assumed that if an author had anything important and new to say he would have brought it out on the "bourse" of periodical literature. Personally, I take a different view, and the correctness of my remarks is shown, among other things, by the fact that a well-known author not so very long ago (in the *Deutsche medicinische Wochenschrift*, Volume 1903, page 716), in speaking of certain sources of error in the auscultation of the lungs, states that among current text-books the present is the only one that devotes a short chapter to this subject, based probably on an address by Treupel delivered in the Verein der Freiburger Arzte. Thus, without taking the trouble to investigate, the author takes it for granted that a text-book cannot contain any new ideas and observations that have not been published elsewhere. If he had tried to inform himself on the point, he would have found that the chapter in my text-book to which he refers appeared almost in exactly the same form in the first edition in 1894—*i. e.*, four years earlier than the time when the address to which he refers as being my source was printed.

HERMANN SAHLI.

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INTRODUCTION.

HISTORY AND OBJECTIVE EXAMINATION.

To diagnose correctly any given case of sickness, it is customary to question the patient or his friends upon his subjective and objective symptoms of illness and upon their manner and course of development. This testimony obtained from the patient or friends is called the *history*. It may be divided into two parts: first, the history in the narrow sense of the term, including the patient's story of his illness up to the time the physician sees him; and, second, his account of his present symptoms, an account which must always be supplemented by an objective examination. The answers to the questions obtained in the history frequently furnish sufficient evidence to make a comparatively accurate diagnosis without examining the patient. Countless examples might be mentioned in illustration; for instance: A patient says that a few days before, when in apparently good health, he was suddenly seized with a severe chill and a stabbing pain upon one side of his chest, and that ever since then he has been feverish and short of breath, coughing, and expectorating rusty sputum. With such a story a physician would naturally make the diagnosis of croupous pneumonia.

Before attempting any objective examination, merely by skilfully directing his questions, in this way can an experienced physician obtain a fair idea of the disease, even in cases where the evidence is less conclusive than in the above example, although an exact diagnosis can be made only after completing the objective examination; for many diseases furnish only subjective symptoms. There are even cases in which the history affords the only clue to diagnosis. A ripe experience is requisite in order properly to utilize the history in making a diagnosis. A complete knowledge of the pictures of all the diseases which might enter into consideration in any given case is very essential in selecting the proper questions, as well as a keen critical power in interpreting the value of the evidence given in the history, since otherwise unessential facts might be made conclusive points in a diagnosis. Only years of experience can overcome one very constant difficulty—the varying individuality of patients. An hysteric society woman describes to her physician symptoms which, in a sturdy laborer, would be properly attributed to pathologic changes; but the adept practitioner understands that such complaints are nothing more than the expression of the peculiar, sensitive and exaggerating mental condition of an hysteric person, and so does not lay too much stress upon their significance. *Vice versâ*, a stolid, callous peasant often complains very little even when afflicted with a serious disease; or, again, a patient normally sensitive may be so

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benumbed by a disease that he does not complain at all. In the latter instance the great contrast between the subjective well-being and the objective visible disease frequently suggests a very unfavorable prognosis.

Without depreciating the value of the statements of the patients and their friends, it is evident from what we have said that the methods of objective examination, with which this book is concerned, contain the most essential elements in diagnosis. Although I have illustrated the possibility and the occasional necessity of making a diagnosis merely from the history, yet I could as easily mention innumerable instances where the most skilled practitioner could not form an approximate conception of the disease without the most searching physical examination. Even in the simplest of cases no physician should neglect the precaution of examining his patient carefully, including in such an examination all the organs. For, on the one hand, some organic diseases do not excite subjective symptoms; and, on the other hand, along with some organic changes which annoy the patient sufficiently to lead him to consult his physician, may go others of the greatest importance of which he has no suspicion and which the objective examination first discloses.

Objective examination includes a number of different methods, some depending on mere observation, but others requiring especial technical, chemical, or physical aids. The beginner must very early acquire facility in all these methods of examination. Their mastery will furnish the groundwork for acquiring extended experience in clinical observation upon the symptomatology, the course, and the prognosis of disease, and for obtaining reliable data for therapy.

A FEW SUGGESTIONS FOR TAKING HISTORIES.

It is very difficult to give any detailed directions which will be generally effective in taking a history. In serious diseases only an able and experienced physician is capable of performing this task thoroughly, and he will need to utilize his entire medical training. Since we shall mention in the appendix upon special diagnosis most of the methods made use of in history-taking, in the following paragraphs we need only give a few rules to serve as a framework for the beginner to broaden and build upon as his knowledge and experience grow.

Few persons are so mentally constituted as to communicate to the physician simply and directly the medically important facts of their ailment. Most patients relate a mass of unimportant matter and say nothing about the essentials, and only skilfully planned questions will prevent the patient or his relatives from irrelevancy. But a patient should never feel that he is being guided, nor that his physician does not enter with interest and sympathy into all the minute details of his trouble. Putting a mild curb upon the patient's volubility does not mean that one should concern one's self exclusively with the typical and characteristic symptoms of disease; because many things apparently immaterial in the eyes of the beginner, who knows a disease only as a scheme, have really considerable interest and a great importance. Even many conditions which have apparently nothing at all to do with the medical aspect of

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the case—for example, occupation, family affairs, etc.—are very helpful in comprehending the clinical picture, especially the etiology and, with it, the treatment. In short, the patient should be led to relate neither too much nor too little.

It is quite as important that the physician himself should be accurate in framing his questions. It is easy enough to ask too little, but difficult to ask too much or to question too minutely, not only on account of the multiplicity of the appearances of disease to be mastered, but even more on account of the danger of considering any important point as proved after a few hasty questions. In the writer's opinion this is the most frequent and serious fault which the beginner perpetrates; *e. g.*, a feverish patient is asked if he has had a chill, because such a symptom would suggest a definite disease, pneumonia. Without much thought most patients answer this question in the affirmative; but more careful questioning develops the fact that the supposed chill is in reality only the slight chilly feelings accompanying nearly all feverish diseases. In the typical chill of pneumonia the patient's teeth chatter, and he shakes as if immersed in ice water. This is quite a different symptom in its significance from the slight shivering of fever. The patient's statement that he has had a chill is not sufficient; we must inquire more particularly as to the nature of the chill. Often enough patients betray their mistake by using the word chill in the plural. Similar errors may arise from the statements patients make in regard to many other symptoms or long-standing diseases. The names they give to their former illnesses are especially apt to be incorrect and often occasion serious errors, for many are diagnoses made by the laity and many others are incorrect; *e. g.*, most cases of so-called "meningitis" which have been cured. Again, patients with tuberculosis often misname an acute exacerbation of their disease as "influenza"; further, so-called "catarrh of the stomach" is usually an early manifestation, or at least a forerunner, of tuberculosis. "Rheumatism" is another diagnosis which must always be looked upon a little skeptically. Frequently enough the clinical picture shows that the so-called "rheumatism" is a manifestation of tuberculosis, or of pleurisy, etc. Similarly, many other names of diseases, such as "nervous fever," "joint rheumatism," "dysentery," if accepted without criticism and without minute interrogation, may lead to errors in diagnosis. The best way to avoid such mistakes is to disregard names given by the patient and to make one's own diagnosis by establishing as objectively and accurately as possible the symptoms of the preceding disease.

A further and just as serious a fault is the tendency of many beginners to start from a preconceived notion of the diagnosis and to extract from the patient all possible facts in the history which will coincide with this supposed disease. To recognize this fault should be sufficient to effectually avoid it.

We can hardly ask too detailed questions as to the *influences of heredity*, inquiring accurately concerning parents, brothers, sisters, children, uncles, and aunts. An inquiry as to whether this or that disease

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has occurred in the patient's family will usually elicit a negative reply. To determine the facts accurately the disease in question must be quite specifically designated, possibly even a summary of the symptoms detailed. A patient may deny the occurrence of pulmonary disease in his family; but should we ask if either parent had a chronic cough, had expectorated blood, or lost a good deal of weight, we can frequently enough become convinced that one or the other suffered from tuberculosis.

In regard to neuropathic taint we must question very accurately and particularly. For example, an epileptic will practically always deny the occurrence of any nervous disease in his family. We may, however, obtain a positive reply by asking whether his father, mother, brother, sister, uncle or aunt was epileptic, if they had suffered from nervous attacks, or if they had been nervous or mentally affected in some other way.

If the histories are difficult to obtain or if patients contradict themselves, it is advisable to repeat questions later on, thus frequently clearing up some complicated point. The repetition of our task with stupid and prattling patients, unfortunately, obtains for us little more than renewed contradictions. Even this is a relative gain, for at least we discover how little we can trust them, and draw no false conclusions.

In general, good history-taking requires much diplomacy, tact, and knowledge of people and of medicine. A physician should never allow a patient to feel that he is in a hurry. The public considers that the physician has time for everything and everybody. Sit quietly, even if you are sitting upon hot coals; and wait for a favorable moment to interrupt, in a diplomatic way, the flow of talk. An excellent medical precept is not to fatigue a patient seriously ill with too thorough questioning, but to obtain as much as possible from the relatives, or to leave parts of the history until a later period. Furthermore, it is always advisable to discuss with the patient alone facts which he might wish to conceal from others. Finally, the beginner puts only a small part of the necessary questions, not realizing how much must be asked in order to make a complete history. Few rules can be given, but the following table will probably be of considerable service:

Scheme for History-taking.

Date; personal statements (name, age, position, occupation, residence); condition—*i. e.*, married or not; complaint; onset of the present illness; a description of the symptoms in the order of their appearance.

Etiology.—More exact information about the occupation and mode of life, injuries, strains, taking cold, errors in diet, etc. Infectious diseases in the neighborhood. Previous treatment and course of disease.

Past History.—Any antecedent disease like the present? If so, course of same. Injuries. Other earlier diseases; infectious diseases; and of these especially: joint rheumatism, scarlet fever, measles, whooping-cough, typhoid fever, erysipelas, malaria, sore throat, gonorrhea, syphilis. Previous symptoms of disease: Edema; dyspnea; cough;

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expectoration; expectoration of blood; palpitation of heart; difficulties of urination and alterations in the urine; constipation; diarrhea; icterus; vomiting; vomiting of blood; abnormalities of hunger and thirst; headache; marked changes of weight. In the female sex: chlorosis; pregnancies; births; menstrual or gynecologic difficulties. All these symptoms and conclusions which the past history furnishes must be analyzed in the same way as the symptoms of the present disease. (See 2. Complaint.)

Heredity.—

THE GENERAL ROUTINE OF A PATIENT'S EXAMINATION.

The following plan the author considers an excellent method for a routine examination; the order of the questions being the natural and practical one. The individual observer may expand the scheme in accordance with the contents of the work in question.

1. *Expression of countenance and general deportment of the patient; voice; speech; psychical behavior.*

2. *Complaint.* (See Scheme for History-taking.) *Kind of sick feeling? Weakness? Loss of flesh? Disturbances associated with the nervous system? Disturbances in connection with the organs of respiration?*

Dyspnea, constant or paroxysmal? The exciting cause of the paroxysms? Breathing slow or rapid during attack of dyspnea?

Cough, with or without expectoration? Characteristics of the expectoration? Admixture of blood? Peculiarities of the latter? Subjective sensation respecting the source of the expectoration (throat, larynx, nose?).

Pain with breathing? Its location?

Disturbances in Connection with the Circulation.—Palpitation, constant or paroxysmal? Apparent exciting cause of the paroxysm (agitation, exertion, or posture)? Palpitation accompanied by sensation of pain (left arm, back, precordia)? Palpitation accompanied by dyspnea? Subjective sensation of arrhythmia (*i. e.*, tripping or skipping of the heart beat)? Edema? Amount of urine?

Disturbances of the Digestion.—Appetite? Pain? Influence of the ingestion of food and drink upon the pain? Time of onset of pain—soon after eating—during the night—in a fasting condition? More exact location and radiation of pain (back, right shoulder)? Nausea? Vomiting? Amount and character of the vomitus (mucus, blood, food)? Its taste (sour, bitter)? Time of vomiting (pointing to retention or not)? Belching (sour, bitter, rancid)? Bowels: Constipation? How often do the bowels move? Movements painful? Character of feces: Color? Large scyballæ, abnormally small lumps? Distention of abdomen and other discomforts when constipated? Flatulence? Diarrhea: Frequency, consistence, color, amount of each dejection? Pain in defecation? Tenesmus? Bloody or slimy evacuations? Evidences of hemorrhoids?

Disturbances of the Urinary Apparatus.—Bladder or kidney pain? Radiation of the pain? Tenesmus of bladder? Amount of urine?

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Conspicuous qualitative changes of the urine (cloudy, bloody, smoky)? Passing of stone, gravel or sand?

Other Disturbances.—Fever? Night-sweats? Headache? Thirst? Insomnia (and its apparent cause)?

3. *History proper*, in conformity with the above plan.

4. *Examination proper*, which should cover the following points:

Build, development, and *nutrition.*

Temperature, frequency of pulse and *of respiration.*

Characteristics of Skin.—Bloating, edema, color (pallor, cyanosis, icterus), eruptions, pigmentation, scaling, striæ, final confirmation of other external diseases (joint-affections, erysipelas, etc.).

Head and Neck.—Mucous membranes, especially conjunctivæ, tongue, gums, pharynx, tonsils, herpes labialis, glands, goiter? Cervical veins (their dilatation or pulsation).

Respiratory Apparatus.—Dyspnea; its character, polygopnea, oligopnea, inspiratory or expiratory, stridor, shape of thorax, type of breathing, breathing excursions, diaphragm phenomenon, drawing in of thorax, topographic and comparative percussion and auscultation of the lungs, fremitus.

Circulatory Apparatus.[1]—Inspection and palpation of the precordia. Visible and palpable pulsation over that area. Location of apex beat. Thrills. Percussion and auscultation of the heart. Exact examination of the pulse. Rapidity, rhythm, fulness, tension, resistance of artery wall, conformity of the frequency of the radial pulse with that of the cardiac pulsation. Exact examination of the venous pulse. Auscultation of the arteries. Liver pulse. Capillary pulse.

Digestive Apparatus.—Inspection and palpation of the abdomen; its shape and fulness; visible peristalsis; sensitiveness to pressure, palpable tumors, resistances. Palpation and percussion of the stomach, intestines, liver, gall bladder, spleen, and peritoneum. Inspection of the vomitus and of the feces.

Urinary Apparatus.—Manner of urination; palpation of the kidneys and bladder; percussion of bladder. Catheterization. Character of urine; amount; color; cloudiness; specific gravity; testing for albumin and sugar.

Special Examinations which may be Necessary or even the Most Important of All in a Given Case.—Examination of the nervous system in accordance with the plan to be mentioned later. Rhino-, laryngo-, ophthalmo-, otoscopic examination. Testing the blood, including counting the corpuscles, estimating the hemoglobin, and the microscopic examination of the fresh and stained blood. Microscopic examination of the sputum, of the urine, of the vomitus, and of the feces. Bacteriologic examinations. Sphygmography and sphygmomanometry. Examination of the esophagus. Examination of the stomach by means of the stomach tube (distending with gas, a test meal). Distention of the colon for the demonstration of kidney tumors. Examination of the rectum; of the male and female genitalia. Needle punctures.

[1] In a routine examination the pulse is usually examined before the heart.

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It is the province of special pathology or of special diagnosis to interpret the signs of disease discovered in this manner, to weigh their mutual values, and to unify them into a definitely conceived disease of etiologic, functional, or anatomic nature.

GENERAL CONDITION OF THE PATIENT.

POSTURE IN BED.

THE general deportment of a patient is the first thing which attracts our attention and influences our judgment as to his condition. In many cases even the patient's relatives will reveal whether the illness is serious or slight. Ordinarily, physicians see seriously sick patients in bed, while those with slight ailments walk about. Yet there are countless exceptions. Sometimes seriously ill patients go to bed only at the last extremity; and, as is well known, patients may walk about even during the height of typhoid fever or pneumonia. *Vice versâ*, many patients take to their beds on account of very slight ills. These peculiarities depend upon the social position and the employment of a patient, and upon the great difference in the individual susceptibility to sickness. Besides, we must remember that even very slight ailments which always run a favorable course are sometimes associated with such distressing symptoms that the patient is compelled to take to his bed. Despite such exceptions, we may say that certain diseases necessitate rest in bed, others are ambulatory. Patients with the acute exanthemata are commonly found in bed by the physician because they feel very ill. The same is true of circulatory disturbances, peritonitis, meningitis, pneumonia, and acute inflammatory rheumatism. It is ordinarily easy enough to determine whether the patient keeps his bed on account of feeling ill, weak, and perhaps feverish, or on account of dyspnea, pain, or other difficulties, which are increased by walking about.

THE EXPRESSION.

The expression is of great diagnostic importance, enabling the skilled physician to draw conclusions as to the subjective feelings and the mental condition of the patient. The terms commonly applied to the expression—suffering, anxious, painful, careworn, uneasy, very ill, agitated, dulled, stupid, flustered—are perfectly plain without further explanation. Feverish patients present a characteristic appearance; sometimes they have a peculiar animated look, at other times an exceptional depression of the mimic faculty, often combined with glistening eyes, a feverish redness, and an increased turgidity of the skin of the face. The facial expression of a patient suffering from dyspnea is quite as distinctive, dependent upon peculiarities in the appearance of the skin (cyanosis, edema) and upon the mimic elements. The dilatation of the

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nares (see later), combined with the open mouth, is especially characteristic. (See p. 49 for a description of the typical *facies Hippocratica.*) The unusual expression of tetanus, called the *risus sardonicus* (sardonic laugh),[1] has been variously described. While the mouth is distorted as in laughing, the upper part of the face, especially the brow, is wrinkled, just as in the expression of trouble or sorrow. Tetanus poison apparently contracts the muscles of the entire facial territory and causes a combined stimulation of practically antagonistic muscles.

MENTAL CONDITION.

A patient's facial expression and his demeanor during our questioning furnish the best means of estimating his mental condition.

ACTIVE AND PASSIVE POSTURE IN BED.

A critical observer obtains diagnostic points from noticing the position which the patient assumes in bed. The less the general feelings are affected, the more natural and unconstrained is his pose. He tosses about, pushes the pillows straight, and shifts his attitude when one position has become uncomfortable. This is called an active position in bed (active dorsal or lateral posture). On the contrary, very weak, helpless, or unconscious patients appear very differently. Their attitude is lax, essentially controlled by the laws of gravity. Should such a patient slide down against the footboard, he would remain lying there, for he is incapable of drawing himself up, even if the position be very uncomfortable and his breathing embarrassed. This is called a passive position in bed (passive dorsal or lateral posture).

CONSTRAINED ATTITUDES.

Some very characteristic postures are almost diagnostic of certain diseases. For example: respiratory, cardiac, or renal affections associated with much dyspnea prevent a patient lying upon his back: in the first place, because the accessory muscles of respiration can be used to advantage only in the sitting posture, with the spine fixed and sometimes the arms; in the second place because, if fluid has accumulated in the abdominal cavity, the sitting posture partially relieves the diaphragm of its pressure;[2] and finally, because the influence of gravity possibly relieves the venous congestion of the brain and of the respiratory center in particular. With extreme dyspnea, the so-called "*orthopnea*," a patient cannot lie down, but, exhausted, is obliged to sit erect, bracing himself with his elbows and forearms upon the arms of his chair in an endeavor to utilize the accessory muscles of respiration and, if fluid be present in the abdomen, to avoid pressure of the anterior surface of the

[1] This name is derived from "sardone," a poisonous plant of the ancients, the ingestion of which produced such an expression.

[2] Except, of course, when an enormously distended abdomen is crowded by the thighs in the sitting posture.

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thighs upon the distended abdomen. It is worth remembering that the accumulation of a considerable quantity of blood in the veins of the lower extremity may afford some relief to the lungs and the heart, so that elastic bandages around the legs, temporarily shutting off a considerable amount of venous blood, may enable the patient to lie down, even though for a very short time.

Constrained lateral positions are very suggestive, for they almost always depend upon unilateral affections of the thoracic viscera. If on account of pulmonary infiltration or of compression by a pleural effusion the function of one lung is abolished, the patient usually lies upon the affected side, in order to afford the sound lung the freest possible expansion. Should there be much pain, such a position is generally reversed, because the weight of the body increases the pain; but sometimes when

FIG. 1.—Case of cerebrospinal meningitis. Photograph taken from above; patient lying asleep. Marked retraction of neck, flexion of thighs and legs (Harlem Hospital, Dr. R. G. Wiener).

the pain depends practically upon the breathing the patient will lie upon the affected side, and so limit the respiratory excursion by partially fixing the painful side with the body weight. In heart diseases, and sometimes in health, one side is more comfortable than the other to lie on. The position which dislocates the heart, the great vessels, and the mediastinum most, thereby rendering the breathing difficult, will be avoided. Lying on one side will sometimes relieve a patient who is perpetually tormented with a cough when in the dorsal decubitus. As one can easily imagine, in pulmonary cavities with constantly renewed secretion certain positions will aggravate a cough if the secretions are being continually poured out upon the healthy bronchial mucous membranes. In some positions the cavity would become entirely filled before any overflow would excite the paroxysm of coughing.

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The latter then completely empties the cavity, thus affording the patient a temporary rest. The diagnosis of a cavity would be strongly suggested by such history.

With colic, with cardialgia, and sometimes with intestinal obstruction patients generally prefer to lie upon the abdomen, because the tension of the distended intestines is diminished or their position shifted and the pain relieved; but in peritonitis the abdomen is so sensitive to pressure that the dorsal decubitus is assumed. On account of the epi-

FIG. 2.—Adiposity: The enormous accumulation of fat over and within the abdomen simulates a collection of ascitic fluid.

gastric tenderness, it is comparatively rare to find a patient with a gastric ulcer lying upon the abdomen, unless such a position frees the ulcer from the contact or pressure of the gastric contents—*e. g.*, if situated upon the posterior wall. Patients with headache sometimes prefer to lie upon the abdomen. The characteristic positions of patients suffering from cerebrospinal meningitis or wry-neck depend upon cramp-like contractions of certain groups of muscles; some peculiar paralytic positions, upon paralyses of muscles.

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GAIT AND THE ATTITUDE.

An alert, erect attitude and a rapid walk usually signify good physical condition, while a stooping, relaxed posture with a slow, fatigued gait indicates that the person is seriously ill or mentally depressed. (For a description of various characteristic gaits see Examination of the Nervous System.)

DEVELOPMENT AND STATE OF NUTRITION.

A robust, vigorous or muscular physique means that the bodily dimensions are rather above the normal, whereas a weakly or puny physique would indicate the contrary. The subcutaneous fatty layer (panniculus adiposus) is perhaps of even more importance than the muscles in estimating the state of nourishment. The former varies within normal limits in accordance with the age, sex, and occupation of patients. Corpulency is generally associated with a weak musculature. Nursing infants possess an extremely well-developed layer of fat; during childhood it gradually diminishes, and in the third or fourth decade increases again, while at old age it finally diminishes. A marked tendency to corpulency is often observed in women especially after the menopause.

Most chronic diseases are accompanied by a noticeable deterioration of the general nutrition. This is due either to lack of appetite, and, hence, insufficient food, to defective assimilation, or to excessive

FIG. 3.—Pronounced emaciation in a chronic disease. Case of multiple myeloma (New York City Hospital).

combustion of the food assimilated. Emaciation is particularly evident in chronic febrile and digestive diseases, and becomes most pronounced in severe and prolonged typhoid fever, in phthisis, in carcinoma, especially esophageal carcinoma, and in certain types of diabetes mellitus. In these diseases the musculature suffers a loss almost as rapidly as the fatty tissue. Marked emaciation nearly always suggests chronicity.

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BODY WEIGHT.

Observation of weight over a certain length of time furnishes an excellent guide to the state of nutrition. Minute cautions in regard to the accuracy of the scales, weight of different clothes, etc., are hardly necessary to enumerate. Unless accurately estimated the weight is of no value. It is more accurate to weigh the body always either before or after eating, since a hearty meal will often make a difference of one or more pounds. General edema or the accumulation of fluid in one of the large serous sacs will considerably increase a patient's weight, while free catharsis, diuresis or diaphoresis will rapidly diminish it. Careful comparative weighing in these cases furnishes very good evidence of the progress of the disease.

Infants should be weighed weekly, to keep track of their nutrition. The normal weight of the newborn is (females) 3000 gm. (6 lbs.) to (males) 3500 gm. (7 lbs.) (Uffelmann). During the first three or four days of life there is a physiologic loss of 200 to 300 gm. ($\frac{1}{2}$ lb.). Gerhardt[1] cites the following table:

Daily increase in			1st	month	25 gm.		Daily increase in			7th	month	15 gm.	
"	"	"	2d	"	23	"	"	"	"	8th	"	13	"
"	"	"	3d	"	22	"	"	"	"	9th	"	12	"
"	"	"	4th	"	20	"	"	"	"	10th	"	10	"
"	"	"	5th	"	18	"	"	"	"	11th	"	8	"
"	"	"	6th	"	17	"	"	"	"	12th	"	6	"

Quetelet's table (an extract from which is inserted below) does not take into account the weight of the clothes, this has been estimated to be in men about $\frac{1}{18}$ and in women about $\frac{1}{24}$ of the total weight; although, of course, such figures must vary considerably:

	Male.	Female.
Newborn	3.1 kg. (6.83 lbs.)	3.0 kg. (6.61)
1st year	9.0 " (19.84 ")	8.6 " (18.96)
2d "	11.0 " (24.25 ")	11.0 " (24.25)
3d "	12.5 " (27.56 ")	12.4 " (27.34)
4th "	14.0 " (30.86 ")	13.9 " (30.64)
5th "	15.4 " (33.95 ")	15.3 " (33.73)
6th "	17.8 " (39.24 ")	16.7 " (36.82)
7th "	19.7 " (43.43 ")	17.8 " (39.24)
8th "	21.6 " (49.62 ")	19.0 " (41.89)
9th "	23.5 " (51.81 ")	21.0 " (46.30)
10th "	25.2 " (55.56 ")	23.1 " (50.93)
11th "	27.0 " (59.52 ")	25.5 " (56.22)
13th "	33.1 " (72.79 ")	32.5 " (71.65)
15th "	41.2 " (90.83 ")	40.0 " (88.18)
17th "	49.7 " (109.57 ")	46.8 " (103.18)
19th "	57.6 " (126.98 ")	52.1 " (114.86)
20th "	59.5 " (131.17 ")	53.2 " (117.28)
25th "	66.2 " (145.94 ")	54.8 " (120.81)
30th "	66.1 " (145.72 ")	55.3 " (121.91)
60th "	61.9 " (136.46 ")	54.3 " (119.71)
70th "	59.5 " (141.17 ")	51.5 " (113.54)

[1] Gerhardt, *Lehrbuch der Kinderkrankheiten*, 1881, p. 2.

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MENSURATION.

An individual's general development must be judged by comparisons between his age, weight, and height. Quetelet[1] is also responsible for the following table:

	Male.	Female.
Newborn	50.0 cm. (1.8 ft.)	49.4 cm. (1.7 ft.)
1st year	69.8 " (2.3 ")	69.0 " (2.3 ")
2d "	79.1 " (2.7 ")	78.1 " (2.7 ")
3d "	86.4 " (2.10 ")	85.4 " (2.10 ")
4th "	92.7 " (3.— ")	91.5 " (3.— ")
5th "	98.7 " (3.3 ")	97.4 " (3.2 ")
6th "	104.6 " (3.5 ")	103.1 " (3.5 ")
7th "	110.4 " (3.7 ")	108.7 " (3.7 ")
8th "	116.2 " (3.10 ")	114.2 " (3.9 ")
9th "	121.8 " (4.0 ")	119.6 " (3.11 ")
10th "	127.3 " (4.2 ")	124.9 " (4.1 ")
11th "	132.5 " (4.4 ")	130.1 " (4.3 ")
13th "	142.3 " (4.8 ")	140.0 " (4.7 ")
15th "	151.3 " (5.0 ")	148.8 " (4.11 ")
17th "	159.4 " (5.3 ")	154.6 " (5.1 ")
19th "	165.5 " (5.5 ")	157.0 " (5.1 ")
20th "	167.0 " (5.6 ")	157.8 " (5.2 ")
25th "	168.2 " (5.6 ")	157.4 " (5.2 ")
30th "	168.6 " (5.6 ")	158.0 " (5.2 ")
40th "	168.6 " (5.6 ")	158.0 " (5.2 ")
60th "	167.6 " (5.6 ")	157.1 " (5.2 ")
70th "	166.0 " (5.5 ")	155.6 " (5.1 ")

Circumference of the Chest.—The chest measurement furnishes a very good idea of the state of development. It is used in many countries in examining army recruits. Fröhlich[2] recommends the following method: The measuring tape is to be applied horizontally at the level of the nipples in front and just beneath the angles of the scapulæ behind while the arms are held horizontally. The measurements during extreme inspiration and again during extreme expiration are to be recorded, the difference representing the *chest expansion.* He found among recruits that the average in men of twenty years was, for expiration 82 cm. (32¾ in.), for inspiration 89 cm. (35⅛ in.), and the chest expansion 7 cm. (2¾ in.), and he attaches considerable diagnostic importance to these figures, because he noted that in emphysema, in phthisis, and pleural effusions the circumference, especially during expiration, was increased, while the chest expansion was diminished, and this diminution might persist for some time after the disease has subsided. The chest expansion is diminished if the costal cartilages become ossified (the so-called Bryson's sign). This is often the case in exophthalmic goiter.

[1] *Anthropometrie*, 1870.

[2] H. Frölich, *Die Brustmessung im Dienste der Medicin*, Leipzig, 1894. A full bibliography is quoted here.

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CONFIGURATION OF THE THORAX.

NORMAL SHAPE OF THE CHEST.

A normal chest, such as one sees so beautifully illustrated in the masterpieces of classic statuary, should be symmetric, the surface well rounded, without sharp corners or depressions; the intercostal spaces only visible between the lower ribs; the subcostal angle about 90°; the sternum nearly straight in profile, without a decided angle between the corpus and the manubrium; and the sternovertebral somewhat shorter than the transverse diameter. The gradual increase in the horizontal diameters of the thorax should form a sort of pyramid, with its base below, but the graduation should not be too marked, and the shoulder girdle, or in females the breast, should offset this difference. The shoulders should be nearly horizontal, the scapulæ lying flat against the back, clavicles not too prominent, and the supra- and infraspinous fossæ not too deep.

FIG. 4.—Emphysematous chest (Dr. W. H. Smith, Massachusetts General Hospital).

PATHOLOGIC SHAPES.

Emphysematous Chests. — These abnormal forms depend upon an emphysematous enlargement of the lungs; and they possess one feature in common—the thorax seems abnormally widened and prominent. The sagittal diameter is generally increased and the subcostal angle more obtuse than in the normal thorax. When the emphysema is diffused over the entire lung or when mostly limited to the lower chest, the thorax looks as if in a normal position of deep inspiration; but when caused by forced expiration (coughing) the emphysema is situated more in the upper chest, because the respiratory power acts more upon the lower parts of the thorax. Then the upper thoracic aperture appears enlarged and the so-called "barrel chest" is produced. These are the two different types.

The Paralytic Thorax.—Contrasted with the emphysematous thorax, the so-called *paralytic thorax* is abnormally flat, long and sometimes narrow; the ribs, both in front and behind, have a marked downward direction; the intercostal spaces are widened; the subcostal angle is very acute; the supra- and infraclavicular fossæ are deep; the intercostal muscles and those of the shoulder girdle are feebly developed (hence the name); and the shoulder blades project noticeably, like wings, because of a weakness of the muscles, especially of the serratus magnus. This type of chest is observed frequently in *weakly* or *cachectic* individuals, very commonly in *phthisis*. It was formerly considered as a

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predisposing cause of consumption, and is now frequently spoken of as "the phthisic chest."

Scoliotic, Kyphotic, and Scoliokyphotic Thoraces.—These terms are applied to twists and deformities of the chest which are observed as a sequence of spinal curvature. They are often quite pronounced. It is often difficult to detect such deformities from the front; but by noting the low stature, the short thorax, and the marked breadth of shoulders an experienced eye can generally discover them.

Rachitic Chests.—Rickets is responsible for many chest deform-

FIG. 5.—Paralytic chest (phthisis) (Dr. W. H. Smith, Massachusetts General Hospital).

FIG. 6.—Paralytic chest (phthisis) (Dr. W. H. Smith, Massachusetts General Hospital).

ities, although often very slight ones. Perhaps the most characteristic is the keel-shaped prominence of the sternum called *pectus carinatum* (pigeon breast). It is associated with a compression of the anterior diagonal horizontal diameter and an increase in the sternovertebral diameter of the chest (Fig. 8). A transverse groove (Harrison's groove) often marks the insertion of the diaphragm to the ribs. The so-called "rachitic rosary," a beaded enlargement at the line of junction of the bony ribs with their costal cartilages, can be felt as well as seen. It generally disappears in later childhood. The other rachitic deformities may persist, or they may become modified to a greater or less extent.

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Boat-shaped Chest of Syringomyelia.—Pierre Marie and Astié[1] described, under the name of "thorax en bateau," a depression of the upper portion of the anterior chest-wall, which, so far as we know, is only observed in syringomyelia. The cavity lies in the median line, as if sunk against the spinal column, and extends downward as far as the lower edge of the pectoralis major; it may be as deep as 5 cm. The atrophy of the pectorals and of the other muscles takes no part in producing this appearance.

Funnel-shaped Chest and Cobbler's Chest.—The true *funnel-shaped chest* is either congenital or else develops in early infant life, gradually and without any known cause. It consists of a funnel-shaped depression of the lower end of the sternum, which frequently reaches quite deeply into the interior of the chest. It may lead to circulatory or respiratory disturbances resembling those observed in kyphoscoliosis. Cobblers may acquire a very similar deformity, due to the constant pressure against the lower end of the sternum. The cobbler type is, however, limited to the inferior portion of the sternum, or even to the xiphoid process alone.

FIG. 7.—Right scoliosis (Moore).

[1] *Soc. méd. des hôpitaux*, 19, ii., 1897.

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Asymmetry of the Chest Due to Disease of the Thoracic or Abdominal Viscera.—An expansion or a contraction of one chest-half may result from various affections of the thoracic viscera—*e. g.*, a large pleural exudate, a pneumothorax, or even to a slight extent a croupous pneumonia. The alteration may be general over one entire side or localized. The affected side may be the larger or the smaller.

A considerable pleural effusion or a pneumothorax produces an enlargement of the affected side of the thorax, an obliteration of the intercostal spaces, a dis-

FIG. 8.—Pigeon breast (Dr. R. C. Cabot, Massachusetts General Hospital).

location outward of the nipple and of the scapula, a convexity of the spine toward the affected side, and an elevation of that shoulder. The last two deformities, both probably due to an alteration of the center of gravity, make the patient look as if he were carrying a weight on the affected side.

The lower thorax may be enlarged by a decided increase in the size of the liver or spleen. Such an enlargement will become still more noticeable if tympanites or ascitic fluid distends the abdomen enough to prevent the dropping of the enlarged liver or spleen. An aortic aneurism or an intrathoracic tumor may cause a localized enlargement of the thorax. This will be situated at the point of contact of the growth with the thoracic wall. Actual contact is not, however, necessary, because, even when the growth or aneurism is merely in close proximity to the wall, a localized bulging will be caused by the diminution

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in the intrathoracic negative pressure—*e. g.*, an aortic aneurism, even when covered by the lung, will cause a local prominence. Neither does a pleural effusion need to be under positive pressure, as is so commonly supposed, in order to enlarge the affected side of the chest. (See what is said upon dislocation of organs by exudations, p. 79.) Cardiac enlargements and pericardial effusions give rise to a precordial bulging. The lower thorax will be quite uniformly and decidedly expanded by any marked increase in the contents of the abdomen—*e. g.*, meteorism, ascites, or large ovarian tumors.

FIG. 9.—Rickets: Note the size and shape of head (see p. 19), the rosary, Harrison's groove, kyphosis, prominent belly, bowing of legs, and the enlargement of wrists (Dr. W. L. Stowell, Randall's Island Hospital).

Chronic indurative processes in the lung—*e. g.*, chronic pneumonia with or without bronchiectasis, chronic tuberculosis—are apt to cause a unilateral contraction of the thorax, sometimes including the entire half of the chest. The coincident pleuritic contraction may be partly responsible for the deformity. After the absorption of a pleuritic effusion it is very common to find a retraction of the affected side of the chest instead of the previous enlargement. This is due to the fact that the lung, incased in thick fibrous adhesions, has been compressed so long that it can never again expand fully, and that the pleuritic adhesions tend to contract and draw the thorax in. These pulmonary and pleu-

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ritic conditions are, of course, very commonly combined. When the contraction of one side of the chest is very decided, the scapula, clavicle, and the neighboring soft parts are plainly depressed; the nipple and the shoulder then approach the median line, and the spinal column presents a curvature with a concavity toward the affected side. The corresponding dislocations of the thoracic viscera will be discussed under Topographic Percussion. Noticeable increase in the depth of the supra- or infraclavicular fossæ suggests a retraction of the apices, and is therefore an early and important sign in pulmonary tuberculosis.

Inspection reveals these chest deformities more accurately than direct measuring with the tape; probably because the eye notes differences not only in the size of

FIG. 10.—Funnel-shaped chest (Dr. R. C. Cabot, Massachusetts General Hospital).

the chest, but in the extent of the respiratory excursions. The latter are, of course, always diminished on the affected side. The extent of the respiratory excursion is the surest way to decide whether a given deformity is due to an enlargement of one side or a contraction of the other side. Another reason why measuring with the tape is apt to be inaccurate in pleuritic effusions is this: The lower end of the sternum is generally dislocated to the affected side, so that the middle line of the sternum forms an acute angle with the true median line of the body. Therefore, measuring from the middle of the spinal column to the middle of the sternum is not accurate. We should measure to a plumb line dropped from the middle of the jugulum (ligne du cordon).

Woillez's cyrtometer is a practical instrument for measuring and picturing chest deformities, and is rather better than the ordinary tape; but as a matter of fact a narrow lead band, about the thickness of a lead pencil, is more convenient than the specially devised instrument. It can be accurately applied to the chest-wall at one level and removed without altering its shape; a tracing can then be made upon paper and measurements taken. Such tracings are sometimes very instructive in

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following the course of an obstinate pleurisy. (For the discussion of more complicated apparatus, the reader is referred to the references cited below.[1])

FIG. 11.—Scoliosis and asymmetry of the thorax: Patient with fibroid phthisis of left lung; left chest smaller than right; left shoulder (affected side) higher than right; left scapula nearer the spine; dorsal spine concave to left; lumbar spine with a compensatory convexity to left (New York City Hospital).

SIZE AND SHAPE OF THE HEAD.

The horizontal circumference of the skull in the newborn is normally 40 cm., at the end of the first year 45 cm., and at puberty 50 cm. (Strümpell).

During the first eight or nine months the large fontanel slightly increases in size; about the tenth month it begins to diminish, and at the sixteenth month it should be closed.

[1] F. Schenk, "Zur Aetiologie der Skoliose." Vortrag, gehalten in der Chir. Section der 58. Versammlung deutscher Naturforscher und Aerzte zu Strassburg i. E. Berlin. Verlag von H. Heinicke. C. Hübscher, "Redresseur und Messapparat." Ein Beitrag zur Therapie der fixierten Skoliose. Beiträge zur klinischen Chirurgie, redigiert von P. Bruns, vol. xiii., part 1.

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Hydrocephalus may be either congenital or acquired early in life. The skull is enlarged, and most of the bones are separated by broad sutures and fontanels.[1] The cranium looks like a big bladder, tapering downward. The orbital roofs are

FIG. 12.—Hydrocephalus: Note size and shape of the head as compared with the face; the prominence of eyes and overhanging orbital roof; marked talipes equinovalgus of left foot (Dr. W. L. Stowell, Randall's Island Hospital).

projected downward, causing the eyes to look downward in a very characteristic fashion; and the face is diminutive in comparison. The cranial bones are very thin, and when pressed upon crackle like parchment.

Rachitis is characterized by a peculiar malformation of the skull. Although the longitudinal and transverse diameters are approximately normal, the cranium has a distinctive appearance, due to the prominent frontal and parietal eminences and to the vertical position of the occipital bone, forming the *square-shaped head* (tête carrée). The large fontanel may remain patent into the third, fourth or even the sixth year, and the sutures a correspondingly long time. The thinned areas of bone over the cranium, and especially over the occipital bone, will furnish the same parchment-crackling described above.[2]

[1] The horizontal circumference may be as much as 50 cm. in the first month of life.

[2] Special Pathology must be consulted for a description of the deformities of the face in acromegaly and of the asymmetry in hemiatrophia facialis progressiva.

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EXAMINATION OF THE SKIN.

AN examination of the skin should not be restricted to cutaneous diseases alone, because in diseases of the internal organs we meet with all manner of changes in the skin, which can be appreciated by the sense of sight or by that of touch.

COLOR OF THE SKIN.

The normal fleshy tint of the skin is due to the color of the blood shining through the epidermis and the superficial layers of the cuticle. The color of the skin may be modified, both physiologically and pathologically, by (1) a quantitative increase or decrease in the amount of flesh color; (2) a qualitative change due to abnormal pigmentation. The first is better observed in the face; the second, in other parts of the body, where the normal flesh color is not so prominent. Many qualitative variations, occasioned by abnormal pigmentation, are, however, determined better over paler uncovered areas.

QUALITATIVE CHANGES OF THE FLESH COLOR.

The intensity of the flesh color depends upon (1) the intensity of the blood color; (2) upon the amount of blood contained in the vessels; (3) upon the thickness of the layers of skin covering the vessels. Any one of these three factors may vary and so cause alterations in the flesh color of more or less diagnostic importance.

PALLOR.

Pallor Depending upon Oligochromemia.—A pale face presupposes anemia, or a diminution of the coloring power of the blood (oligochromemia)—that is to say, a qualitative alteration of the blood. For we do not recognize any real quantitative diminution ("blood poverty" of the Germans) except that which occurs after a profuse hemorrhage, and this is rapidly made up by reabsorption of lymph and water, thus causing again the condition of oligochromemia. As we shall see later on, pallor is a very uncertain guide in the diagnosis of oligochromemia.

Pallor without Oligochromemia.—It is impossible to diagnose anemia from pallor alone. A careful blood-examination (see Section on p. 659) is absolutely essential in order to avoid the mistake of diagnosing anemia in a patient with normal blood simply because his face is pale; or of reassuring a patient with red cheeks whose hemoglobin percentage is only 50.

Aside from oligochromemia, the causes of pallor of the face are numerous—*e. g.*, many people absolutely well, without a symptom or sign of disease, are abnormally pale. The only way to explain such pallor, providing, of course, the conjunctival and mucous membranes are not also pale, is to assume either an abnormal opacity of the

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epidermis or a scanty supply of blood-vessels in the face. During the course of an illness a patient sometimes gradually becomes paler and paler, and although the hemoglobin percentage remains normal, the physician, unless he examines the blood, will certainly consider the condition one of oligochromemia. Here we must suppose that the pallor is due either to a diminution of the total amount of blood, or that the skin contains less blood than normally as a result of morbid changes in the circulation. As suggested above, except in severe hemorrhages, we know no way of determining that there is a diminution in the total amount of blood as compared to the weight of the body, so we must content ourselves with the second explanation[1] —an alteration in the circulation. The latter is all the more probable in connection with certain other symptoms—*e. g.*, weakness of the pulse, slight grade of cyanosis (see p. 40 *et seq.*), dilatation of the external cutaneous veins, slight dizziness, malaise, general physical debility, etc. It is difficult to determine whether the diminished amount of blood in the small arteries and capillaries, which is certainly the cause of the pallor, depends upon an alteration in the cardiac activity or in the vasomotor control. A low blood-pressure is associated with pallor if entirely dependent upon diminished cardiac activity, the vasomotor tonus remaining constant; but if the small arteries dilate, pallor does not result. High blood-pressure is characterized by a marked pallor, provided there is a very decided vasomotor contraction.

A closer study of the connection between a normal hemoglobin percentage and this pallor would be of some service for careful treatment; because so long as every pale patient is considered anemic, as is unfortunately too often the case in practice, we shall not progress very far toward proper therapy. Affections of the stomach, heart disease, and phthisis, although associated with pallor, often show a normal blood. All such conditions will, of course, lead later on to oligochromemia.

The temporary pallor caused by nausea, collapse, or violent psychical impressions practically comes under the head of an alteration in circulation, being due partly to vasomotor, partly to cardiac, influences.

ABNORMAL REDNESS OF THE SKIN OF THE FACE.

If the above conditions which produce pallor are reversed, an abnormal redness of the skin will result. A hemoglobin percentage as high as 120, although very rare, may be responsible for an increased color. We do not know as yet whether there is such a condition as true plethora; v. Recklinghausen thinks there is, and so explains the appearance of full-blooded people. In many cases such an appearance is caused by an abnormally thin or transparent skin; in others by a local increase in the amount of blood sent to the face. The so-called "rosy chlorotics" are supposed to possess blood-vessels of abnormal transparency. In all such cases an examination of the blood

[1] Although it seems probable that extreme emaciation is associated with a diminution in the amount of blood, it has never been proved that this diminution is out of proportion to the loss of body weight.

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is absolutely essential before a diagnosis of oligochromemia can be made. A suggestion is, however, often obtained by comparing the pallor of the conjunctivæ with the rosy hue of the cheeks.

People who spend most of the day out of doors, and especially those who are exposed to all sorts of weather, generally acquire a ruddy face from the constant increase of blood in the exposed portions.

Alcoholics are usually flushed on account of the dilating influence of alcohol upon the vessels. Minute dilated radicals may also be seen just underneath the epidermis, or an acne rosacea, which is so frequent in drinkers. As is well known, this same peculiarity is often observed in perfectly healthy individuals, in whom it is difficult to determine any cause. Fever produces a characteristic redness, ordinarily combined with some puffiness and moisture of the skin of the face. The lax condition of the muscular coat of the vessels is apparently responsible. This is evident in a sphygmographic tracing. The flush of fever is most noticeable in the cheeks, especially when contrasted with a pallor such as is observed in tubercular patients (so-called hectic flush). Psychical excitement, shame, violent physical exertion, external applications of heat to the skin (baths, insolation), the inhalation of nitrite of amyl, or a moderate grade of atropin- or opium-poisoning, all cause a flushing of the face from vasomotor influences. The reddening in these cases may spread to the neck and to the upper part of the trunk. Poisoning with carbon monoxid ordinarily produces a very intense redness of the face, which is supposed to be due to an alteration of the blood. The latter, however, contains so little carbon monoxid that it is even difficult to determine it with the spectroscope, so that the explanation is unsatisfactory.

Hemicrania and some affections of the cervical sympathetic sometimes cause a unilateral redness of the face, which is commonly associated with a unilateral pupillary change. The redness of the face associated with various cutaneous diseases and the acute exanthemata need not be discussed.

CYANOSIS.

By this term is meant a bluish tinge of the skin and mucous membranes which depends upon the poverty of the capillary blood in oxygen and its abnormal richness in carbon dioxid. Since it is even a deeper blue than ordinary venous blood, we must assume in explanation some peculiarity in the skin-covering.

General Cyanosis.—Two chief factors take part in the production of general cyanosis:

(1) Insufficient oxidation of the blood in the lungs, so that the arterial blood in the capillaries is poor in oxygen and therefore dark.

(2) Obstruction to the venous return, so that the veins are dilated, the skin contains more venous blood than normally, the current is sluggish, and the blood is charged with more carbonic acid.

With true general cyanosis the bluish tinge will naturally be more pronounced in certain body parts—*i. e.*, there are certain points of predilection. The face, especially the cheeks, lips and ears, shows cyanosis

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very plainly, on account of the first of the two factors, because here the color shows through more plainly; the feet and the toes, the fingers and the finger-nails, on account of the second of the two factors (the venous stasis), the parts in question being so remote from the chest. The nose and the knees, too, often show cyanosis very noticeably.

General cyanosis will therefore appear in any respiratory or circulatory affection. These two systems are so intimately related and so dependent one upon the other that if the cyanosis is very pronounced we should naturally attribute it to an interference with both respiration and circulation, no matter which is primarily affected.

The following disturbances, primarily *respiratory*, cause cyanosis: Any affection preventing the free entrance of air to the lungs—*e. g.*, retropharyngeal abscess; croup; pseudocroup; edema or spasm of the glottis; paralysis of the inferior laryngeal nerve; tumor of the larynx; foreign bodies in the pharynx, larynx, trachea or bronchi; any kind of tracheal stenosis, as by goiter or other tumors; strangulation; bronchitis; bronchial asthma, etc.

Affections which diminish the amount of breathing surface of the lungs—*e. g.*, emphysema; all varieties of pulmonary infiltration; atelectasis; compression of the lungs by an exudation or by pneumothorax. In this connection it is interesting to note that in pulmonary consumption the cyanosis does not reach as high a grade as the amount of breathing surface involved would lead us to expect. This is probably because the emaciated body contains less blood and needs less oxygen, so that what remains of the healthy lung tissue is nearly sufficient to aërate the diminished amount of blood.

All affections which influence the activity of the respiratory muscles: paralyses and atrophies of the muscles of respiration (bulbar paralysis, etc.); as well as spasms (tetanus, epilepsy); further, any affection causing pain enough to inhibit respiration.

In all these disturbances the arterialization of the blood in the lungs is primarily affected, and later on some venous congestion results because the circulation lacks the help of the pumping action of respiration.

The following disturbances, primarily *circulatory*, cause cyanosis from congestion:

Uncompensated cardiac affections; valvular lesions and affections of the heart muscle; arteriosclerotic and nephritic changes; strains upon the heart; pericarditis, etc., as well as paralysis of the vasomotors.

Mitral defects, both those of insufficiency and those of stenosis, even when compensated, cause a certain degree of cyanosis. This is respiratory in nature, and depends upon the increased pressure in the pulmonary circulation, upon the resulting rigidity and brown induration of the lungs, and upon the accompanying bronchial catarrh, all of which impede the respiration.

The cyanosis of *congenital heart disease* is often more pronounced than in any other condition. It depends upon venous congestion, or upon the admixture of arterial with venous blood, or upon both. (See section upon Congenital Heart Lesions.)

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The cyanosis which is caused by *vasomotor paralysis* depends upon a dilatation of the small vessels (the smallest arteries, capillaries and smallest veins). It differs from the other types of general cyanosis in a lack of distention of the larger veins, and in the peculiar grayish-blue color of the entire surface of the body without the localization at certain points of predilection, which is so characteristic of the other forms. (See the following section.)

Certain poisons, notably antifebrin and nitrobenzol, turn the blood very dark by changing the hemoglobin into methemoglobin. This is not a true cyanosis, although the body surface is blue.

Cyanosis from Local Causes.—Localized areas of the skin may become cyanotic without any disturbance of the general circulatory or respiratory system. Either a local venous congestion from compression or thrombosis of large or small veins, or else some vasomotor disturbance, will be found to be responsible. Examples of the latter variety are the cyanosis of the hands, feet and ears produced by the local action of cold, the cyanosis of paralyzed extremities, of the hands and feet of hysteric individuals, and occasionally of perfectly healthy individuals. In the hysteric cyanosis is frequently combined with edema (œdeme bleu des hystériques).

This purely vasomotor cyanosis evidently depends upon a degree of vascular dilatation sufficient to produce stagnation from inadequate *vis a tergo*. The process may be compared to a running stream which, from the lack of a definite river-bed, becomes a swamp. One might think that such a dilatation of the vascular system, by diminishing the resistance, would assist the current. In this case, however, the current would not flow uniformly over so wide a territory, but much more rapidly by the centrally directed paths, while practically stagnant at the borders. The bright-scarlet spots which the writer has frequently observed scattered through the blue of vasomotor cyanosis furnish a proof of the accuracy of this theory. They correspond to areas of increased capillary current.

A cyanosis of similar origin appears in certain inflammatory processes. When extreme it is, of course, an unfavorable sign.

Cyanosis of the skin is naturally associated with increased loss of heat, so that cyanotic parts generally feel cold.

ICTERIC COLORATION OF THE SKIN (JAUNDICE).

Icterus is the peculiar pathologic yellow discoloration of the skin and mucous membranes, caused by bile pigment or some of its derivatives. The ordinary form (*obstructive icterus*) depends upon a total or a partial occlusion of the bile duct, so that instead of being emptied into the intestine, some or all of the bile is absorbed and stains the various tissues—*e. g.*, the skin and the mucous membranes. This discoloration varies in tint from a pale lemon yellow to a dark brown, olive green or almost black. The darker shades (*melasicterus*) always signify an obstruction of long duration. They are caused either by a great excess of pigment accumulated in the skin or by a transformation of the original bile pigment to darker shades.

Icterus is usually first noticed in the conjunctiva of the sclera and upon the unexposed body parts, which are covered with but a thin layer

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of epidermis and are only slightly pigmented. After the obstruction has persisted for a short time the skin of the face and the mucous membrane of the mouth share in the discoloration. Pressure of the finger, by diminishing the amount of blood, sometimes makes the yellow of a mild icterus plainer. Because the gums are normally so pale they show icterus before the rest of the mouth-lining. Unless very intense, jaundice cannot be appreciated by artificial light.

In looking for icterus we generally begin by examining the conjunctivæ. We should remember that the yellowish color of the subconjunctival fat, so pronounced in cachectic individuals and in drunkards, is sometimes mistaken for icterus. The distribution of this fat is largely limited to the neighborhood of the folds in the mucous membrane, so that a careful examination of the tightly drawn conjunctiva nearer the cornea generally prevents a mistake. *Pingueculæ*, peculiar formations of colloid degenerated connective tissue which contain yellow pigment, are situated within the palpebral folds on either side of the cornea. They may be another source of confusion.

Certain races and some individuals exhibit a yellowish tint of skin, which a beginner might confuse with a faint jaundice; but a careful examination of the conjunctiva would prevent this mistake.

The yellowish discoloration in picric acid poisoning can be easily differentiated from icterus by the history, etc., as well as by the urinary examination.

Bile pigment occurs in the urine and sweat of icteric individuals. Yellowish stains upon the linen sometimes attract a patient's attention before he has observed the yellow color of his skin. The saliva is for the most part unaffected. The feces become pale and more or less clay-colored on account of the lack of bile. The absorption of certain biliary salts with the biliary pigment produces, at least in fresh cases, a slowing of the pulse and considerable itching of the skin.

Although obstructive jaundice arises most frequently from a catarrh of the biliary passages, it may depend upon serious changes in the liver itself—*e. g.*, cirrhosis, carcinoma, abscess, etc.

Another type of jaundice, which differs from the obstructive by the fact that the feces are unaffected (*i. e.*, of normal color), depends upon very imperfectly understood causes.

Icterus neonatorum belongs to this type. The old idea was that the drying up of the blood-current in the umbilical vein after birth caused a sufficient lowering of the pressure in the portal vein to enable the bile pigment to pass from the liver into the portal vein. Quincke[1] and Schreiber[2] defend Peter Frank's theory of the cause of jaundice in the newborn. Frank argued that the bilirubin absorbed from the meconium, instead of being stored up in the liver for further use, escapes directly into the general circulation by way of the ductus venosus arantii. This duct generally remains open for a short time after birth. One argument in favor of this theory is the fact that icterus neonatorum occurs most frequently in premature infants.

The cause of the jaundice which is not infrequently observed in infectious diseases (pneumonia, pyemia and yellow fever) is not perfectly clear; it probably depends upon the absorption of bile pigment from the liver. It has been claimed that a catarrh of the biliary passages, due to the general venous congestion, is responsible for the jaundice in pneumonia, but microscopic examinations of the liver tissues in such cases have shown that the bile ducts were normal. Stagnation and reabsorption of bile from the lack of the usual rhythmic pressure exerted by the diaphragm upon the liver has been cited as another explanation of the icterus in pneumonia. If jaundice occurred only with pneumonia which

[1] *Arch. f. exper. Path.*, vol. xix., p. 34 *et seq.* [2] *Berlin. klin. Woch.*, 1895, No. 25.

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involved the lower right lobe, this theory might seem more plausible. Experience in acute yellow atrophy of the liver has shown that under some circumstances a parenchymatous degeneration of the liver without any biliary obstruction may be the cause of a reabsorption icterus, so that it does not seem impossible that a similar process, which is not so well understood, may be the cause of the icterus in pneumonia, pyemia and yellow fever. Liebermeister created an expression to embody this idea in the term "acatectic" icterus (*κατέχειν*, to hold back) or *diffusion icterus.*

The term *hematogenous icterus* was formerly applied to all these cases of obscure jaundice as well as to that found after poisoning by ether, chloral, chloroform, potassium chlorate, arsenious acid or toluylendiamin. It was assumed that the biliary or other pigment was formed in the circulatory system from the coloring-matter of the blood, more especially because most of these poisons are capable of destroying the red corpuscles. More accurate experiments in the poisoning of arsenious acid and toluylendiamin have proved that the yellow pigment is formed from an alteration of the hemoglobin of the red corpuscles into bile pigment; but within the liver, not in the blood itself. This excessive accumulation of biliary pigment can be demonstrated microscopically in the capillary bile ducts, and a part of it is absorbed. Again, it was shown that in some cases of poisoning the destruction of red corpuscles was so extensive that the liver was incapable of altering all the hemoglobin to bile pigment, so that a part of it had to be eliminated as such in the urine. These results explain the connection so frequently observed between icterus and other cases of hemoglobinuria. It is therefore advisable to distinguish from the *diffusion icterus* just described all these cases of hematoheptogenous or pleiochromic icterus, and to include under the latter term only those in which we can demonstrate a destruction of the red corpuscles, or a hemoglobinuria.

This modern conception of hematoheptogenous jaundice has obviated the necessity which formerly existed of distinguishing between hematogenous and heptogenous jaundice by the presence of biliary acids in the latter and their absence in the former. Such a distinction was, however, unnecessary, because cases of obstructive jaundice sometimes present no biliary acids in the urine, and, conversely, biliary acids are sometimes found in perfectly healthy individuals.

So far we have assumed that the pigmentation which occasions jaundice is real bile pigment—*i. e., bilirubin.* In most cases this supposition is correct, for bilirubin can be demonstrated in the urine. But there are numerous other cases in which only *urobilin* (hydrobilirubin) can be found. The latter have been designated as *urobilin icterus* —*i. e.*, icterus due to the deposition of the urobilin in the skin. But it is probable that such an assumption is unnecessary, and that as a matter of fact the urobilin excreted in the urine is derived from bilirubin in the tissues: for, in the first place, urobilin possesses a very much slighter coloring power than bilirubin; in the second place, the serum in cases of urobilin icterus contains bilirubin; in the third place, a considerable amount of urobilin may be found in the urine of a healthy person who does not show a trace of jaundice; and, finally, Leube has demonstrated bilirubin in the sweat of patients with so-called urobilin icterus. The term should therefore be limited to cases in which urobilin alone is excreted in the urine, whereas the supposition that any yellowish discoloration of the skin can arise from urobilin is probably erroneous. Such a type of icterus is generally of a very mild grade. It is observed especially in cirrhosis of the liver and in the early stages of ordinary jaundice.

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ABNORMAL PIGMENTATION OF THE SKIN.

Pigmentation of the skin, as is well known, varies a great deal within physiologic limits. It is most pronounced in brunets, and more marked upon the exposed body parts of anyone—*e. g.*, face and hands, as well as about the genitals and the anus, in the axillæ, on the nipples, and in the linea alba. During pregnancy the pigmentation is generally markedly increased about the areolæ of the nipples and in the linea alba, and, besides, areas of irregular mottling may appear upon the face or other body parts—the so-called "chloasma uterinum" (chloasma gravidarum, masque des femmes enceintes).

Ephelides or *freckles* can scarcely be called pathologic pigmentation. Their distribution upon the face and hands, especially in blonds, and their frequent disappearance during the winter, is well known to everyone.

Itching cutaneous diseases often leave behind a permanent pigmentation, due to the scratching. The characteristic arrangement of this pigmentation in streaks, as well as the history, always points to its origin. It is often decidedly marked between the shoulder blades in individuals who have suffered from body lice, because these insects seem to have a preference for the folds of the shirt in this region. The so-called "*vagabonds' disease*," sometimes presenting a very dark-brown color (due to serious and persistent investment with all sorts of vermin and constant scratching, as well as partially to accumulation of dirt), might in rare instances be confused with Addison's disease.

Melanosarcoma, and especially *melanosarcomatosis*, is accompanied by a diffuse gray to black discoloration of the skin, and by the excretion of melanin in the urine. (See Examination of the Urine.)

Pulmonary tuberculosis is sometimes associated with a decided brownish coloration of the face or of the entire body. *Measles* occasionally leaves behind traces of its existence, in the shape of brownish pigmented spots, whose form and arrangement are sufficiently characteristic for recognition. *Mustard plasters, blisters, Baunscheidtismus*, produce a localized pigmentation which is sometimes permanent.

Morbus Addisoni causes a smoky-gray to a bronze discoloration of the skin, which is the most important diagnostic symptom. Other associated symptoms are: well-marked digestive disorders—*e. g.*, dyspepsia, vomiting, diarrhea; certain nervous symptoms; and a gradually increasing cachexia. The pigmentation usually appears first upon areas where the pigment of the skin is normally intensified, such as the face, hands, axillæ, etc., also wherever there is any friction upon the skin, such as the waist-line or around the neck. At first a pale smoky gray, the discoloration deepens to a bronze brown, resembling the color of a mulatto, and tiny spots of an intense brown or even black color are observed scattered over this uniformly brown background. The mucous membranes are involved as well as the skin, and although not so commonly observed in the conjunctivæ, a few grayish patches upon the buccal membrane, especially just inside the lips and cheeks, are rather characteristic of Addison's disease. In well-marked examples of this

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disease the only parts exempt from pigmentation are the palms of the hands, the soles of the feet, and the nails. To distinguish this affection from *melasicterus*, the urine should be tested for bile pigment; it is invariably present in the latter. The mucous membranes of the mouth should also be examined for pigmentation spots; they are commonly present in the former. The patient's history and his general condition will clinch the diagnosis. The pigmentation which is occasionally observed in pulmonary tuberculosis may be a source of error, especially since patients afflicted with Addison's disease often suffer from pulmonary tuberculosis as well. The selection of the mucous membranes, the progressive nature of the pigmentation, as well as the entire symptom-complex in morbus Addisoni, usually facilitate a distinction.

It is well to caution the reader that, although this pigmentation of the mucous membranes is one of the most characteristic signs in morbus Addisoni, an identical appearance is sometimes observed in perfectly healthy individuals.

Hepatic cirrhosis and some of the other liver diseases occasionally exhibit a peculiar brownish discoloration of the skin, which differs from icterus in being rather more of a dirty-gray shade and somewhat spotted in arrangement. The absence of bile pigment in the urine and of a yellowish tinge in the conjunctivæ will prevent any mistake. The cause of this pigmentation is as yet undetermined, but since the description of bronzed diabetes its occurrence has acquired a certain importance. Diabète bronzée, first described by the French, presents a combination of diabetes, hepatic cirrhosis, and pigmentation of the skin. In the cases observed by the author there was no pigmentation of the mucous membranes, nor any of the intense brownish-black spots of pigment so characteristic of Addison's disease. The presence of sugar in the urine, the demonstration of cirrhosis of the liver without icterus, and the lack of the characteristic symptom-complex of Addison's disease will ordinarily very readily determine a diagnosis.

The continued administration of silver or arsenic preparations is sometimes responsible for a dark pigmentation of the skin. *Argyria* depends upon the deposition of metallic silver, not only in the internal organs, but also in the skin and mucous membranes. It may look like Addison's disease, but is not associated with any symptoms of illness. Besides, the nails are discolored as well as the rest of the integument. To-day silver nitrate is employed so little in the therapy of nervous diseases that this form of pigmentation is of very rare occurrence. *Arsenic melanosis* is very much more difficult to distinguish from Addison's disease, for the small dark spots scattered about in the diffuse pigmentation, the involvement of the mucous membranes of the mouth, and, in fact, all the peculiarities of Addison's disease have been observed to follow the persistent use of arsenic. The pigmentation itself seems to be derived from the blood-coloring matter. A further difficulty in differential diagnosis depends upon the fact that the arsenic has generally been employed for some cachectic condition—*e. g.*, pernicious anemia—so that the accompanying history will not always solve the difficulty. If the pigmentation diminishes after the arsenic has been stopped, as in a

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case observed by the author, the diagnosis could be made with probability. We must acknowledge, however, that even very small doses of arsenic sometimes cause arsenic melanosis, and that stopping the drug does not always diminish the pigmentation.

The reader is referred to text-books upon skin diseases for the consideration of the numerous cutaneous affections accompanied by pigmentation—*e. g.*, *nevus pigmentosus*, *lentigo*, *albinismus partialis* and *universalis*, *vitiligo* and *poliosis*.

MOISTURE OF THE SKIN; SWEAT EXCRETION.

An abnormally dry skin will be observed in any condition where the amount of water absorbed by the digestive organs is limited, or where a large amount of water is removed from the body in some other way than by means of the sweat glands—*e. g.*, profuse diarrhea, excessive vomiting, digestive diseases with diminished absorption, diabetes mellitus and insipidus, chronic nephritis with polyuria, marked edema of

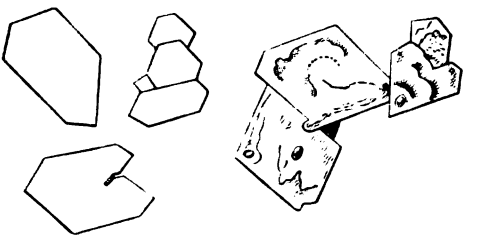

FIG. 13.—Crystals of nitrate and oxalate of urea obtained from deposits upon the skin (after Leube).

the skin, etc. An increased production of sweat is observed in certain fevers, especially in acute articular rheumatism, acute nephritis, and epidemic sudamina.

The excretion of sweat is one of nature's methods of cooling the body, both under physiologic and under pathologic conditions. When the temperature in any febrile affection drops rapidly, whether spontaneously or under the influence of drugs, a profuse sweat usually accompanies the fall—*e. g.*, at the crisis of pneumonia, at the drop of temperature in malaria, and during the third week of typhoid fever, when the temperature begins to remit. The hectic sweats of phthisis, which are so annoying and prognostically serious, coincide with the fall of temperature. They usually occur at night or in the early morning. In some patients, to be sure, they are independent of any fever and, just as is the increased pulse-rate, are merely a symptom of tuberculous intoxication. They are generally associated with a sense of considerable weakness, probably connected with the vascular relaxation from the loss of water, for copious draughts of water sometimes counteract it.

We may mention the sweating produced by pilocarpin, by ammonium acetate, and by most antipyretic measures, by hot baths and hot drinks. Salvia, atropin, agaricin, camphoric acid and sulphonal check the sweat secretion.

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In certain types of nephritis, interference in renal activity is associated with an increased production of sweat, and sometimes with the deposition of crystalline scales of urea upon the skin (Fig. 13), particularly of the face. To recognize these crystals, the deposit should be scraped off upon a glass slide, dissolved in dilute nitric or oxalic acid, and then allowed to evaporate. Crystals of sodium chlorid are often found instead of urea. As a general rule, especially with much edema, nephritis cases are associated with a diminution in the sweat production.

The sweat in jaundice may be colored yellow. In other conditions different colors have been noted, although not explained (chromidrosis). Blue sweat has been described, and is probably due to the growth of the Bacillus pyocyaneus in the skin. Naturally, variously colored underclothes may be a cause of coloring the sweat, especially since the use of anilin dyes has become so universal. Bloody sweat (hemidrosis) is exceedingly rare, though it has undoubtedly been observed.

SWELLING AND EDEMA OF THE CUTANEOUS AND SUBCUTANEOUS TISSUES.

Swelling or turgidity of the skin depends upon an increase in the amount of fluid contained in its tissue—*i. e.*, of the blood and lymph. *Increased turgidity* accompanies plethora, fever, and all conditions associated with an increased supply of blood to the skin, or with circulatory excitement. *Diminished turgidity* accompanies all conditions associated with a diminished amount of fluid in the skin—*e. g.*, cachexia, and an impoverishment of general watery elements of the body. *Increased turgidity* presents an increased fulness, a rounded outline of the surface; it obliterates the lines, especially of the face, and is generally combined with a deepening of the fleshy tint. *Diminished turgidity* is characterized by sharper outlines of the parts and by a pallor. With the poverty in fluid the skin loses some of its elasticity, and the wrinkles and lines of the face are emphasized.

Anasarca, general dropsy or *edema of the skin*, means the pathologic increase of fluid in the lymph spaces and subcutaneous tissues. The difference between edema and turgidity is one of degree. Despite the increase of fluid, the elasticity of the skin is diminished in edema, as is evidenced not by the wrinkles, but by the so-called pitting of the skin to pressure.

Edema always indicates an obstruction to the flow of lymph, with some lack of proportion between its influx and exit, so that an equilibrium is obtained only after the high pressure has distended the meshes of the tissues.

ALTERATIONS IN THE CUTANEOUS TURGIDITY OF THE SKIN.

Almost all the appearances which the laity, or we as physicians, designate by the terms "run down," "worn out," etc., depend upon alterations in the turgidity of the skin. Worry, excessive fatigue, fever, collapse, exophthalmic goiter, all these, which mar the beauty of a face, depend upon alterations in the turgidity of the skin. In emaciation,

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pallor, cyanosis, edema, there is more than turgidity responsible for the appearances. Diminution in turgidity plus depression of the mimic faculty is responsible for the characteristic facial expressions of the *moribund*, and for the so-called *facies Hippocratica* accompanying intestinal obstruction, peritonitis, cholera, etc. In the latter the features seem sunken; the wrinkles and lines of the face more pronounced; the nose sharp; the eyes fallen deep into their sockets; the skin is cool, even cold, and damp with cold sweat; while the color is pale and dusky with cyanosis. In diminished turgidity the wrinkles are very plainly marked on the trunk, especially in cholera morbus or "cholera infantum," where the organism has been deprived of so much water. Oftentimes in cachexia, where the subcutaneous fat is also diminished, the wrinkles become even more marked. A certain grade of diminished turgidity is a physiologic accompaniment of advanced age and senility.

EDEMA.

Edema presents a plump swelling, and a tense, shiny appearance of the surface of the skin; it obliterates the wrinkles, the hollows, and the normal bony prominences. When the edema suddenly disappears, the epidermis exhibits fine lines and creases and perhaps scales off. When quite fresh, edema often imparts an increased transparency to the skin. Over certain regions, especially the thighs and abdomen, transparent lines or streaks may appear, following the grain of the skin and resembling striæ gravidarum. These lines may remain permanently, even after the disappearance of the edema. They are due to a stretching and distention of the connective tissues and lymph spaces. Very pronounced edema, especially over areas exposed to considerable mechanical irritation, may be associated with the formation of blebs and blisters in the external layers of the epidermis. If these burst or are opened artificially a thin fluid will ooze from microscopic openings of the inner surface, sometimes persisting quite indefinitely. As is natural, the danger of infecting these tiny lacerations is considerable, and, of course, serious.

Edematous skin is usually pale because the blood-vessels are compressed by the pressure of the lymph, and the same amount of blood must be spread over a greater area. Inflammatory edema and edema associated with cyanosis are exceptions to this rule.

Pitting of the skin is quite essential for the diagnosis of edema. It is due to a loss of the cutaneous elasticity, from the distention of the meshes of the tissues by the lymph. Where the skin is very elastic and easily stretched, as on the face or prepuce, the imprint of the finger may vanish almost immediately. Children's skin is so much more elastic than adults' that, even over other parts, pitting may sometimes be wanting. Again, brawny edema which has persisted for some time may pit with difficulty, because there is a certain amount of connective-tissue growth. We notice this particularly in inflammatory edema and in the chronic edema of the lower extremities, which is sometimes converted to elephantiasis.

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Edema may be classified etiologically into four varieties:

1. Edema from venous obstruction, where it may be combined with other transudations, especially into the serous cavities.

2. Edema from hydremic plethora, where hydrops may be present elsewhere.

3. Edema from inflammation.

4. Edema from angioneurotic disturbances.

Obstructive Edema, or Edema from Congestion.—It is by no means clear whether the decisive factor in producing this type of edema is a diminished power of lymph absorption upon the part of the beginning veinules, due to an excess of pressure in the veins (*e. g.*, local congestion), or whether it is an excessive formation of lymph, due to an increased capillary pressure. In any general circulatory congestion we must also take into account the obstruction in the lymphatic circulation occasioned by the back pressure in the large lymphatic ducts which empty into venous trunks. The relations between these different factors in obstructive edema, and the influence of prolonged impairment of the vessel-walls by the disturbed circulation, merit further research.

Any affection of the heart or of the lungs which will produce cyanosis from congestion may also produce obstructive edema (see p. 41). The edema begins and becomes most intense in the body parts most remote from the heart and most influenced by gravity—*e. g.*, the hands, the feet, and, in the recumbent posture, the loins. It only appears very late in the face except under some conditions, not very well understood, which probably depend upon vasomotor phenomena. The influence of gravity upon the distribution of the edema becomes very evident with change of posture—*e. g.*, ambulatory patients will suffer from edematous feet; but upon going to bed the edema soon disappears from the extremities and appears in the region of the loins. Lying upon one side for a considerable time will distribute the edema to that side of the body, and, if a hydrothorax be present, will make it more marked upon that side. When the obstruction is local the edema will, of course, be confined to the region supplied by the obstructed veins.

A fluid exudation in the abdominal cavity, due to portal obstruction (cirrhosis, thrombosis) or to a chronic affection of the peritoneum (tuberculosis), will sometimes compress the vena cava inferior or the common iliac veins, and so give rise to an edema of the lower extremities. To decide the true nature of this symptom-complex is often very difficult, for both ascites and edema of the lower extremities may be co-ordinated sequences of some general obstruction located in the thorax. The onset will sometimes permit a differentiation. If the abdomen swelled before the legs, the cause will undoubtedly be one of the above-mentioned affections of the abdominal cavity; if the legs swelled before the abdomen, the cause will undoubtedly be some general circulatory disturbance.

Hydremic Edema.—Magnus' experimental researches[1] indicate

[1] *Arch. f. exper. Path.*, vol. xlii., 1899.

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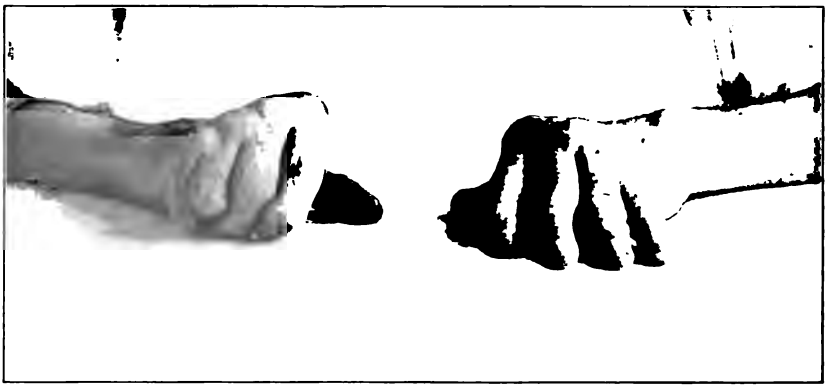
PLATE 1.

Haygarth's nodosities. Case of arthritis deformans (Dr. J. M. Jackson, Massachusetts General Hospital).

Heberden's nodes in arthritis deformans (New York City Hospital).



Heberden's nodes and ulnar deformity. Case of arthritis deformans (New York City Hospital).

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that hydremic plethora—*i. e.*, an increased volume of the blood due to retention of its water—may be the cause (although not the only one) of edema and of other transudations if the vessel-walls are coincidently damaged. This latter condition occurs in renal disease from the retention of the urinary solids. In this sense much of the edema of renal affections, and the edema occurring in chronic wasting diseases, and in the anemias of the most variable type are all to be attributed to hydremic plethora from retention of water. Nevertheless, circulatory disturbances are so often combined with these diseases that the edema must then depend partly upon the resulting venous congestion as well as upon the hydremic plethora and the injury to the vessels. As a result of experimental investigations we may say that pure hydremia—*i. e.*, a watery blood without any increase in its volume or without injury to the vessel-walls—does not cause edema.

Hydremic plethoric edema, as contrasted with the variety due to congestion, is practically independent of the influence of gravity and of the distance of the affected part from the heart. Its location seems to depend upon some peculiar arrangement of the lymphatics in the different parts of the body. Nephritic edema (in acute and subacute nephritis) is a typical example of this selective action, appearing generally in the face, and especially about the eyelids. Edema localized in this way is very suggestive of the diagnosis of nephritis. The more chronic types of nephritis (contracted kidney), however, rarely exhibit any edema until quite late in the disease, and then it usually begins in the feet, because the hypertrophied heart, unable to maintain the circulation against the obstacles in the kidneys and at the periphery, begins to flag. In other words, in these very chronic cases of nephritis dropsy is generally of cardiac origin.

The absence of cyanosis is another characteristic of purely hydremic edema.

Purely hydremic plethoric edema is generally very rare except in acute and subchronic cases of nephritis. Almost all cachectic conditions, such as grave anemia and carcinoma, frequently exhibit a swelling about the ankles as the first sign of a disturbance in the circulation, so that, despite the common impression that it is due to water retention, the edema is not purely hydremic, but of a mixed type.

Inflammatory Edema.—From the diagnostic point of view, the only importance inflammatory edema has is in pointing to some deep-seated inflammation. It must be attributed to the saturation of the neighborhood of an inflammatory area with the fluid components of the exudation (Samuel).

It is of more interest to the surgeon than to the clinician. Although so rarely present, it is important in suggesting the purulent nature of an underlying pleuritic effusion. In acute articular rheumatism the inflammatory edema about the joints is quite as important as the effusion in the joint itself. Inflammatory edema may aid the surgeon in recognizing a deep suppuration, as in purulent perforative appendicitis, liver abscess, etc. Here it would depend upon adhesions which connect the inflammatory focus with the skin.

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Angioneurotic Edema.—Under this heading are included certain rare types of edema of the face or of other areas of the skin. Some of them are combined with analogous conditions in the mucous membranes (laryngeal, bronchial, gastric or intestinal). Apparently idiopathic, we have as yet no clear notion of the mechanism of their origin, except that they appear and disappear suddenly, and are, therefore, credited with a dependence upon the nerves of the vessels.

A similar conception is held of the edema observed in nervous affections—*e. g.*, the edema in *polyneuritis* and the localized edema in *urticaria*. The latter consists in the formation of rapidly appearing wheals. (See Examination of the Nerves.) The blue edema of the hysteric is usually included under angioneurotic edema (see p. 42).

It is difficult to classify the general edema which occurs without nephritis in certain infectious diseases, especially in scarlet fever and diphtheria, as well as that appearing after the injection of diphtheric antitoxin, and that after the employment of potassium iodid.

EMPHYSEMA OF THE SKIN.

This is due to the presence of gas (generally air) in the meshes of the subcutaneous tissues. Upon mere inspection, emphysema of the skin resembles edema. Palpation, however, over emphysematous regions produces a peculiar crackling, audible as well as palpable. It is due to the movement of bubbles of air in the tissues. Percussion elicits a tympanitic note. (See Percussion.)

In very rare instances the gas is developed in the skin by the activity of gas-producing bacteria—*e. g.*, in *glanders* and in *malignant edema*. In most cases air penetrates the subcutaneous tissues from the rupture of some air-containing organs or from some external wound. The most frequent source is through the mediastinal connective tissue, resulting from an ulcerative destruction of the esophageal wall (carcinoma) or from a rupture of the pulmonary alveoli (following coughing or external trauma). The emphysema will then be first noted at the lower part of the neck or over the manubrium. In a patient with a tracheotomy wound, air may be forced under the skin by a fit of coughing. The chief diagnostic significance of cutaneous emphysema is its dependence upon the rupture of some air-containing viscus. If the source persists, the entire body surface may be swollen; but ordinarily the air is readily absorbed and the cutaneous emphysema disappears.

CUTANEOUS HEMORRHAGES.

Hemorrhages into the skin appear as spots of variable size, at first red, later violet to black, gradually changing to green and yellow, and finally disappearing. These changes in color are due to the alterations in the blood-pigment. In contrast to hyperemic spots, cutaneous hemorrhages do not disappear under pressure. They are not usually elevated except in certain types of *purpura*, where the epidermis may be raised like a bleb. Absorption of the hemorrhagic spots usually begins in the

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center. When small, pin-point in size, they are called *ecchymoses* or petechiæ.

Cutaneous hemorrhages occur:

1. From *trauma* (ordinary black-and-blue spots). The ecchymoses caused by *flea bites* (purpura pulicosa) belong to this group and may be mistaken for a form of purpura. They are, however, unlike purpura, found most abundantly upon the trunk. They frequently exhibit the bite-mark as a dark spot in the center of the hemorrhage, and when

FIG. 14.—Emphysema of left breast and chest-wall, due to broken ribs (Massachusetts General Hospital).

fresh they are surrounded by a hyperemic zone, the color of which will disappear upon pressure.

2. Spontaneously in *all severe cachexias and infections* which are associated with a hemorrhagic diathesis; particularly as a characteristic symptom in the various types of *purpura*, in *grave anemias*, especially in *pernicious anemia*, *leukemia* and *scurvy*, in *acute yellow atrophy* of the liver, in *phosphorus-poisoning*, in *ulcerative endocarditis*, in *pyemia*, and in the *terminal stages* of certain cases of *pulmonary tuberculosis*.

3. In the hemorrhagic forms of the *acute exanthemata: scarlet fever, measles, small-pox.* These are notoriously more serious and critical

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than the ordinary types, especially in "black small-pox," where the hemorrhage occurs in the interior of the pustules; and still more so in that rare, very fatal form, "purpura variolosa," where extensive cutaneous hemorrhages occur without the formation of any rash. But there are some cases of measles and scarlet fever which are not much more serious than the ordinary cases, although they are shown to be hemorrhagic, by the fact that the coloration of the rash does not entirely

FIG. 15.—Purpura (New York City Hospital).

disappear upon pressure, and that even into the convalescence slight remains of the rash still show as a pigmentation.

Erythema nodosum (contusiformi) not infrequently simulates bruises of the skin, presenting rather extensive cutaneous hemorrhages upon the extensor surface of the extremities.

4. From *marked venous stasis*, especially when accompanied by severe paroxysms of cough, which suddenly increase the congestion—*e. g.*, *pertussis*—where hemorrhages in the mucous membranes, particularly in the conjunctivæ, are not uncommon.

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PLATE 2.

Purpura (New York City Hospital).

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COLLATERAL CIRCULATION IN THE SKIN.

A visible distention of veins or arteries in the skin often suggests some deep-seated obstruction to the circulation—*e. g.:*

I. The collateral circulation (arterial), when the aorta is occluded at the isthmus. (See Congenital Heart Diseases.)

II. The collateral circulation (venous) upon the anterior thoracic wall. This is of some importance in diagnosing mediastinal or pulmo-

FIG. 16.—Collateral circulation in the abdominal wall (New York City Hospital).

nary tumors, which compress the big veins within the chest, especially the vena cava superior and inferior. Here the intercostal and internal mammary veins dilate and furnish a channel of communication between the two vena cavæ when either one is occluded.

III. The very striking collateral circulation in the abdominal wall caused by thrombosis of both iliac veins or of the vena cava inferior (Fig. 17). The blood from the lower extremities, and even from the kidneys, reaches the thorax by way of distended tortuous veins, which are arranged longitudinally, and are more pronounced upon the sides than upon the front of the abdomen. This selection of the sides of

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the abdomen is of some importance in distinguishing this form of obstruction from the next.

IV. The collateral circulation caused by obstruction of the portal veins (cirrhosis of the liver or portal thrombosis, Fig. 18). In the latter case it is furnished by anastomoses between the tiny veins at the root of the mesentery and those of the peritoneal covering and suspensory ligaments of the liver, even sometimes by a patent vena umbilicalis. In

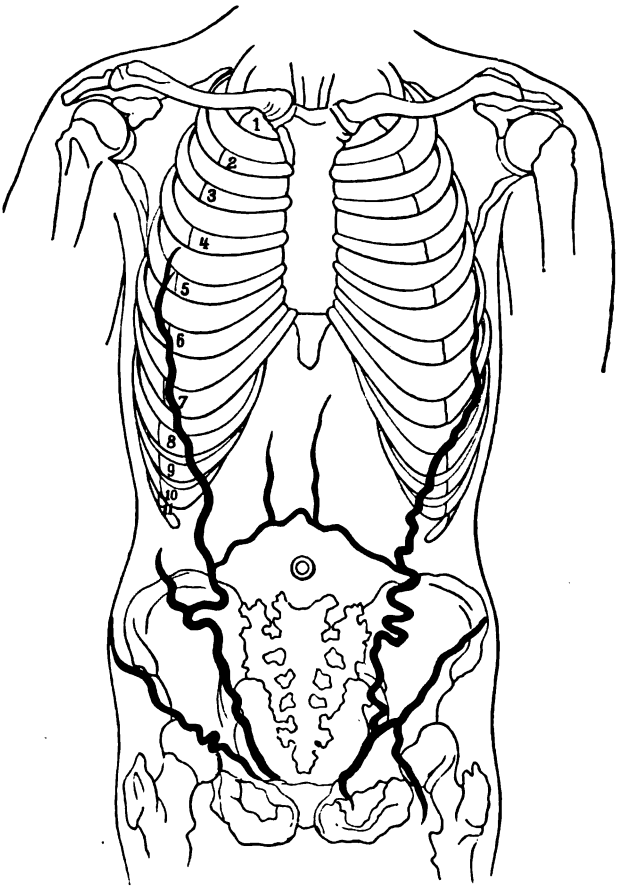

FIG. 17.—Collateral circulation in thrombosis of the vena cava inferior.

this latter type of anastomosis the distended veins of the abdominal wall are apt to be very characteristically arranged about the navel, forming the so-called "caput medusæ."

Figs. 17 and 18 illustrate the diagnostic distinction between the distention of the abdominal wall veins, due in the one case (Fig. 17) to obstruction in the vena cava, and in the other case (Fig. 18) to obstruction in the portal system. Numerous exceptions make this distinction

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less valuable for diagnostic purposes—*e. g.*, when ascites follows portal stasis the presence of the fluid in the peritoneal cavity will naturally produce a secondary obstruction in the vena cava, and the veins of the sides of the abdomen will also become distended.

In any of these cases of collateral venous circulation it is always important to determine the direction of flow of the venous blood. This

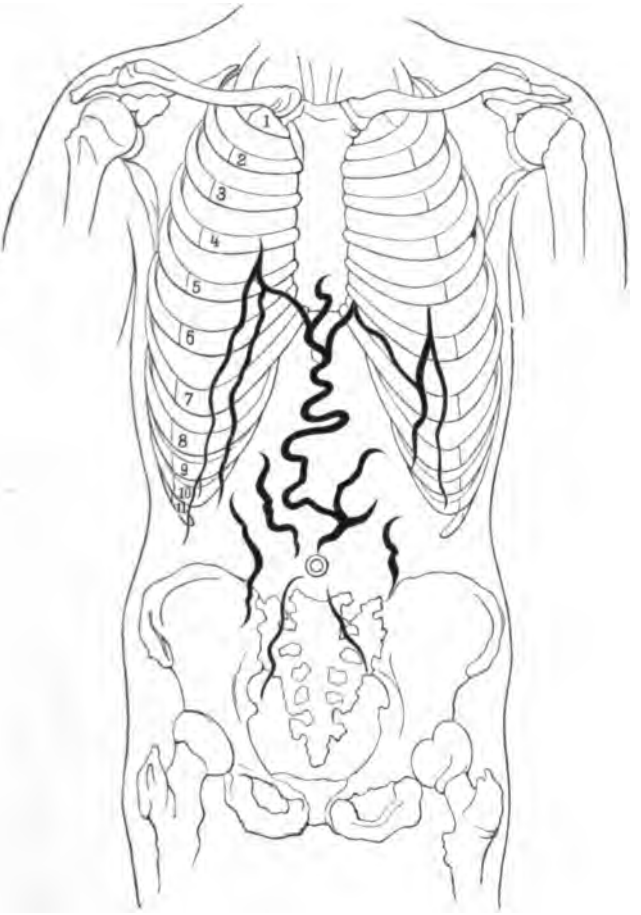

FIG. 18.—Collateral circulation in thrombosis of the portal vein or in hepatic cirrhosis.

is readily done, particularly when the valves of the veins are intact, by emptying the blood from the vein by means of pressure with the fingers, and then, when the pressure is removed, by watching, first at one end and then at the other end of the vein, to see from which direction the blood comes first. If there are no valves or if the valves are incompetent this test is valueless, because the blood would stream back with equal rapidity from either end.

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A very fine dendritic, irregular enlargement of the thoracic skin veins is often observed in all chronic affections of the lungs and pleuræ, especially when adhesions are present between the two pleural surfaces. It probably represents a collateral circulation between the lung and skin. In the very chronic forms of phthisis it is observed frequently, and then quite often in the supraspinous fossa.

Nearly twenty years ago the writer described a zone of small dilated skin veins which are arranged dendritically and extend in the form of a band about 1 to 3 cm. broad along the anterior lower border of the lung and over the superficial cardiac dulness.[1] It has no especial pathologic significance, because it is also observed in perfectly healthy individuals; but clinically it represents a rough picture of the position of the lung borders. It may be situated either within or without the pulmonary margins, but is generally nearby, unless modified in some fashion, as by pleural adhesions. Percussion-results prove that a low position of this zone corresponds to emphysema of the lungs.

It is very difficult to explain the appearance of this zone of vessels, but, without question, it is due to a difference in pressure between the inside and the outside of the chest-wall. Suppose we take the region where the zone of vessels marks the internal pulmonary hepatic boundary. Above the intrathoracic pressure is negative, and below the intra-abdominal pressure is positive, which, of course, in and of itself explains nothing. Respiration, however, alters the pressure relations (see p. 77, *et seq.*). It produces a distinctive change of shape in the chest-wall, as evidenced by the normal sinking in of the lower intercostal spaces and by the so-called diaphragm phenomenon. Both these conditions furnish an inspiratory, localized, girdle-shaped depression of the chest-wall, which must occasion a rhythmic obstruction to the venous blood-current, because the superficial veins of the part are compressed by the external atmospheric pressure. Now it is conceivable that this rhythmic constriction may give rise to an area of small distended veins. Perhaps a similar explanation would account for the formation of the zone which surrounds the heart, because evidently similar differences in pressure exist at that portion of the chest-wall against which the heart beats and at that portion against which the lung rests. In fact, one oftentimes sees an inspiratory depression surrounding the cardiac area, quite similar to that about the margin of the lung. Local variations in pressure caused by the maximum distention of the heart, and its minimum size during systole, would also influence the circulation in the thoracic wall. Paroxysms of coughing were formerly considered to be the most important, if not the sole, cause of this vascular distention. They probably do exert a considerable influence, because during a cough the strong positive pressure from the inside causes a marked congestion of the subpleural and subperitoneal veins. This in turn reacts upon certain cutaneous veins which are connected with the subserous veins, and the congestion will be marked along the lines of attachment of the diaphragm. The latter, to a certain extent, acts as a watershed for the venous blood, flowing upward and downward. Everyone with a cough does not present this zone of veins, so that we must assume as another factor in its origin a certain indefinite abnormal distensibility of the smallest vessels. As a matter of fact, it is found oftenest in such patients as exhibit a similar tendency to the formation of dendritic venous dilatations in other parts of the skin.

[1] Ueber das Vorkommen und die diagnostische Bedeutung einer Zone ektasierter feinster Hautgefässe in der Nähe der unteren Lungengrenze, *Corr.-Bl. f. Schw. Aerzte*, 1885.

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TROPHIC AFFECTIONS OF THE SKIN.

The so-called "decubitus" and the trophic disturbances accompanying nervous affections will be considered later under Examination of the Nervous System.

Clubbed fingers, the peculiar swellings of the terminal phalanges of the fingers, are observed in congenital heart disease, in chronic pulmonary diseases, most frequently in bronchiectasis and empyema (hence the name empyema fingers), and less often in phthisis. The deformity may develop within a few weeks, and then disappear again when the causal disease has decidedly improved. In the more chronic cases even

FIG. 19.—Clubbed fingers: Chronic phthisis (New York City Hospital).

the bones share in the deformities. The tendons are also affected, though less decidedly. As yet we have little accurate information in regard to the mode of origin.

ACUTE EXANTHEMATA; CUTANEOUS DISEASES; DERMATITIS MEDICAMENTOSA.

Although the scope of this book does not include the subject of skin diseases and the acute exanthemata, it seems fitting, from the standpoint of diagnosis, to describe the rash of typhoid fever, herpes febrilis, miliaria, and the medicinal eruptions.

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ROSE SPOTS (Roseola).

These are small, round, very slightly elevated, hyperemic, rose-red spots, varying in size from that of a pin-head to that of a small pea. They appear after the beginning of the second week of typhoid fever. They are sparingly scattered over the abdomen, more rarely over the chest, back, and lower extremities. Though most marked at the height of the fever, they may persist throughout the whole duration, and even after convalescence. Relapses are frequently accompanied by a fresh

FIG. 20.—Skiagraph of hand of patient with clubbed fingers (New York City Hospital).

crop of spots. Their hyperemic nature is shown by their immediate disappearance under the pressure of the finger, only to reappear instantly when the pressure is removed. From the rash of acne, rose spots are differentiated by the absence of a decided center. This tip in acne is caused by some cutaneous gland or hair follicle, or by a suppuration starting in one of them. The opening of a cutaneous gland or hair follicle only rarely is found occupying the center of a rose spot, or a vesicle tipping the center. In doubt we can generally find in a case of typhoid fever other absolutely characteristic rose spots elsewhere upon

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PLATE 3.

Typhoid spots (rose spots) in the region of the umbilicus (natural size).

Rose spots in a case of typhoid fever, showing distribution upon the trunk. The three spots upon the face are rather unusual.

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the abdomen. The spots usually pass through a cyclic development, ripening in the course of two or three days and then fading away, while in the meantime new spots have sprung up in other places. In a doubtful case it is often advisable to mark out with a skin pencil the few spots that appear, in order to observe them carefully at each visit. For although rose spots may never appear during the entire course of some few cases of typhoid fever, the positive evidence of this peculiar rash is perhaps the safest diagnostic sign of the disease. (See Plate 3.)

HERPES FEBRILIS.

This consists of a group of vesicles, ½ to 2 cm. in diameter, situated upon a slightly inflamed base. The watery content of each vesicle rapidly becomes purulent and turbid, and the vesicle then either bursts or dries up, leaving an irregular scaly crust upon a somewhat reddened

FIG. 21.—Herpes cervicalis: Cerebrospinal meningitis (Dr. E. G. Cutler, Massachusetts General Hospital).

background. Although they may appear anywhere upon the face, nose, cheek, lip, or ear, they develop most frequently on the outer edge of the lip. According to their position they are called herpes labialis, facialis, nasalis, frontalis, and auricularis. The eruption generally appears at the beginning of febrile diseases; rarely in the latter stages. It is most common in those fevers with a rapid onset, particularly in croupous pneumonia.

[Howard[1] has very recently been able to demonstrate in two cases of labial and nasal herpes and herpes of the body occurring in acute lobar pneumonia pathologic changes in the posterior-root ganglia, the Gasserian ganglia, and the skin. The changes are apparently identical

[1] Howard, *Am. Jour. Med. Sci.*, Feb., 1903.

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with those shown by Head and Carpenter[1] to be invariably associated with *herpes zoster*.

From evidence available at the present date, it is probable that lesions identical in character and similarly localized are present in the herpes of other infectious diseases, and in the light of our present knowledge, that the lesions of the sensory ganglia, nerves, and skin are due to the action of the soluble toxins of various micro-organisms.—ED.]

Herpes is very rarely observed in typhoid fever. There is a certain type of slight ephemeral fever, with nothing objective to be discovered except the increased temperature and herpes, which is termed *febris herpetica*. Herpes, as a rule, is considered a favorable prognostic sign. Our experience in the use of streptococcus toxin in the treatment of malignant tumors proves that herpes febrilis depends upon a toxic action. It is common in *malaria*, *cerebrospinal fever*, and *meat-poisoning;* and therefore a sign of some value in the diagnosis of these affections from typhoid.

MILIARIA (PRICKLY HEAT); SUDAMINA.

Under this head are included various eruptions of tiny vesicles developing most frequently upon the abdomen and chest and usually associated with profuse perspiration. It is generally supposed that this eruption is due to the plugging of the orifices of the sweat glands with swollen epithelium and to the formation of small retention cysts, some of which may be associated with a slight inflammatory reaction in the neighborhood. Three main types are to be distinguished:

Miliaria crystallina (sudamina; crystal rash): absolutely transparent vesicles, resembling a dew drop and without reddened base.

Miliaria alba: vesicles with slightly turbid contents upon a faintly reddened base.

Miliaria rubra: small red papules with a faintly developed vesicle in the center. This type is apt to itch.

Miliaria vesicles are generally situated close together.

DERMATITIS MEDICAMENTOSA (DRUG ERUPTION).

Numerous medicinal agents have the peculiarity of exciting in certain individuals eruptions which resemble urticaria, measles or even scarlet fever. The rash generally fades promptly and disappears when the drug is omitted. Examples of such drugs are most of the antipyretics, especially antipyrin, antifebrin, phenacetin; not infrequently sodium salicylate; many preparations of balsams—*e. g.*, balsam of copabia; and mercury used externally, or, in rare cases, internally. The injection of antitoxin produces an eruption resembling urticaria, measles or scarlet fever very closely. Iodid of potash is apt to produce a rash which may closely resemble erythema multiformi or to bring out a purulent acne-like eruption which may be confounded with syphilis or small-pox. The prolonged administration of considerable doses of bromid of potash frequently causes an eruption very much like acne. It is usually localized

[1] Head and Carpenter, *Brain*, 1900, Part III.

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upon the face and chest, and an experienced observer generally distinguishes it by its marked nodular infiltration and bluish appearance. It may develop into suppurating sores covered with crusts which resemble chancroids, and which persist in the resemblance even microscopically by exhibiting a marked overgrowth of epithelium.

OTHER CUTANEOUS MANIFESTATIONS IMPORTANT FROM THE DIAGNOSTIC STANDPOINT.

Striæ.—The striæ caused by the stretching of the skin in edema, their resemblance to striæ gravidarium, and their persistence for some time after the edema has disappeared or even permanently, have already been noted on page 49. Similar striæ may appear from the rapid accumulation or disappearance of the subcutaneous fat. Any cause which rapidly increases the size of the abdomen, such as pregnancy or the growth of tumors, may produce these striæ.

Desquamation.—In cachexia and emaciated conditions one frequently observes a diffuse bran-like scaliness covering the skin of the trunk and extremities (pityriasis tabescentium). A characteristic lamellar desquamation of the skin, usually most conspicuous upon the palmar surfaces of the feet and hands, occurs after scarlet fever; a characteristic bran-like desquamation, after measles; a more crust-like desquamation, after small-pox; and a flaky desquamation, after erysipelas.

Furunculosis, as a complication of some general disorder, such as diabetes, is of diagnostic interest to clinicians; otherwise, it concerns only skin specialists.

Scars.—The forms of various scars may be of some importance in considering the past history of a patient—*e. g.*, marks of vaccination, of small-pox, of furunculosis, of carbuncle, of lupus, of inguinal buboes, of tubercular glands, and the bean-shaped scars of serpiginous syphilides. Unfortunately even a slight scar very rarely persists to show the site of the primary sore of syphilis. Surgical operations, various therapeutic measures, such as moxas, cautery, venesections, leeches, Baunscheidtismus, and epispastics, and wet-cupping leave behind permanent characteristic scars. They may be important in marking the dates of previous illness.

DETERMINATION OF THE BODY TEMPERATURE.

PHYSICIANS in ancient times recognized as a principal symptom of fever an increase in the temperature of the blood. But the amount of fever was measured by the acceleration of the pulse. About the middle of the last century Traube, v. Bärensprung, and Wunderlich perfected the methods for taking the body temperature, considering it one of the essential points to be observed in an examination. Since

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their time the tendency has been to consider that an increase of the body temperature is the essential sign of fever.

The classical symptoms of fever include, in addition: weakness, malaise, anorexia, thirst, digestive and psychic disorders, rapid breathing, alteration in the amount of urinary excretion, and especially "consumption of the body."

Some, though not all, of the above-mentioned symptoms may be produced by an artificial overheating of the body. Certain of them, however, by no means run parallel to the increase of temperature; for sometimes, either with or without antipyretics, the temperature may drop without any especial improvement in the other symptoms. Hence, an increase of body temperature is the most important and constant accompaniment of what we call fever, and to-day the terms are used interchangeably. But still we must not neglect the associated phenomena of the febrile system-complex in determining the severity of an attack, because they are partly independent of the temperature, and are sometimes much more important than the amount of temperature increase.

Before the use of thermometers physicians estimated the temperature by the sense of touch. Though generally this is a perfectly satisfactory method of telling whether or not fever is present, many mistakes may arise. The hand appreciates only the temperature of the skin. This is not always parallel to the internal temperature of the body or of the blood; because the skin temperature evidently depends not only upon the temperature of the blood, but also upon the amount of blood in the skin at a given time, as well as upon the conditions of heat radiation. For example, during a chill the cutaneous temperature is sometimes diminished as a result of a contraction of the peripheral vessels, while the blood temperature, as shown by the thermometer, is increased.[1] Conversely, the cutaneous temperature of perspiring patients seems increased, provided evaporation and subsequent cooling is prevented by their being wrapped up, because the skin contains an increased amount of blood. Yet the internal temperature need not be raised. These possible sources of error make it plain that thermometers are essential for the accurate determination of the temperature relations. Nevertheless, palpation of the skin is of some value in disclosing the condition of the cutaneous circulation. It is oftentimes accurate, provided that evaporation, chill, and profuse perspiration can be excluded. Perhaps the best place for such palpation, when the patient is in bed, is the back; for there the temperature must be nearly the same as that of the blood. In spite of a high temperature, the skin need not feel very hot to the physician's hand if the room temperature is low and the skin, as well as the blood itself, has been cooled by the surrounding air. This is a point especially to be remembered for the diagnosis of ambulatory typhoid.

[1] According to Liebermeister, fever depends upon an alteration in the temperature regulation. Either heat production or heat radiation, one or both, are affected. This view has been supported by more recent experiments (Hildebrandt, Stern, and others). During a chill it is supposed that the cutaneous temperature is not diminished actually, but only relatively, compared with the internal temperature, and that the cutaneous sensibility in response to this relation replies by the excitation of a sensation of chilliness.

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THE THERMOMETER.

The thermometer carried by practising physicians is a mercury instrument, subdivided in England and America, according to the Fahrenheit scale, from 95° to 115°, and on the Continent, according to the Celsius scale, from 20° to 45°. To convert Fahrenheit into Celsius and *vice versâ*:

A° Celsius = ($\frac{9}{5}$ a + 32)° Fahrenheit, or

A° Fahrenheit = (a — 32) $\frac{5}{9}$° Celsius.

A variety of small clinical thermometers can be obtained in America or in England (perhaps the best are the English make). They are very accurate, so that an explanation of the method of correcting and tabulating their errors is hardly necessary. [The one-minute Hick's maximum clinical thermometer, furnished with a prismatic lens to magnify the column of mercury, is perfectly satisfactory.—ED.] The cylindric is ordinarily more convenient than the old-fashioned spheric bulb. The so-called "maximum" thermometers in most common use to-day require vigorous shaking to push the mercury down below the normal point. Then the mercury remains at whatever height it may reach until shaken down again.

METHOD OF TAKING THE TEMPERATURE.

The temperature is ordinarily taken by placing the bulb of the thermometer beneath the side of the tongue and instructing the patient to keep the lips tightly closed during the necessary interval (one to five minutes). In some cases it may be necessary to obtain the temperature in the axilla, and, whenever in doubt, in the rectum or vagina. It is hardly necessary to caution a physician to be most careful in disinfecting the thermometer after each time it is used.

In many of the hospitals in Germany they employ a large thermometer, leaving it in the axilla from fifteen to twenty minutes. With comatose, stupid or violently delirious patients or with patients suffering from severe dyspnea or from nasal obstruction sufficient to impede breathing, the temperature must be taken in the rectum or vagina.

[The ordinary one-minute clinical thermometer is sufficiently accurate when used in the mouth, and absolutely so in the rectum.—ED.]

In any case the temperature should be measured at least twice a day, and under many conditions every two to four hours. When the morning and evening temperatures are taken, the morning commonly represents the temperature between 7 and 9 o'clock, and the evening between 4 and 6 o'clock, which furnishes a practical maximum and minimum without disturbing the patient's sleep (see pp. 66 and 68). The temperature is ordinarily indicated upon temperature charts ruled either for every two hours or merely for morning and evening. The normal line is generally printed more deeply or in another color. (See Figs. 28 and 29 for a convenient method of indicating night and day temperatures.)

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The temperature curve alone sometimes suffices to make a diagnosis with considerable certainty; as, for instance, in typhoid fever, pneumonia, chronic tuberculosis, malaria, and suppurative processes. Even a single estimation of the temperature will often throw considerable light upon the diagnosis. Simulation may be excluded if there is decided fever.

THE NORMAL BODY TEMPERATURE.

The normal temperature is 0.2° to 0.5° C. (0.4°–1° F.) higher in the rectum or vagina than in the axilla. von Bärensprung gives the following figures as the normal mean in the axilla at the various ages:

First 10 days	37.75° C. (99.95° F.).
Up to puberty	37.43° C. (99.37° F.).
15 to 20 years	37.19° C. (98.94° F.).
21 to 70 "	36.85° C. (98.93° F.).
80 years	37.26° C. (99.07° F.).

These, of course, are the daily averages. The daily variation of temperature in a normal person is shown in Fig. 22. The minimum of

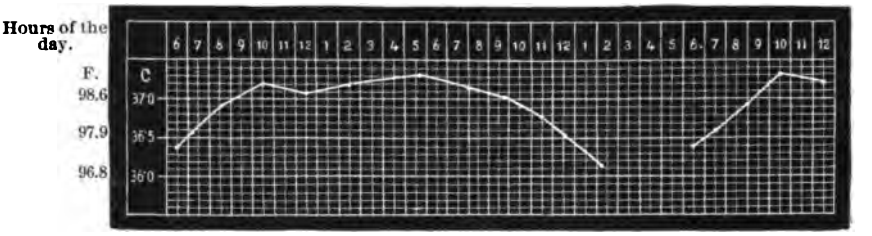

FIG. 22.—Daily curve of the normal body temperature (Liebermeister).

temperature is observed in the first few hours after midnight; its first maximum is reached during the forenoon, generally between 9 and 10 o'clock; it falls again before the midday meal, rises and reaches a second, the proper maximum, some time between 5 and 8 o'clock in the evening; it then begins to fall till the first minimum is reached. The periodicity of the normal temperature curve probably depends primarily upon the change between waking and sleeping. At least there have been numerous examples of people accustomed to reversing the day, sleeping in the daytime and working in the night, in whom the temperature variation is reversed.

Food and physical exercise influence the temperature. Mountain-climbing, for example, has raised the temperature in normal individuals as high as 40° C. (104° F.). The temperature of the external air has some effect upon the body temperature. Sometimes just before a thunder shower a slight rise has been noted in normal individuals. Liebermeister's chart shows that the daily variation amounts to 1° C. (1.8° F.), comparing the absolute maximum and minimum, but to not more than 0.5° C. (0.9° F.) comparing the difference between the morning temper-

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ature at 8 or 9 o'clock and the evening temperature at 5 o'clock.[1] An evening temperature of 37.4° C. (99.32° F.) is within physiologic limits; although in consumptives it would probably correspond to a rectal temperature of 38° C. (100.4° F.), and so indicate fever.

[Unless otherwise stated these temperatures are mouth temperatures. —ED.]

FEBRILE TEMPERATURES.

Wunderlich has computed the following fever scale:

I. Normal temperatures, 37.0°–37.4° C. (98.6°–99.3° F.).
II. Subfebrile temperatures, 37.4°–38.0° C. (99.3°–100.4° F.).
III. Febrile temperature:
  (*a*) Slight fever, 38.0°–38.4° C. (100.4°–101.1° F.).
  (*b*) Moderate fever, 38.5–39.0° C. (101.3°–102.2° F.) in the morning to 39.5° C. (103.1° F.) in the evening.
  (*c*) Considerable fever, up to 39.5° C. (103.1° F.) in the morning to 40.5° C. (104.9° F.) in the evening.
  (*d*) High fever, above 39.5° C. (103.1° F.) in the morning and above 40.5° C. 104.9° F.) in the evening.

Hyperpyrexia.—Very unusual temperatures, 41° or 42° C. (105.8° or 107.6° F.) are spoken of as hyperpyrexia. Teale's case (injury to the spine and recovery) is the highest recorded temperature, 50° C. (122° F.). At the Insel Hospital, Berne, a patient with typhoid fever, who subsequently recovered, once exhibited a temperature of 45° C. (113° F.). Similar cases are quoted in literature as medical curiosities. A temperature above 42° C. (107.6° F.) is rare to-day, because we now have at our command more efficient means of combatting fever.

Stern has recently given to the term hyperpyrexia a general pathologic significance, limiting it to those cases in which the heat regulating apparatus is insufficient, in contradistinction to febrile temperatures where the regulating apparatus is capable of preventing a further increase or an undue cooling-off. Hyperpyrexia includes, therefore, the excess of temperature added to fever by insufficient heat radiation. We do not really know in every case whether fever is injurious or beneficial; but it is evident that hyperpyrexia is a dangerous surplus of heat, and therefore to be controlled therapeutically. Stern considers that true fever may be distinguished clinically from hyperpyrexia by the fact that when the temperature of a patient with fever is reduced by means of a cold bath a reaction takes place directly (chilliness, shivering), whereas no such reaction occurs in hyperpyrexia, nor does the patient experience any discomfort. However, we may still consider almost any excessive temperature under the heading of hyperpyrexia.

PROGNOSTIC SIGNIFICANCE OF HIGH TEMPERATURES.

Although a certain significance can be attributed to the height of the temperature, we must be very guarded in making the prognosis of a disease, and not assume that every high fever is necessarily fatal. In general a typhoid fever with a high temperature curve is more severe

[1] Debcynski, ref. in Hermann's *Handbuch der Physiologie*, 1882, vol. IV., p. 323. Further, U. Mosso: Experienze fatte per invertire le oscillazioni diurne della temperatura nell'uomo sano. Laboratorio di fisiologia nella R. Università di Torino, 7th ed.

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than one with a low range, and in the same patient an increase of temperature generally coincides with some more serious condition or with a complication. The bearing of a certain height of temperature upon the prognosis varies greatly in different diseases. In malarial fever, for example, the temperature may be extremely high without rendering the prognosis more serious; or again, quite innocent throat affections in children are responsible for temperatures of 40° C. (104° F.) or more.

Unverricht[1] has published a collection of abnormally high temperatures in which, nevertheless, recovery followed.

THE FEVER COURSE.

DAILY VARIATIONS OF THE FEVER; THE FEBRILE TYPE.

The daily course of temperature in any case of fever lasting one or more days is usually much the same as in healthy individuals. If the curve pictured upon page 66 were elevated one or two degrees throughout it would correspond accurately enough to the daily variation of many a fever. But in numerous febrile diseases the maximum or minimum point may appear at a different time; for example, the maximum point may occur in the forenoon or at midday. Again, the daily variations may be much greater than in a normal individual, or in the morning the patient's temperature may be normal and in the evening elevated two or three degrees. It is therefore evident that to obtain the accurate temperature of a patient with fever we must not be content with measuring the temperature at morning and night alone, but take it at regular, shorter periods, such as once in two hours or once in four hours. An irregular fever is often first discovered by taking the temperature at an unusual time; for example, late at night. A very good indication of the amount of fever is, of course, the patient's feelings; he generally feels worse when he has most fever.

Various types are distinguished according to the variation of the fever during the day. In prolonged or *continued fevers* the daily oscillations rarely vary more than in a normal individual—*i. e.*, not more than 1° C. (1.8° F.). In *remittent fevers* the daily variation is more than 1° C. (1.8° F.). In *intermittent* or *interrupted fevers* the daily minimum is normal or below normal. Since malaria is now so commonly called intermittent fever, it is perhaps advisable to designate this type as *interrupted fever.*

COURSE OF FEVER FOR LONGER PERIODS; COURSE OF FEVER IN A RESTRICTED SENSE OF THE WORD; THE FEVER CURVE IN DIFFERENT DISEASES.

The *fever type*, meaning more particularly the daily variations, is of less diagnostic importance than the so-called course of fever or *fever curve,* including a longer period of observation.

[1] Unverricht, "Ueber das Fieber." *Sammlung klinischer Vorträge*, No. 159, p. 724, 1896, Breitkopf and Härtel.

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EPHEMERAL VARIETIES OF FEVER (SINGLE-DAY FEVERS) (FEBRICULA).

Ordinarily these are either well-known infections whose nature is shown partly by the objective examination, partly by the prevalence of an epidemic of similar cases, but which run an abnormally rapid or abortive course; or else slight infections of some unknown origin, or else are caused by temporary digestive disorders, or else due to some nervous influence, such as hysteria or mental excitement; children, as is well known, exhibit a rise of temperature from very insignificant causes; in the beginning of an ephemeral fever a diagnosis is almost impossible—so that the height that the temperature reaches will often cause apprehension.

Under this heading is included the fever which appears after *catheterization*, whose origin we do not perfectly understand, and the brief fever following *aseptic operations*. The latter is to be explained by purely psychic influences, such as anxiety as to the result of the operation, by the toxic action of the chloroform, or

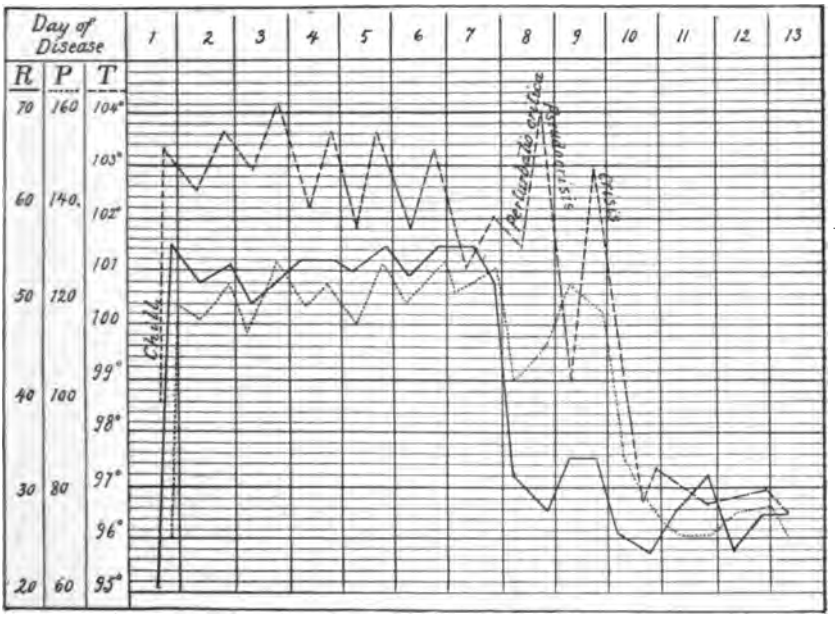

FIG. 23.—Temperature, pulse, and respiration curve in croupous pneumonia.

of the antiseptics employed, and by the absorption of the relatively harmless secretion of the wound, etc. Where the consistence of the blood is altered by some therapeutic interference, such as transfusion of blood or of salt solution, we often observe fevers of similar slight import. In such cases the origin may depend upon some slight infection or upon fermentative intoxication. In any case these rises of temperature are generally transitory and innocent.

Notwithstanding these numerous types of ephemeral fever no careful physician, in any febrile attack, will neglect a most careful search for some objective explanation or some evidence of infection.

FEVER CURVE OF CROUPOUS PNEUMONIA AND ERYSIPELAS; CRISIS AND LYSIS.

There are a number of diseases with continued fever in which the course of the temperature is sufficiently characteristic to furnish the diagnosis. Croupous pneumonia is one of these. It is generally ushered in with a chill, and within a few

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hours by a rapid rise of temperature—39° to 40° C. (102.2°–104° F.)—although shortly before the chill the patient had been feeling apparently well. The fever persists with but slight variation for several days (5 to 9 days), and then subsides

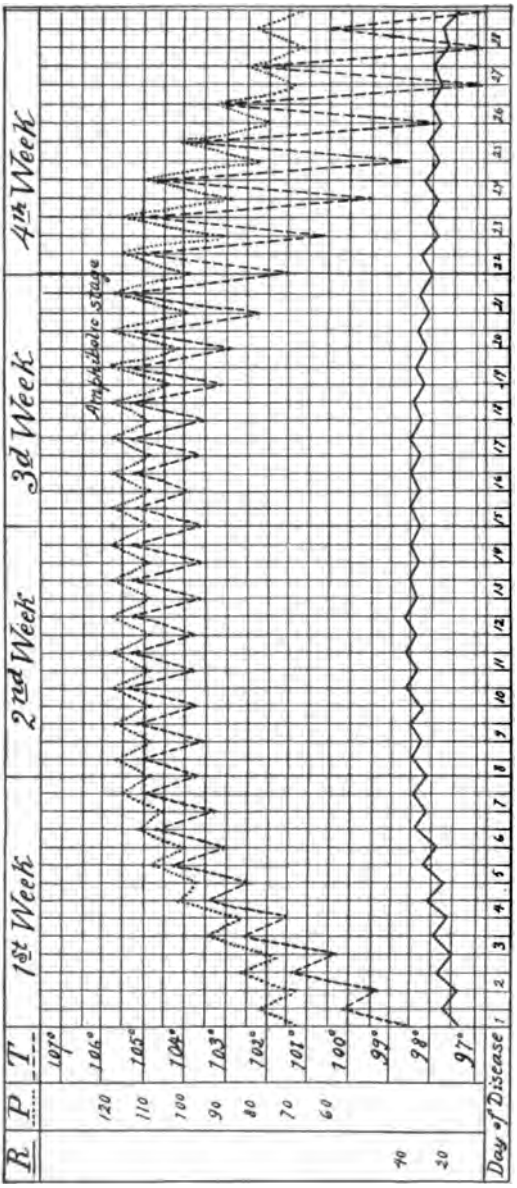

FIG. 24.—Temperature, pulse, and respiration curve in typhoid fever (schematic).

as rapidly as it developed, usually accompanied by a profuse perspiration. This sudden drop in the temperature, so characteristic of pneumonia, is called the *crisis*. *Lysis*, in contradistinction, signifies a gradual drop of temperature during two or three days. The latter is rarely observed in croupous pneumonia, but very com-

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monly in many other febrile diseases. A *protracted crisis* is a transitional type between the two. An *interrupted crisis* is a critical drop of temperature interrupted by a transitory rise. A *pseudocrisis* is one in which a critical fall is rapidly followed by a rise and persistence of the fever. A decided rise of temperature associated with a marked disturbance of the patient's general condition, the so-called *perturbatic critica*, sometimes precedes the crisis. All of these conditions may be variously combined in pneumonia (Fig. 23). So far as the fever is concerned, erysipelas resembles it very closely.

THE COURSE OF TYPHOID FEVER.

The temperature of typhoid fever is characterized by a gradual stepladder-like onset, so that each evening it reaches a little higher point than the evening before.

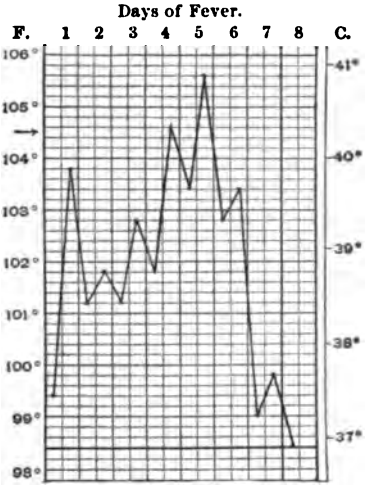

FIG. 25.—Fever curve in measles.

The *initial* stage lasts four to seven days; it is followed by a period of continued high temperature without much of any diurnal variation, the so-called *fastigium*, (seven to ten days); and then by a period of remittent fever, the *amphibolic stage*,

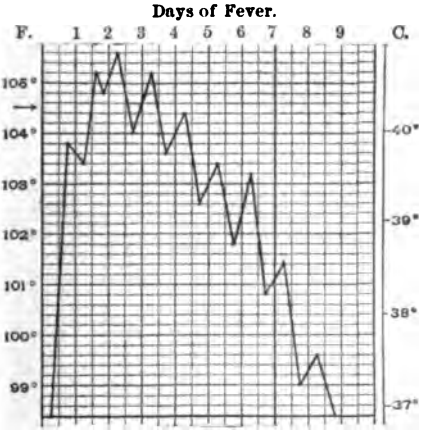

FIG. 26.—Temperature curve in scarlet fever.

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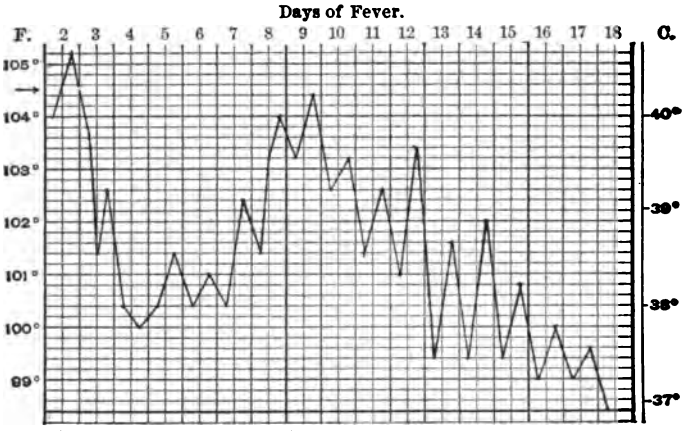

FIG. 27.—Temperature curve in small-pox.

in which the diurnal variations are very marked, often with a difference of several degrees. This lasts five to ten days and merges into the *defervescing stage*, which is as

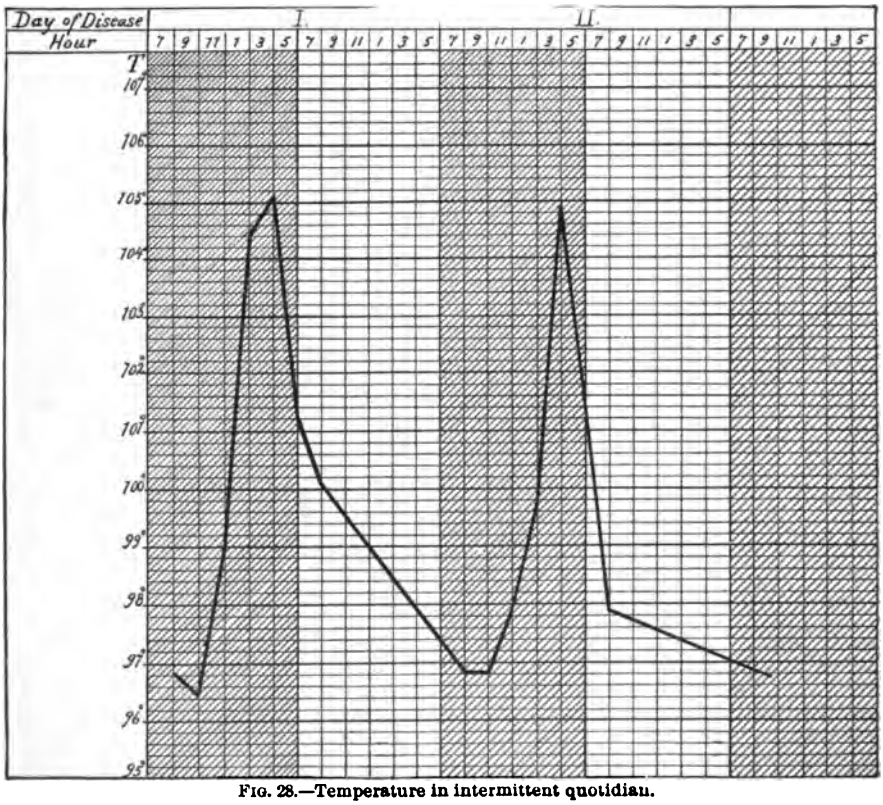

FIG. 28.—Temperature in intermittent quotidian.

gradual as the initial rise. The chart on page 70 (Fig. 24) shows this peculiar temperature curve better than we can describe it.

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TEMPERATURE CURVE OF MALARIAL FEVER.

The curve of malarial fever (Figs. 28 and 29) is characterized by a critical rise and fall of the temperature every two or three days, associated with the other symptoms of an acute disease. In typic cases the patient feels quite well during the interval; the rise of temperature occurs very suddenly, is accompanied by a

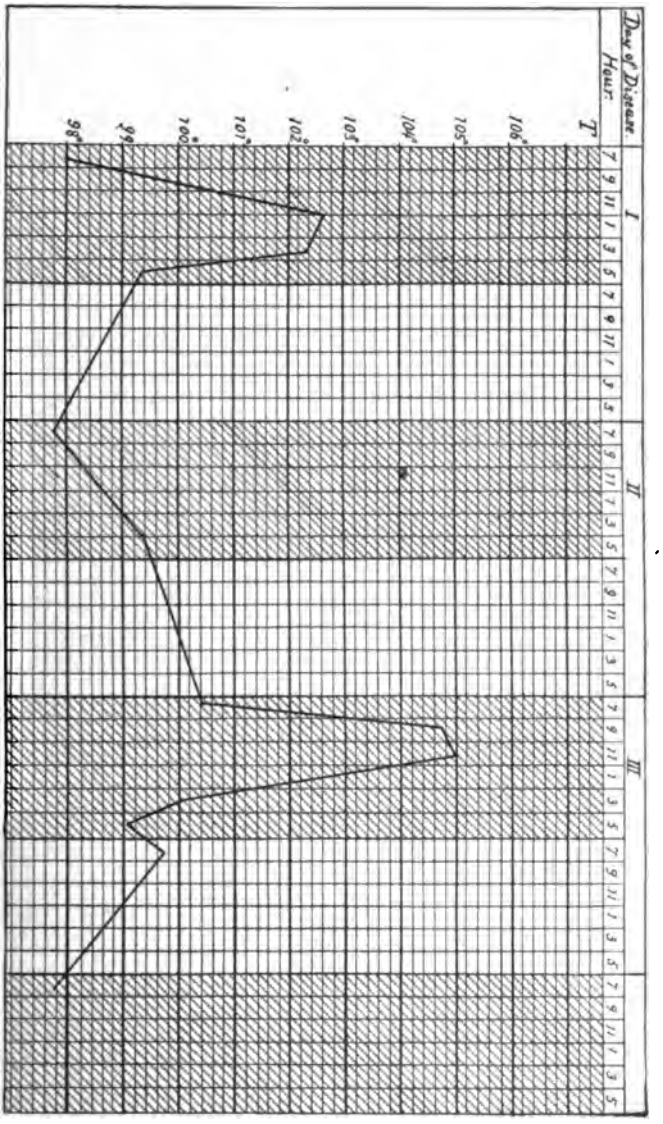

FIG. 29.—Temperature in intermittent tertian anteponens.

marked chill, and is followed later by a sudden drop and a profuse perspiration. The various types are named *quotidian*, *tertian*, and *quartan* fever. As a rule, in malaria the attacks repeat themselves at exactly the same hour. Numerous exceptions do, however, occur; in the anticipating variety, the attack of fever appears a little earlier each day; in the postponing variety, a little later each day.

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CURVE IN RECURRENT FEVER.

As in malaria so in *relapsing fever* the temperature curve consists of a number of individual attacks. Each attack and each interval, however, persist for several days in recurrent fever (Fig. 30). The relapses may be repeated several times, but finally become shorter and less severe, until complete defervesence takes place.

RELAPSES.

In any acute infectious disease there may be a recrudescence of the specific symptoms, and especially of the fever, after convalescence has apparently begun. Such recrudesences are most common in typhoid. They should not, of course, be

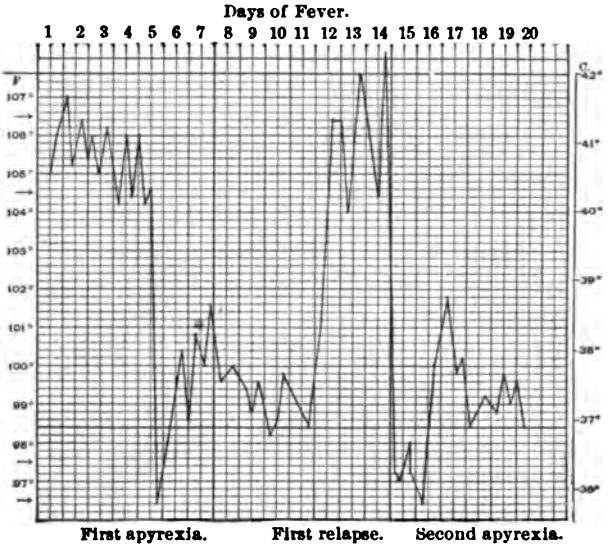

FIG. 30.—Temperature of recurrent fever (Wunderlich).

confused with the individual attacks in febris recurrens or malaria, nor with any complication of the original disease; as, *e. g.*, an otitis in measles or scarlet fever.

HECTIC FEVER.

This is the typic fever of *chronic tuberculosis*. It generally persists a considerable length of time and is of a remittent or interrupted character, with sudden rises and falls. The minimum temperature is usually in the morning and the maximum in the evening, or the opposite type may occur—inverted hectic fever. A two-hour chart exhibits a very irregular course, with many slight rises and falls throughout the day.

PUS, OR SUPPURATIVE FEVER; ERRATIC CHILLS IN PYEMIA, ULCERATIVE ENDOCARDITIS, AND GALL-STONES; CHILLS IN INFARCTIONS.

Though often resembling the chart of hectic fever, that of suppuration is generally more irregular in the time and the degree of the exacerbations.

In pyemia the fever frequently occurs in very intense paroxysms, accompanied by severe chills resembling malaria, except that they are more irregular. Closely related to these so-called erratic chills of pye-

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mia are the chills which appear in ulcerative endocarditis and in gall-stone affections. Non-purulent infarctions are associated with a similar, though a less pronounced, chill.

ATYPICAL FEVER.

In some diseases there is no type to the temperature course, so that, although the clinical picture is sufficiently constant, the temperature

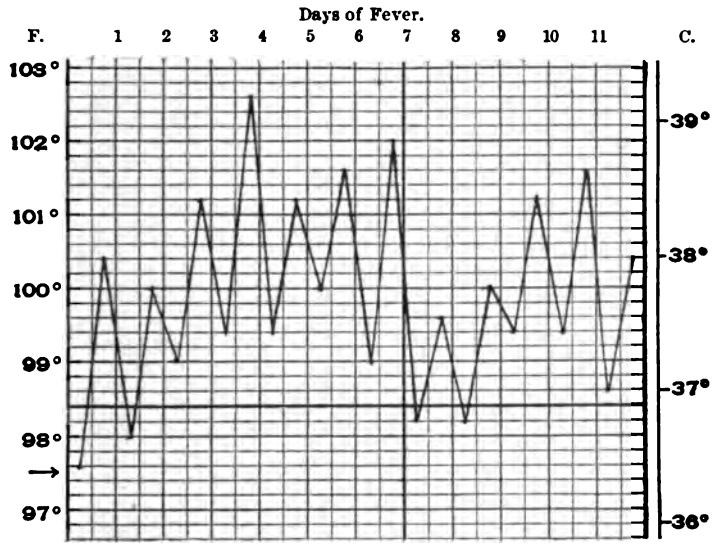

FIG. 31.—Hectic fever in phthisis.

chart alone would furnish very little information of the nature of the disease. Examples are *diphtheria* and *septic processes*.

SUBNORMAL TEMPERATURES.

These are quite as important clinically as febrile temperatures.

Wunderlich considers anything below 36.25°C. (95.25°F.) a subnormal temperature.

Marked depressions of temperature are noted:

1. In the prolonged action of intense cold.[1] A temperature of 27° C. (80.6°F.), as in a case of freezing, is not necessarily fatal.

2. Following a pronounced critical fall of temperature in fever. After the crisis in pneumonia the temperature often drops to 34° or 35° C. (93.2° or 95° F.).

3. In so-called "collapse," where there is a sudden fall in the temperature. This occurs in very sick patients (especially those with fever), and is associated with marked weakness, numbness, rapid pulse, profuse per-

[1] Compare Glaser, "Ueber Vorkommen und Ursachen abnormale niedriger Körpertemperaturen, Inauguraldissertation," Bern, 1878; and Janssen-Quincke, "Ueber subnormale Körpertemperaturen," *D. Arch. f. klin. Med.*, vol. liii., p. 247.

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spiration, etc. The crisis in pneumonia is sometimes mistaken for a collapse. The pulse, however, in the latter is weak and rapid; whereas in the former it remains of good strength, and diminishes in frequency proportionally to the fall in the temperature. Collapse is a precursor of death. The accompanying symptoms are usually attributed to critical weakness. Vasomotor inhibition may also be a factor.

4. Sometimes *after a severe hemorrage;* in *chronic heart and lung diseases*, which lead to imperfect aëration of the blood (cyanosis), and

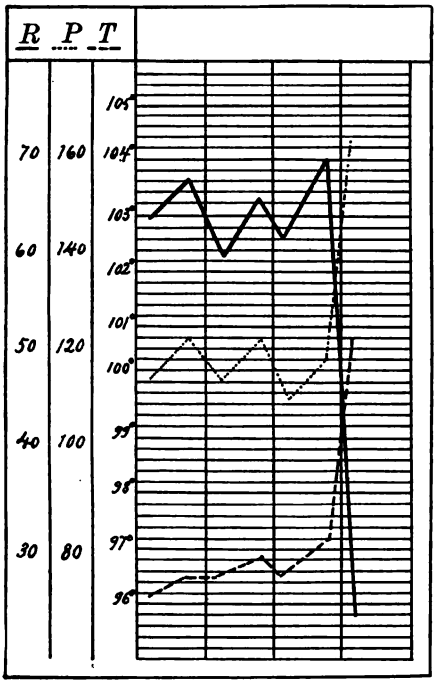

FIG. 32.—Pulse, temperature, and respiration in collapse.

therefore to insufficient oxidation of the various processes throughout the body; further, in *chronic wasting-diseases* (carcinoma of the esophagus), where metabolism is reduced to a minimum; in *sclerema neonatorum;* and finally in *mentally afflicted patients*, especially of the melancholic type.

Prolonged subnormal temperatures accompanying such conditions as are mentioned above always indicate a grave diminution of the metabolism or some equally serious disturbance of the heat regulation.

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CHARACTER OF THE RESPIRATION.

FREQUENCY OF RESPIRATION UNDER PHYSIOLOGIC CONDITIONS.

HUTCHINSON quotes the normal respiratory frequency in adults as between 16 and 24, or about 1 to every 4 pulse-beats. Quetelet gives for the newborn an average of 44, for the five-year old child 26, respirations to the minute.

It is a good plan to count the respirations while apparently feeling the pulse, because the patient's attention might otherwise modify the rate. For greater accuracy, the number of respirations should be counted during an entire minute.

Physical exertion will increase the rate of respiration; lying down or sleep will diminish it somewhat. A stomach distended by food and drink increases the rapidity of the respiration because the diaphragm excursion is thereby somewhat limited. Irritation of the skin may increase or diminish the rate of respiration. Psychic or sensory impressions, movements, clearing the throat, eating, drinking, or smoking will alter the rapidity of the respiration. Before considering that a certain rate of respiration is pathologic we must therefore bear in mind all these physiologic variations.

NORMAL TYPES OF BREATHING.

The distention of the lungs with air takes place partly by means of raising the ribs and sternum and rotating the former outward and upward, and partly by depressing the diaphragm. Although in everybody both these factors take part in the movement of respiration, yet one or the other is apt to be more prominent, thus producing a more *costal* or a more *diaphragmatic* (*i. e.*, abdominal) type of breathing. Women commonly breath costally; men, costo-abdominally. This selection, which seems to depend upon the formation of the chest, is naturally very suitable during pregnancy, when the diaphragmatic excursions are interfered with. A child's breathing is essentially costal.

PATHOLOGIC VARIATIONS IN THE TYPE OF RESPIRATION.

Either the costal or the diaphragmatic element may be implicated and so alter the type of respiration.

Limitation of Diaphragmatic Breathing.—The excursions of the diaphragm may be impeded by some mechanical interference with its descent—*e. g.*, by a paralysis of its muscular structure; by an abnormal flattening of its vault (in certain types of emphysema); by painful breathing; by any increase in the abdominal contents—*i. e.*, pregnancy, meteorism, abdominal tumors, ascites. *Any inflammation* in the vicinity of the diaphragm—*e. g.*, pleurisy, pericarditis, peritonitis—will

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limit the excursions of the diaphragm, partly on account of the pain and partly from a slight paralysis of the muscle fibers of the diaphragm (especially in diffuse peritonitis, on account of the disturbances in the circulation following the inflammation). Actual diaphragmatic paralysis occurs in *multiple neuritis*, in *progressive muscular atrophy*, etc. In any of the above instances the costal breathing may appear to be increased at the expense of the abdominal effort.

Limitation of Costal Breathing.—Costal respiration may be interfered with in a mechanical way by extensive ossification of the costal cartilages (ankylosis of the articulations of the ribs in arthritis deformans, etc.). Under such conditions the costal type in women and children and the costo-abdominal type in men may become purely abdominal.

DIAPHRAGM PHENOMENON AND ALLIED APPEARANCES (LITTEN'S SIGN).

Litten[1] called attention to this almost forgotten sign, analyzed a series of cases, determined its practical significance, and named it the "*diaphragm phenomenon.*" (Plate 4). It consists of a horizontally placed shadow observed with inspiration near the lower pulmonary edge, most constantly anterolaterally, but occasionally running in a ring around the whole chest. This shadow seems to slip downward, corresponding to the inspiratory descent of the pulmonary margin, as shown by percussion. It is very fittingly named, because, as a matter of fact, it is the most distinctly visible evidence of the depression of the diaphragm. Normally, the moving shadow begins above in the sixth intercostal space, descends with superficial inspiration one to one and one-half intercostal spaces, and with deep inspiration two to three spaces. It intersects the ribs at an acute angle. To show it most distinctly, the patient should lie as flat as possible (without extra support for his head) and with his feet toward the window, so that the region between the sixth rib and the costal margin is lighted obliquely. The observer should stand between the patient's feet and the window, with his eye at a distance of 3 or 4 feet and at an angle of about 45 degrees with the lower thorax. Provided the patient breathes deeply and the light, although not necessarily strong, slants sufficiently, the phenomenon is generally plain. Even a candle light may be sufficient, but a very diffuse light is unfavorable. There is no corresponding expiratory sign. The flattening of the lower intercostal spaces from below upward during expiration is of quite different significance.

The explanation of Litten's sign is comparatively simple. As the diaphragm in its descent begins to peel off from the thorax and both widens and deepens the complimentary pleural sinus, it exerts a suction upon the intercostal spaces just below the margin of the lung. This produces the shadow. It is evident that no analogous process will occur during expiration, because the elevation of the diaphragm is regulated only by the elastic retraction of the lung. In health, particularly in lean individuals, the diaphragm phenomenon is a nearly constant appearance. Marked development of fat or muscle or edema of the thoracic wall will prevent its appreciation, and even under perfectly physiologic conditions it may be sought for in vain. Its presence proves that at the particular place where it is observed the diaphragm and the lung lie against the thoracic wall, both freely movable; its absence points to the opposite conclusion. Therefore it cannot be made out opposite pneumonic infiltrations nor in those places in pleurisy where the exudation is situated, or where the lung is adherent to the chest, nor in hydro- or pneumothorax, etc. Litten has called attention to the fact that this sign is of great value in differentiating an empyema from a subphrenic abscess; it is absent in the

[1] *Deutsch. med. Woch.*, 1892, No. 13. Das Zwerchfellphänomen und seine Bedeutung für die Praxis, Deutsche Aerztezeitung, 1895, No. 1. *Verhandl, des Cong. f. inn. Med.*, 1895.

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PLATE 4.

Diaphragm phenomenon (Litten's sign), from a patient with fibroid phthisis of left lung (New York City Hospital). The linear shadow has been emphasized in the reproduction of the photographs.

1. *Full Expiration.*—Note the height of the shadow and the slight concavity of the abdomen corresponding to the respiratory phase.

2. *Medium Inspiration.*—Note the descent of the linear shadow and the slight change of contour of abdomen corresponding to the respiratory phase.

3. *Deep Inspiration.*—Note the further descent of the linear shadow and the rigid abdomen corresponding to the respiratory phase.

Although the artist has intensified the shadow in the reproduction, the excursion of the right lung and right side of the diaphragm were so pronounced in this patient that the distance between the shadows in the extreme positions of respiration was greater than has been represented. The patient's left lung was practically useless; hence the abnormal extent of the right lung's excursion.

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former but present in the latter.[1] Under some conditions the diaphragm phenomenon may facilitate a distinction between pneumothorax and diaphragmatic hernia. A broken or irregular shadow is said to point to partial adhesions of the diaphragm to the thorax-wall. The extent of the diaphragm phenomenon will furnish some idea as to the excursion of the lung in emphysema and phthisis. Paralysis of parts supplied by the phrenic nerve is associated with an absence of the diaphragm phenomenon. Since this sign is by no means absolutely constant in healthy individuals, it is evident that its absence upon one side alone is much more important than upon both sides.

We must be careful not to confuse the diaphragm phenomenon with two other conditions which exhibit shadows in the same area—viz., the visible depression of the lower ribs with expiration and the retraction of the lower intercostal spaces with inspiration.

The former, being expiratory, is easily differentiated from the diaphragm phenomenon; but the latter, being inspiratory, is more difficult to distinguish. The lower intercostal spaces situated along the attachment of the diaphragm are under a positive intra-abdominal pressure when the latter is in a position of expiration, whereas they are exposed to a negative intrathorax pressure when the diaphragm is depressed by inspiration, and therefore they are retracted in the same way as the diaphragm. The physiologic retraction of the lower intercostal spaces as a whole will thus present a descending shadow easily to be confounded with the Litten sign, but a careful observation will detect the difference. The shadow of the Litten sign is linear, passing through the intercostal spaces as a line, while the shadow of the physiologic retraction of the intercostal spaces is more diffuse, each space, one after another, becoming shaded *in toto*. Very often also the descending character of the inspiratory retraction cannot be recognized at all, because the diaphragm in its contraction exerts a suction upon those intercostal spaces from which it has not yet separated. A further distinction is, that an exactly opposite movement from below upward takes place during expiration—*i. e.*, an expiratory bulging. This does not occur in the diaphragm phenomenon.

The depression in stenosis of the air passages, and the essentially identical peripneumonic retraction, differ from this physiologic retraction in that the appearances are very much more marked in the former, affect the ribs as well, and are not confined to the intercostal spaces in the territory of the diaphragm.

ASYMMETRIC RESPIRATION AND PATHOLOGIC INSPIRATORY RETRACTION OF THE CHEST.

An obstruction to respiration which affects but one lung will cause asymmetric breathing; the diseased side will make a less extensive excursion and will also lag somewhat behind the healthy side. The conditions which may give rise to such a unilateral limitation of breathing are: the various types of pulmonary infiltration (pneumonia, phthisis, tumors); pleurisy with effusion or pleurisy merely with the formation of fibrous bands and tough adhesions; and pericarditis with effusion. Asymmetric respiratory excursions may be appreciated by inspection, but they will oftentimes be much better appreciated by palpation. With one hand placed upon each side, the examiner can at the same time appreciate an asymmetry of the chest contour. The chest, half of whose mobility is diminished, may be either expanded or contracted, as explained upon p. 34 *et seq.*

Compare p. 86 *et seq.* for the occurrence of inspiratory retraction in

[1] It is well, however, not to lay too much stress upon this statement, because, although cases have been demonstrated, we can as yet scarcely be sure that the diaphragmatic movement is not sometimes impeded by a subphrenic abscess.

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the jugulum (suprasternal fossa), epigastrium, and flanks with stenosis of the upper air passages.

But similar local retractions occur without any stenosis over parts of the lung area where the normal inspiratory distention of the lung is prevented by atelectasis or by infiltration. This retraction, especially when it occurs quickly and violently (as with dyspnea), is due to the variation in the inspiratory negative pressure within the interior of the chest, which draws in the more expansible portions of the lung along with the overlying portions of the chest wall under the influence of the external atmospheric pressure. We see this particularly well marked in the catarrhal pneumonia of children, whose soft and more flexible chests favor the retraction. It is called a "*peripneumonic retraction*" or a "*peripneumonic groove.*" It is usually situated in the lateral and anterior region of the chest along the lower lung border, even when the pneumonia is, as usual, situated in the back. This probably depends upon the fact that the stiff infiltration located in the back interferes mechanically to a certain extent with the excursions of the lung margins at the sides and in the front, and that the anterior and lateral portions of the chest are especially flexible. Peripneumonic retractions are, however, sometimes observed posteriorly, near the inferior pulmonary margin. The direct pull of the diaphragm, just as in stenosis of the upper air passages, may, in addition to these mechanical factors, help to create a peripneumonic depression (see p. 87). The epigastrium and the jugulum, too, may be retracted in a similar way in pneumonia, so that in small children the diagnosis of croup is not infrequently suggested. The inspiratory stridor, and hoarseness of the voice and of the cough in the latter ordinarily facilitate a distinction. Peripneumonic retraction must not be confounded with the normal inspiratory depression of the lower intercostal spaces, the explanation and differentiation of which have been discussed upon page 78 *et seq.*

D. Gerhardt[1] has recently called attention to another cause of inspiratory retraction of the lower thoracic margin. Besides the diaphragm's well-known effect upon the inferior surface of the lung, Duchenne demonstrated that by resting on the abdominal contents and moving over the parts contained in its vault, as over a roller, it has in inspiration the action of lifting the margin of the thorax. If this decided elevation of the lower ribs is prevented by immobility of the costal articulations or by the horizontal position of the ribs, as in some emphysematous chests, or by the lack of any firm point of attachment of the abdominal contents in enteroptosis, then during inspiration the contraction of the diaphragm will depress the chest-wall inward.

ABNORMALITIES IN FREQUENCY AND RHYTHM OF THE RESPIRATION (not Including Dyspnea).

Alterations in the frequency of respiration depend for the most part upon difficulties of pulmonary aëration or upon increased demands upon the lung, and will therefore be considered and explained with the symptom-complex of dyspnea in the following chapter. Only in very rare

[1] *Zeit. f. klin. Med.*, 1896, vol. xxx., Nos. 1 and 2.

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instances are changes in the frequency of breathing independent of dyspnea.

To this group belongs the diminution of respiratory frequency (oligopnea) found in certain conscious states, especially in severe brain affections (meningitis, hemorrhage, or in tumors of the brain); uremia; diabetic coma; severe infections; some cases of poisoning; and in a similar way in the final agony. Irregularity of the respiration may be noted as well in any one of these conditions. Unquestionably these disturbances are distinctly dependent upon an alteration in the function of the respiratory center.

Either one of two very characteristic types of pathologic breathing, each one associated with a change in the rhythm, may arise under exactly similar conditions instead of the slowed respiration just mentioned. These are the so-called Biot's or meningeal and the Cheyne-Stokes respiration.

Biot's respiration, especially common in meningitis, may also occur in other cerebral disorders and in other grave general conditions. It is characterized by very decided pauses in the breathing, which last from several seconds to half a minute or longer. They are more or less periodic, but at times repeat themselves irregularly. It is a phenomenon of grave prognostic significance.

Cheyne-Stokes respiration is characterized by similar long pauses in the breathing. It differs, however, from the above by the fact that the breathing begins very slowly and superficially after the pause, gradually increases in depth and intensity to a maximum, diminishes again, and finally stops entirely, thus producing another respiratory pause. It is, therefore, a distinctly periodic type of respiration. It occurs under similar conditions to the Biot's meningeal respiration, especially in grave affections of the brain, of the respiratory and of the circulatory organs, and quite frequently in arteriosclerosis and chronic nephritis. Although more frequently observed in the unconscious, it is not uncommon even when consciousness is maintained, especially in patients with chronic respiratory or circulatory disorders. In these affections consciousness oftentimes oscillates with the respiration, is periodically obliterated during the pause, and returns again when respiration is resumed. There are other phenomena more or less characteristic. During the pause in the breathing the rate of the pulse may be decidedly slowed, its tension may be altered, and the pupils may be contracted. Very often, though by no means always, patients have a subjective sense of dyspnea during the period of increasing respiration; and if unconscious during the pause, they are awakened, as they express it, by a sensation of suffocation or dyspnea. Cyanosis usually accompanies the onset of the dyspnea, and it may sometimes even increase with the augmentation of the breathing. With some patients Cheyne-Stokes respiration occurs only during sleep. Medicinal doses of morphin usually intensify the phenomenon and may even originate the condition. Generally speaking the prognostic significance is very grave, although not invariably fatal. According to the severity of the causal condition, the symptom is either

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a transitory sign which rapidly disappears, or else one directly preceding death. Only in cardiac or renal disease has it ever been observed to persist for some months.

The explanation of Cheyne-Stokes respiration is still not wholly agreed upon. It is indisputable that, like meningeal breathing, it depends upon a diminished excitability of the respiratory center. This seems to be the only part of the explanation beyond a doubt. Traube's original theory assumed that the symptom arises when the excitability of the respiratory center is so decidedly diminished by the insufficient supply of oxydized blood resulting from the circulatory disturbance that, at a certain moment coinciding with the first breathing pause, there is no longer a sufficient physiologic stimulus to arouse the respiratory movements. With the cessation of respiration the blood becomes still more decidedly venous. This irritates the respiratory center intensely, so that, despite its diminished susceptibility, breathing begins again. This in turn diminishes the venous character of the blood, and the breathing gradually becomes weaker in proportion to the oxidation of the blood.

Traube's explanation does not make it very clear why the breathing increases gradually nor why the second respiration is stronger and deeper than the first. For as soon as breathing begins, the venous character of the blood must immediately lessen the respiratory center's irritation, and so the second respiration should be weaker. Traube explains that the first breath is so diminutive that it cannot affect the venous character of the blood, and so cannot lessen the irritation to the respiratory center; but that, on the contrary, the irritation increases despite the return of breathing, until it (the irritation) reaches a maximum. Such a hypothesis will explain the peculiarity that the patient appears most cyanotic and complains most of the subjective sensation of dyspnea during the period of increasing respiration. Another objection to Traube's explanation is that after an improved supply of oxygenated blood has once restored the respiratory center's normal excitability, there is no apparent reason for the breathing's fading away again. If we believe with Traube that the respiratory center's asphyxia is the sole cause of its diminished excitability, the objection cannot be answered. If, however, we regard the diminished excitability as entirely or partially independent of the circulatory disturbance and more self-dependent, the objection loses its force. Evidently, then, if the blood is better aërated, the intense irritant to the respiratory center will be removed, although the latter's sensibility has not been improved. Hence, the breathing will gradually diminish in frequency in proportion to the improvement in the oxidation of the blood.

With these modifications, Traube's theory seems plausible enough. Partly on account of the difficulties mentioned above, numerous other explanations of Cheyne-Stokes respiration have been advanced. We will only mention two of these which are the most generally known—viz., Filehne's and Rosenbach's.

Filehne's hypothesis, beginning with the pause in respiration, assumes that the venous condition of the blood irritates the vasomotor

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centers and so produces a spasm of the cerebral vasomotors. Therefore the respiratory center, whose irritability has already been reduced, becomes anemic. This anemia acts as a further irritant to respiration. As soon as the breathing has again oxygenated the blood sufficiently, the vasomotor spasm vanishes, but the respiratory stimulus is not sufficient to induce breathing, hence the pause reappears. Filehne's theory is more complicated than Traube's; the same objections apply to it; and more accurate investigations seem to have completely disproved any such relationship between the vasomotor system and Cheyne-Stokes breathing.

Rosenbach maintains that the periodicity of this type of breathing results simply from an abnormal fatigue of the respiratory center, but that this fatigue should not be confused with a diminished irritability. The center is active for a time, works against a gradually increasing difficulty, finally stops entirely, and begins its activity again only after the pause has to some extent recuperated its powers. It seems doubtful whether we can assume that such recuperation of the respiratory center occurs during the breathing pause, while the respiratory irritant persists. The laws of fatigue in the central nervous system are, however, so imperfectly understood that this objection to Rosenbach's explanation is not sufficient to make us reject it entirely. At all events it has the advantage of depending upon the same law that applies to the other organs—that periodic activity usually arises as a result of fatigue.

Most types of increased respiratory frequency (polypnea) necessitate an increased demand upon the respiratory activity, and will therefore be discussed in the following section on dyspnea. We should, however, mention the fact that increased frequency of breathing may arise from purely nervous causes—*e. g.*, in hysteric people and in certain cases of cerebral disease.

DYSPNEA.

The term dyspnea applies to a large group of variations in respiratory activity, which, in spite of considerable diversity in detail, have this in common, that they serve to promote the object of breathing—*i. e.*, the proper oxidation of the blood—despite all kinds of obstacles. On this account, the breathing of dyspnea is generally increased in frequency or in depth. The obstructions to breathing are, however, sometimes insurmountable, so that neither a frequent nor a deep type of breathing is possible—*e. g.*, in marked stenosis of the air passages. Therefore quickened breathing must not be considered absolutely the distinction of dyspnea. The only definition which will apply clinically to all cases is that dyspnea is an increased respiratory exertion produced by obstruction to breathing or by increased demands upon the blood-oxidizing process. This does not coincide with the explanation frequently given, that dyspnea is identical with quickened breathing, which is unquestionably inaccurate from the clinical standpoint, for not every case of quickened respiration is dyspnea, nor is the breathing quickened in every case of dyspnea. As we shall see, there are types of dyspnea with quickened and others with retarded respiration.

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To conform with the definition cited above and to avoid any misunderstanding, it is advisible to apply the terms *polypnea* to quickened and *oligopnea* to retarded breathing.

The word dyspnea is, however, employed in still another signification to express the subjective sensation of oppressed breathing possessed by patients with objective dyspnea. Ordinarily, subjective and objective dyspnea go hand in hand; but exceptions do occur. For under certain conditions, despite the presence of some obstruction which is responsible for an objective dyspnea, breathing may continue so satisfactorily that the patient does not experience any shortness of breath, because the aëration of the blood proceeds as completely as ever, owing to the modification of the respiratory movement. Again, despite a very pronounced objective dyspnea which is by no means adequate for blood-aëration, as evidenced by the accompanying cyanosis, a patient may have become so accustomed to it (see 93) or his sensorium may be so benumbed that he has no appreciation of subjective dyspnea. It is a very benevolent provision of nature that in the death agony, where the breathing is difficult, the brain becomes, as it were, so narcotized by the carbon dioxid intoxication that it no longer appreciates the sensation of the struggle for breath. Conversely, the objective dyspnea may be almost or entirely overshadowed by the intense "air hunger." Examples are the so-called "precordial terror" of melancholics, which (because the individual locates the sense of anxiety in the chest) we prefer to consider as a purely subjective dyspnea. Again, some nervously organized individuals will complain of a transitory desire to draw an extra deep breath; they have the sensation of dyspnea without any evidence of obstructed breathing. In other words, it is a purely cerebral phenomenon, of which they instinctively try to rid themselves by taking a long breath.

Hence, it is important to differentiate sharply between objective dyspnea—*i. e.*, difficult and hence modified breathing—on the one hand, and subjective dyspnea, or the sensation of lack of air, on the other. Sometimes breathing modified by dyspnea will be accompanied by subjective dyspnea, but at other times this is not the case.

Cyanosis, like subjective dyspnea, is not always proportional to the degree of objective dyspnea, for in one case the objective dyspnea may be sufficient to regulate the proper aëration of the blood and to bring conditions back more or less to a normal standpoint; whereas in other cases this is not possible for the organism.

The occurrence of decided objective dyspnea without cyanosis is a clinical proof that the intensity of the respiratory movement does not depend exclusively upon the grade of aëration of the blood, but may be produced directly by some obstruction to breathing without the appearance of cyanosis. A similar proof is furnished by the dyspnea which follows physical exertion. This in no way depends upon an excess of carbon dioxid nor upon a deficiency of oxygen in the blood so long as the breathing and the circulation remain sufficient.

To aërate the blood properly under adverse circumstances the organism avails itself of an increase either in the frequency or in the

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depth of the respiration. With an increase in the depth of the individual breaths, the frequency may be either accelerated, normal or slowed. There are, therefore, various types of dyspnea, but generally we find that it modifies the normal breathing in that way which seems best adapted to meet the existing deficiencies.

In the following we shall characterize the kinds of objective dyspnea occurring in different diseases.

VARIOUS TYPES OF DYSPNEA.

1. **Dyspnea Caused by Painful Breathing.**—Patients are not infrequently prevented from drawing a deep breath on account of the pain associated with each respiratory movement in certain pulmonary, and especially pleural, disorders, in affections of the intercostal muscles (rheumatism, trichinosis) and of the diaphragm and its vicinity (peritonitis). The breathing then becomes superficial and, to satisfy the demand, more frequent—in other words, dyspnea results. In this instance the obstruction is functional, not mechanical.[1]

2. **Dyspnea Due to a Diminution in the Breathing Surface of the Lung or to a Mechanical Limitation of the Respiratory Excursions of the Lung.**—The two factors ordinarily occur together. Under this heading are included all affections of the pulmonary parenchyma which lead to a diminution of the air contents of the lung—*i. e.*, all sorts of pulmonary infiltration; all conditions which limit the capacity of the thorax, such as pleuritic effusions, pneumothorax, intrathoracic tumors, lateral curvature, upward displacement of the diaphragm; further, all conditions which decrease the respiratory excursions, such as brown induration and emphysema (with regard to emphysema, see p. 90 *et seq.*), as well as paralyses and spasms of the respiratory muscles.

In any of the above conditions each breath aërates the lung less efficiently than normally, with a resulting dyspnea and increased frequency of respiration. Under some conditions the demand for air will be completely satisfied, so that in spite of the interference with respiration neither cyanosis nor any sense of dyspnea (subjective) ensues. Under other conditions the compensation is not always complete, especially when any increased demand for air arises, so that physical exertion will occasion cyanosis and a subjective sense of dyspnea.

If such an interference with breathing is unilateral—*e. g.*, infiltration or a pleuritic exudation—the dyspnea can be partially overcome by deeper breathing of the healthy side (vicarious respiration), as well as by the increased frequency of respiration.

(See p. 80 *et seq.* concerning local depressions of the thorax in this variety of respiratory interference.)

3. **Dyspnea Due to General Circulatory Disturbances.**—Non-compensated valvular lesions may be regarded as a type. Here

[1] In this connection the editors wish to call attention to a type of dyspnea associated with a remarkable slowing of the respiration which is sometimes observed in pneumonia when the accompanying pleurisy excites intense pain with each respiration.

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the chief factor is the stasis or congestion—*i. e.*, the circulation is slowed and the blood accumulates in the veins whether the difficulty affects the left, right, or both sides of the heart. As a consequence of the retarded current the different organs receive less arterial blood in a certain time and retain more venous blood; and since the respiratory center is also affected by this disturbance, more rapid and deeper breathing results.

The conditions become more complicated when the circulatory trouble originates in the left heart, because the congestion then includes not only the general systemic veins, but also the pulmonary veins, thus adding another cause for dyspnea—namely, a marked distention of the pulmonary capillaries with blood. It was formerly assumed that this dilatation of the alveolar vessels impaired respiration by diminishing the amount of air in the lung. But this conception was shown to be incorrect by v. Basch's experimental investigations upon pulmonary congestion, for he proved, on the contrary, that the lung overdistended with blood contains more air because the distention of the alveolar vessels also increases the circumference of the alveoli. Nevertheless, the process in question does impair respiration, because the lung, stiffened by the distention with blood, becomes more and more permanently fixed in the position of inspiration and expands but little. Pulmonary rigidity acts as a direct impediment to breathing (Category 2, p. 85) and increases the respiratory rate. If it is persistent, its effect is intensified by the formation of the so-called "brown induration."

In opposition to this mechanical theory of pulmonary rigidity we would emphasize the work of Krause, who found that, in spite of the engorgement of the lung, the respiratory excursion may even be increased. In this case the dyspnea is to be explained by the fact that the respiratory surface of the dilated pulmonary vessels is relatively decreased in comparison with their contents.

When the lung is affected paroxysmally by pronounced engorgement with blood and rigidity, in disturbances of cardiac activity, the resulting attacks are named "cardiac asthma." This term is often wrongly applied to any kind of dyspnea which appears in heart disease. These paroxysms of cardiac asthma are brought on sometimes by exertion, sometimes by increased flow of blood to the lung in the recumbent posture, sometimes by altered innervation at the moment of falling asleep, and sometimes by entirely unknown causes.

Pulmonary rigidity and brown induration arise in mitral diseases, especially when compensation is good—*i. e.*, when the right ventricle does its work well. For this reason patients with a mitral lesion, despite good compensation, experience dyspnea, even with very moderate exertion.

As a further cause of dyspnea may be added the *bronchial catarrh* that almost always accompanies circulatory disorders.

4. **Dyspnea Dependent Upon Obstruction of the Upper Air Passages.**—Any obstruction in the large upper air passages makes breathing difficult because it furnishes a resistance to the entrance of air and to the inspiratory pull of the respiratory musculature. The latter must therefore perform more work. It is evident that under these

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circumstances an increase in the rate of breathing would be not only very difficult, but also of little avail, while on the other hand, simple reflection shows that slowed breathing would more readily overcome the obstruction. But to supply the requisite amount of oxygen the breathing must be deeper as well. And this is sometimes observed to be the method which patients adopt to overcome obstruction in the larger air passages, but only when there is sufficient inspiratory power to entirely overcome the impediment in breathing. Lacking such power, the economy is obliged to depend more and more upon the less efficient means of increasing the rapidity of the breathing, which, of course, becomes superficial, but that it is always suited to the particular kind of mechanical obstruction is shown by the fact that the increased frequency is slight in comparison with the extent of the respiratory obstruction, since too rapid breathing would be of no avail. This peculiar type may be termed *dyspnea with a tendency to slowed breathing.* The frequency or the depth of the breathing preponderates, depending upon the relation between the stenosis and the respiratory power. It is observed in stenosis of the pharynx from swelling of the tonsils; in retropharyngeal abscess; in true and pseudocroup; in edema and in spasms of the glottis; in paralysis of the abductors of the vocal cords; in stenosis of the larynx or trachea from tumors or foreign bodies; in obstruction of the trachea from external compression (glands of the neck, aneurism, etc). If the stenosis is situated in one of the main bronchi, it will depend again upon the degree of obstruction whether the dyspnea is combined with slowed or accelerated breathing. If the lung upon the affected side can still be made use of, the dyspnea will be of the former type; but if the healthy lung must do all the work, the breathing will be rapid. In either case the economy selects the more advantageous method. Thus, in all the conditions in question, the demand for air will be supplied, and the subjective dyspnea and cyanosis avoided, unless, of course, the obstruction is too great.

When the obstruction in the upper air passages is very considerable, and when, despite the changes in respiration resulting from the dyspnea, the lung can no longer completely fill itself with air again, the chest "pumps empty," so to speak. During inspiration an abnormally rarefied space is found in the lung, and the lateral flexible portions of the chest-wall, the epigastrium, the supraclavicular fossæ, and the suprasternal notch (jugulum) sink in under the influence of the external atmospheric pressure.

There is another cause for depression of the lower lateral chest regions in this form of dyspnea. As a result of the empty pumping, the vault of the diaphragm is not depressed with inspiration, but may even be sucked upward. Besides, as a result of the stenosis, the ribs can not be lifted up by the diaphragmatic action (described by Duchenne, p. 80), and so the contraction of the diaphragm practically accomplishes nothing but drawing in its points of attachment to the ribs. This pathologic retraction of the lower lateral portion of the entire chest-wall must not be confounded with the purely physiologic retrac-

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tion of the lower intercostal spaces in inspiration (see p. 79). The differentiation is easy enough. In the latter only the lower intercostal spaces along the line of the diaphragm are affected, and there is never any retraction of the ribs nor of the epigastrium. The retraction of stenosis is observed most distinctly in children because their chests are very flexible. It is very common in croup.

A peculiar stridor or a sort of whistling, due to the passage of air through a narrowed place, is a most characteristic accompaniment of dyspnea caused by stenosis of the upper air passages. It is ordinarily heard much more distinctly with inspiration than with expiration. In laryngeal croup the stridor is due to the membrane covering the vocal cords. The inspiratory accentuation of the stridor is explained by assuming that, in spite of the inspiratory opening of the glottis, the vocal cords are not materially separated, and that as a result of their slanting, roof-like position they are approximated still more closely, valve fashion, by the pneumatic tug of inspiration. Hence, the obstacle to inspiration is greater than to expiration. This explanation is probably correct for cases of laryngeal croup. Nevertheless, the same inspiratory increase of stridor is observed in other types of stenosis of the upper air passages where we cannot assume the result of any such valve-like effect in the larynx—*e. g.*, from obstruction either above or below the larynx (retropharyngeal abscess, goiter, etc.). The probable explanation of such cases is that the obstruction prevents the free ingress of air during the inspiratory effort at a moment when suction is exerted upon the surrounding parts by the external atmospheric pressure, so that the trachea is constricted below the sternum (inspiratory retraction of the jugulum, suprasternal notch). Therefore, any obstruction in the upper air passages, even if not actually located in the larynx, acts much more effectually during inspiration than during expiration. In other words, the dyspnea is essentially inspiratory. Another factor may influence this inspiratory accentuation of the stridor. The velocity of the air current in inspiration is much greater than in expiration, which is passive, depending upon the elasticity of the lungs and the thorax. The importance of this factor is shown by the fact that as soon as patients with stenosis of the upper air passages experience a sufficient increase in the respiratory obstruction to necessitate their employing the help of abdominal pressure, the expiratory stridor will become more marked, and may even outweigh the inspiratory stridor.

5. **Dyspnea in Bronchitis.**—The types of bronchitis which usually lead to dyspnea are chiefly those affecting the smaller bronchi. The dyspnea is caused by the stenosis of the bronchial lumen, due to swelling of the mucous membrane and to secretion. The stenosis affects so many places that ordinary breathing is insufficient and dyspnea arises.

This type of dyspnea varies with the conditions which prevail. If the stenosis affects only a small number of bronchi, simple increase of the breathing rate will usually overcome the difficulty. The stenosed areas are not benefited much by this method, but the uninvolved portions receive more air, and so the effect of the disturbance is equalized.

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This type of dyspnea then comes under our second heading. If the stenosis affects a large number of bronchi, the breathing varies according to whether the bronchial closure is quite complete and insurmountable, as in *capillary bronchitis* proper, or whether it is incomplete and still conquerable by the respiratory effort, as in *diffuse dry bronchitis of the medium-sized tubes.* In the former variety, of which capillary bronchitis is a type, the chief effect is again merely a diminution of the breathing surface, so that, as before, dyspnea with rapid respiration results. This, of course, only assists bronchial areas which still remain patent. In the second type, however, where we suppose that most if not all of the medium-sized bronchi are stenosed, although only moderately so, the respiratory effort must attempt to get a sufficient quantity of air through the constricted areas into the pulmonary tissue proper. This can generally be best accomplished by an abnormally deep respiration, very much as in stenosis of the upper air passages, especially of the larynx. And just as in these cases, the respiratory accommodation and the frequency of the individual efforts will vary according to the amount of obstruction as compared with the respiratory force available. In rare instances even a slowing of respiration may result. More frequently, however, we merely observe a certain tendency to retardation in that the increase in rate does not correspond to the degree of constriction, and this increase is comparatively slight as contrasted with the amount of subjective dyspnea and cyanosis. The passage of air through these constricted areas is often associated here, as well, with a stridor—*i. e.*, a stenotic noise which may perhaps be heard at a considerable distance from the patient. This type of dyspnea in bronchitis presents, therefore, a prolongation of expiration and stridor, chiefly expiratory, and so can be distinguished from the other retarded type or laryngeal dyspnea, where expiration is of normal length and the stridor is chiefly inspiratory.

These two points of distinction, which are responsible for the application of the name expiratory to this type of dyspnea, require some further explanation. The prolongation of expiration is easily understood, because even under normal conditions expiration is longer than inspiration, and, of course, the increased resistance caused by the bronchial stenosis must be felt more during expiration, since the excitation of dyspnea produces, first of all, marked increase of the inspiratory power. To be sure, if the dyspnea is pronounced, the expiratory exertion must be also increased by the participation of abdominal pressure in the expiratory act, so that the lungs will then be emptied somewhat more quickly by the active expiratory compression of the thoracic contents than by the influence solely of elasticity. This extra abdominal pressure, however, will also compress the small bronchi still more; hence the prolongation of expiration as compared with inspiration, and the increase of expiratory stridor. Increased expiratory stridor may be present without the participation of abdominal pressure. For if the elastic retraction of the lung is interfered with by stenoses in the bronchi, the latter will be still more compressed by this elastic expiratory move-

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ment just as by abdominal pressure. Hence, abdominal pressure is not absolutely necessary for the increased expiratory stridor. All sorts of intermediate types occur between this type of retarded breathing with prolonged expiration and expiratory stridor on the one hand, and the type in which the breathing is simply increased in frequency on the other. These types will vary according to the preponderance of bronchial stenosis or of the available respiratory power.

6. **Dyspnea in Bronchial Asthma.**—According to the modern conception, bronchial asthma depends upon a stenosis of the smaller bronchi, due to spasm of the bronchial muscles. Hence, it corresponds exactly to dyspnea in the type of bronchitis just discussed, that with moderate stenosis of the bronchi. The breathing is retarded and expiration is prolonged and combined with stridor (so-called *expiratory dyspnea*). The absolute number of respirations may be diminished; or an increase in rate which would correspond to the amount of subjective dyspnea may be prevented. This depends upon the degree and the extent of the bronchial muscles' spasm. Therefore, in bronchial asthma we may observe either a diminished, normal, or increased respiratory frequency. The conditions are analogous to those in emphysema (see next section).

7. **Dyspnea in Emphysema.**—Pulmonary emphysema causes dyspnea because the lung is in a permanent condition approximating the inspiratory position, and because with inspiration and expiration it makes but small excursions from this position. Besides this, pronounced emphysema destroys numerous alveolar septa and their capillaries, thus diminishing the breathing surface very extensively. For this reason the dyspnea of pure uncomplicated emphysema is evidenced by rapid and superficial respiration. When the patient is quiet the disturbance remains slight, but when some physical exertion makes extra demands upon the respiration the dyspnea increases very decidedly. As a rule, patients with emphysema appear for the treatment of marked dyspnea only when this affection is complicated by a bronchitis (usually the dry variety). Consequently, in emphysema the same influences which we have just discussed in bronchitis tend to render the respiration now quicker and now slower. The tendency to retardation is very pronounced, because the bronchitis complicating the emphysema is usually diffuse and produces a stenosis of most of the bronchi. The loss of elasticity in the emphysematous lung especially promotes a retardation of breathing and prolongation of expiration. But the dyspnea in emphysematous bronchitis is not always associated with a slowing of respiration, for the system will accommodate itself to the respiratory obstruction with or without an increase of respiration, depending entirely upon the proportion between the loss of elasticity in the lung, the degree of bronchial stenosis, and the respiratory power. As a matter of fact, in the bronchitis of emphysema there is very often a certain amount of increase in the frequency of breathing. Still, the subjective oppression outweighs the slight increase so decidedly that, unless we count, the impression is made that the respirations are slow, although actually 20 to 25 a minute. In some cases this tendency to retardation leads to an actual

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diminution in the number of respirations; in other cases, only to a lack of any increase; in still another class of cases, to an increase in respirations which is but slight in proportion to the subjective dyspnea. Thus, the bronchial dyspnea of the emphysematous may be characterized as a dyspnea with a prolonged expiration and expiratory stridor (so-called expiratory dyspnea) and also with a more or less pronounced tendency to a diminution in the frequency of the breathing. These conditions are unchanged if, as is often the case, the emphysema is complicated with bronchial asthma.

8. **So-called Uremic Dyspnea of Nephritis.**—This exhibits no uniform symptom-complex. In individual cases, and particularly if retarded breathing and prolonged expiration occur, we are justified in assuming the existence of some true uremic phenomenon—*i. e.*, an actual bronchial asthma due to uremia. Many diagnostic conditions in nephritis are, however, erroneously considered uremic. They depend rather more upon some cardiac disorder, upon the accompanying bronchial catarrh, or upon a beginning pulmonary edema, etc. Corresponding to the diversity in causation, these types of dyspnea vary decidedly in their character.

9. **Febrile Dyspnea.**—A febrile elevation of the body temperature is nearly always combined with an increase in respiratory frequency. Even the artificial application of heat to the body will increase the respiratory rate, so that it seems plausible that the irritation of warmer blood upon the respiratory center is responsible for the febrile rise of the respiratory rate.

However, the frequency of respiration in febrile diseases with exactly the same degree of temperature may differ widely, so that the nature of the toxin probably exerts some direct influence upon the respiratory center. The increased respiratory frequency in fever probably corresponds to an increased demand upon the metabolism. Therefore, according to our definition (p. 83), we are correct in speaking of febrile dyspnea as well as of febrile polypnea.

The respiratory frequency found with a certain degree of fever varies with each individual case. Experience shows that, unless some respiratory complication exists, only the severe types of fever increase the respiratory frequency very noticeably. Fig. 24, the curve of an uncomplicated typhoid, illustrates the usual ratio between temperature and respiration rate.

10. **Anemic Dyspnea.—Exaggerated Breathing in Diabetic Coma.**—Dyspnea occurs in anemia (oligochromemia) because the available amount of hemoglobin is so small that only the most complete aëration will be sufficient to supply the demand for oxygen. With no mechanical impediment to respiration, the organism can adapt itself very completely to the altered conditions. This is accomplished by a simultaneous increase in the frequency and in the depth of respiration. The resulting dyspnea produces a peculiar clinical picture, for the respirations are not only very rapid, but also of maximum depth. This type is observed especially in pernicious anemia; rarely, however, in other disorders.

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The same type of breathing is observed in diabetic coma.[1] We do not know its cause, so that it seems doubtful if we should consider it as actual dyspnea (in the sense of p. 83, *et seq.*)—*i. e.*, analogous to anemic dyspnea. However, the outward resemblance of the two phenomena makes such a supposition possible (diminution of the hemoglobin oxidation capacity in diabetic coma).

11. **"Mixed" Dyspnea—Inspiratory and Expiratory.**—Thus far we have been discussing the various factors which determine the different types of objective dyspnea. Two main types occur: (1) breathing with increased frequency and (2) slowed breathing with increased depth. In the former "mixed dyspnea" inspiration and expiration seem equally accelerated. In the latter, inspiration and expiration are affected differently; in some cases the inspiration, in others the expiration appearing prolonged. One type is termed "inspiratory dyspnea," the other "expiratory"; because in the one it is principally inspiration, and in the other expiration, which is rendered more difficult.

In this connection compare the paragraphs on the types of dyspnea in obstruction of the upper air passages in bronchitis, in emphysema, and in asthma. *Inspiratory dyspnea* can usually be distinguished immediately by inspiratory, *expiratory dyspnea*, by expiratory stridor, without necessarily estimating the length of the two phases in respiration. If the examiner is inclined to do this he should remember that normally expiration lasts longer than inspiration (4 : 1).

AUXILIARY OR ACCESSORY BREATHING; FORCED ATTITUDES IN DYSPNEA.

In all types of dyspnea the economy ordinarily employs all the available aid to lighten the burden of the respiration. In this way a number of muscles are used for breathing which, under normal conditions, serve other purposes. These are the so-called accessory respiratory muscles, chiefly the scaleni, trapezius, levator angulæ scapulæ, sternocleidomastoid, sternothyroid, thyrohyoid, serrati, and the pectorals. The last five mentioned muscles may serve as inspiratory muscles, because their usual fixed attachment to the chest is made the mobile point, the other end becoming stationary.

We have already considered upon page 24 how the erect posture facilitates the task of these accessory muscles (orthopnea), and the importance of constrained lateral positions with a unilateral obstruction to breathing. The abdominal muscles are the only ones employed as accessory muscles to expiration. Naturally they are chiefly active in expiratory dyspnea, but they may also be utilized to advantage in mixed and in inspiratory dyspnea to accelerate expiration and thus to facilitate a new inspiration.

If the dyspnea is very pronounced various other accessory muscles to respiration may be called into play. Their activity is of certain diagnostic importance, although it must be acknowledged that they do not materially assist the respiration. The facial muscles of expression are examples. During inspiration they distend the openings of the

[1] Kussmaul, *D. Arch. f. klin. Med.*, vol. iv., 1874.

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mouth, and especially of the nose, to a maximum. The patient thus presents quite a characteristic and pitiful appearance. This dilatation of the alæ nasæ is especially pronounced in small children with pneumonia. Such a maximal distention of the entrances to the respiratory tract may sometimes be of real value in diagnosis; but it ordinarily means that some effect of the vigorous respiratory stimulation has been transferred to related muscle groups.

RELATION OF OBJECTIVE DYSPNEA TO CYANOSIS AND TO SUBJECTIVE DYSPNEA; HABITUATION TO RESPIRATORY OBSTRUCTION AND TO DYSPNEA.

Objective dyspnea serves to diminish both cyanosis and the subjective sense of dyspnea. This task is not always performed equally well. The more completely the dyspnea aërates the blood, the less danger to the individual; but cyanosis can only be entirely overcome when the obstruction to inspiration is slight. Wherever a marked obstruction occurs, some little (oftentimes considerable) cyanosis must persist. The economy then performs its functions with blood rich in carbonic acid and poor in oxygen, and the dyspnea only succeeds in preventing a progressive deterioration of the blood. In cases of chronic dyspnea the economy may become more or less accustomed to the cyanosis. This is evidenced not only by the relatively good maintenance of the other body functions, but also because the sense of subjective dyspnea—"air hunger"—gradually and progressively disappears. Conversely, with the same degree of respiratory obstruction, the quicker the latter develops, the more intense the subjective dyspnea.

Pneumothorax furnishes one of the most striking examples of the economy's power of adaptation to respiratory obstruction. After the sudden onset of the respiratory obstruction, both the objective and subjective dyspnea are at first very pronounced, but soon diminish. In pneumothorax this does not merely depend upon a simple adjustment of the economy to cyanotic blood any more than in other conditions. For in this type, as in many others, the disturbance itself is lessened by a number of complicated, equalizing factors which proceed especially from the circulatory system. This also becomes plain from the diminution of objective dyspnea.

We need scarcely add that dyspnea with a slight degree of cyanosis suggests a far better prognosis than dyspnea with pronounced cyanosis.

SPIROMETRY AND PNEUMATOMETRY.

Spirometry and pneumatometry—*i. e.*, the mensuration of the vital capacity and of the respiratory pressure variations in the upper air passages—have not as yet acquired any great diagnostic importance. This failure is due to the great difficulties in technic and to the pronounced influence of practice in those examined. Readers who wish to study thoroughly this method of examination should consult textbooks upon physiology and several of the original works upon pneumatometry.[1]

[1] P. Donders, *Zeit. f. rat. Med.*, N. F., vol. iii., 1853, p. 287 *et seq.*; Waldenberg, "Die Manometrie der Lunge, oder Pneumatometrie als diagnostische Methode," *Berlin. klin. Woch.*, 1871, N. 45; Eichhorst, *D. Arch. f. klin. Med.*, 1873, vol. xi., p. 268; Biedert, *ibid.*, 1876, vol. xviii., p. 115; Rollet, *ibid.*, vol. xix., p. 284; Neupaur, *ibid.*, vol. xxiii., p. 481.

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The pneumoscope described by Bloch[1] seems to be the most useful for clinical purposes. This instrument calculates the respiratory power without determining the respiratory pressure manometrically, but by estimating the minimum diameter of a breathing cannula held in the mouth, through which a sufficient quantity of air may be breathed. More accurate information of the respiratory mechanism can usually be obtained by directly measuring the thoracic excursions with a tape measure than by spirometry or pneumatometry (see p. 29, *et seq.*).

CHARACTER OF THE VOICE UNDER PATHOLOGIC CONDITIONS.

PATHOLOGIC alterations in the voice arise partly from demonstrable disorders of the vocal organ proper (the larynx) and partly from other influences, either direct affections of, or disorders indirectly connected with, the respiratory apparatus. We shall mention here only such alterations of the voice as are of some diagnostic importance.

If the expiratory current of air does not make the vocal cords vibrate normally the voice will be "*hoarse*"—*e. g.*, in all inflammations, ulcerations, tumors, or paralyses of the cords. A hoarse voice always indicates some affection of the larynx and necessitates a laryngoscopic examination. If combined with inspiratory dyspnea, hoarseness is very suggestive of laryngeal obstruction. It may, however, be due to some general condition—*e. g.*, the loss of the cords' normal tone, in weak cachectic patients. Again, it may result from a fit of violent coughing. Here a paresis of the tensors of the cords is caused by the marked stretching of the cords during the glottis closure which precedes the cough. The hoarseness in phthisis, therefore, does not always mean an ulceration of the larynx or cords.

The voice is frequently lost in hysteric aphonia without preliminary hoarseness; whereas a similar loss of voice is always preceded or introduced by hoarseness if the aphonia is due to anatomic changes in the larynx. This is a point of some diagnostic importance.

The voice may acquire a nasal twang as a result of alterations in the conditions of resonance in the mouth or nasal cavity. We distinguish a closed or stopped from an open nasal voice. The former is observed when the nasopharynx or the nasal cavity itself is obstructed by any pathologic products, such as tumefactions of the mucous membrane, polypi, adenoids. The latter, the "open nasal voice," is observed when anything prevents the normal closure between the nasal cavity and the mouth—*e. g.*, palate paralysis, cleft palate, syphilitic affections of the palate, etc.

Aphonia (lack of voice) results either from an inability to approximate the vocal cords or from a prevention of the normal vibration. It may be preceded by hoarseness.

The voice is, moreover, very dependent upon the general condition and upon the thoracic organs. Patients who are very ill usually have a weak voice, corresponding to their general muscular weakness. In diseases of the respiratory and circulatory organs the voice is affected by

[1] *Arch. de Physiol.*,1897, p. 1.

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the disturbed respiratory excursions and by the various sorts of dyspnea. In many patients with heart disease the voice—*i. e.*, the cord tonus—is a very delicate indicator of the condition of their general circulation—it becomes weak and feeble when they fail, and full and sonorous when they improve. In painful affections of the lungs or pleura and in peritonitis the voice becomes characteristically feeble, soft, and often broken. Cholera patients speak with a "toneless" voice (vox cholerica), and the moribund with a faint, scarcely audible voice. [Further reference will be made to these and similar points in the section on Auscultation (bronchophony)].

COUGH.

A COUGH consists of a single or of a consecutive series of explosive expiratory movements produced by abdominal pressure, which result in overcoming the preliminary closure of the glottis. It is a very important *reflex act*, serving the purpose, on the one hand, of expelling any foreign bodies which may have become lodged in the respiratory passages, and, on the other hand, of expectorating any material which may have accumulated there from some pathologic process—*e. g.*, bronchial or alveolar secretions, effused blood, pus which has perforated into the bronchi, disintegrated necrotic or tubercular lung material, etc. A cough may be excited from various places in the body. The commonest type of cough is one in which the reflex arises from the region supplied by the sensory branches of the vagus. Experimental investigations have shown (especially Nothnagel's) that irritation of the laryngeal mucous membrane *above* the vocal cords produces spasmodic closure of the larynx, but no cough. Coughing will, however, be induced if the irritation affects the parts *below* the vocal cords. The most sensitive areas are the interarytenoid mucous membrane and the region of the bifurcation of the trachea. Coughing may also be excited from any other part of the tracheal, as well as from all parts of the bronchial, mucous membrane, but not from the pulmonary parenchyma proper.

Experimental results do not agree as to whether a cough can be excited directly from the pleura; but it seems probable, judging from experience with individuals whose pleura has been opened.

These are the most important sources of coughing, but there are other less common ones. In some individuals coughing may be produced by irritation of the pharynx or of the base of the tongue, or occasionally of the esophagus. Coughing has been observed very exceptionally to result from tickling the external auditory canal (auricular branch of the vagus) or from manual pressure upon the spleen or liver. Some people cough as soon as their feet become cold or whenever their body surface is exposed. This suggests the therapeutic value of warm clothing in diseases complicated with a cough. The existence of a

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stomach cough is very doubtful, although the lay public believe in it very thoroughly. Cough has not as yet, however, been experimentally excited from the stomach, and the so-called stomach cough which is so common in drunkards can probably be most satisfactorily explained by assuming that an affection of the pulmonary passages is combined with the stomach disorder.

Nervous Cough.—The so-called nervous cough merits a few words. We cannot deny the possibility of a nervous cough if we consider that a cough can be produced by an abnormal excitability anywhere in the course of the coughing reflex arc. When such reflex excitability is accentuated sufficiently, even physiologic irritants may cause a cough. As a matter of fact, a purely nervous cough is a very rare occurrence, and should only be diagnosed as such when it is observed in an exquisitely nervous or hysteric person, and when at the same time it differs decidedly from the ordinary type of cough. Under no circumstances is such a diagnosis justifiable from exclusion—*i. e.*, because the cough is the only sign of disease of the respiratory tract that can be made out from physical examination. It is well known that a cough frequently precedes all other signs of tuberculosis by weeks, often by months. (See under Barking Cough).

Much more frequent than a purely nervous cough is one which is disproportionate to its cause—*i. e.*, secretion or inflammatory irritation of the air passages—on account of an abnormal excitability of the coughing reflex. In such cases soothing remedies afford great relief.

On account of the rarity of other causes, a cough is usually an important symptom for the recognition of some state of pathologic irritation in the region of the respiratory branches of the sensory vagus. This irritation is in many cases produced by the accumulation of secretion in the air passages. Experience teaches us that a cough caused by accumulation of secretion differs from the other types of cough. This difference is often appreciated by the laity. Its task, which is generally fulfilled, is to remove the secretion. It thus acquires a peculiar ring, recognized as a combination of the noise of the cough explosion with the noise of the moving secretion. (See later Râles, Rattling.)

Such a cough is called a **moist** or **loose cough** as contrasted with a **dry cough,** in which either the secretion is too tenacious to be set in motion or no secretion exists. The acoustic distinction between a moist and a dry cough is of considerable diagnostic importance, because the secretion is by no means always expelled from the mouth by the cough, and so appreciated by the patient and the examiner, but very frequently after leaving the larynx is unconsciously swallowed.

There are certain other important peculiarities in a cough which may point to the cause of the disease.

The **peculiar rough barking cough** observed in simple laryngitis is characteristic of swelling without marked ulceration of the vocal cords. Here the voice may be clear, but it is usually very hoarse or aphonic. The barking tone is probably due to the swelling of the false cords which aid in closing the glottis. A similar barking is sometimes

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observed in the cough of hysteric individuals. Here the laryngoscopic examination proves that it is due to some abnormal innervation without any swelling. With a certain amount of practice one can really cough with a bark by adding phonation to the cough impulse. Like most other hysteric phenomena it can be reproduced voluntarily. If not associated with swelling of the larynx, a barking cough suggests its hysteric nature.

If the margin of the vocal cords is irregular from deposition of secretion or from ulceration, the cough is equally rough but not barking, and the voice is hoarse. If the closure of the glottis is imperfect on account of marked ulceration of the cords or on account of paralysis of the approximating muscles, or, if there is paresis of the expiratory muscles or general debility, the cough is noiseless. This peculiarity is observed in phthisis of the larynx, in paralysis (bulbar paralysis, myelitis), in any patient who is severely ill or weak. If a patient is unable to approximate the glottis perfectly, he may instead be compelled to close the mouth for the purpose of coughing, and then the resonation from the distended buccal cavity will furnish a very hollow ring to the cough. Such a hollow ringing is so generally observed in advanced phthisis that even the laity appreciate its prognostic significance.

A **hacking cough** consists of a series of very weak, frequently repeated coughing explosions. It is to be attributed to the mildness of the irritant, which in these cases is usually continuous and is not dependent upon any great amount of secretion. A hacking cough is most commonly observed in chronic catarrh of the upper air passages, in pharyngitis and laryngitis, and especially in beginning pulmonary tuberculosis; hence its importance.

Conversely, **violent coughing paroxysms** are observed in acute, intensely irritated conditions of the air passages from acute inflammations or from foreign bodies in the air passages; in "swallowing the wrong way"; in affections of the air passages associated with a profuse secretion, especially where cavities or bronchiectatic dilations empty themselves periodically (see p. 25), and finally in *whooping-cough*. In the last-mentioned affection we may attribute the cause to the abundant production of glary mucus and to the increased irritability of the nervous coughing mechanism. The "*whoop*," so important for diagnosing this disease, is a resounding crowing or whistling inspiration combined with a spasm of the glottis, and separates individual groups of coughing attacks. The glottis spasm is evidently due to some radiation of the irritation from the coughing center to the neighboring structures of the central organ, and depends merely upon the intensity of the irritation. During violent coughing paroxysms of other diseases we sometimes observe resonant inspirations very much like this whoop, but so rarely that the "crowing inspiration" is the most important diagnostic sign of whooping-cough.

Vomiting sometimes complicates very intense coughing paroxysms; it is due to the central diffusion of the irritation.

Hemorrhage into the skin or mucous membranes, unconsciousness,

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or even epileptic convulsions may be produced by the general venous congestion due to compression of the intrathoracic veins.

A physician must be very careful in taking the history of a patient in regard to coughing. So long as the cough or the hacking causes neither inconvenience nor discomfort, patients, especially the phthisical, persistently deny that they cough even when the physician hears the hack himself. By imitating the sound and movements of such a "hack," a physician can sometimes get the patient to acknowledge that he does "hack," but that he never considered it a cough.

It must, however, be acknowledged that some cases of phthisis or bronchitis with very intense inflammation of the lungs occur without any cough or even a "hack." Here the secretion is probably brought up to the vocal cords by the ciliary motion of the mucous membrane, and is then removed by "clearing the throat." If, as frequently happens, the secretion is swallowed, the patients neither cough nor even expectorate.

LOCALIZED PROMINENCE OF THE CHEST IN COUGHING.

As a result of the marked positive pressure and variations in the chest interior which are combined with the act of coughing, certain pliable portions of the thorax may be pushed forward at the commencement of the coughing spell in quite a remarkable way. If the glottis is closed, expiratory pressure distends the upper part of the chest chiefly, because the expiratory power attacks the lower thoracic aperture. In coughing, therefore, we generally observe a bulging forward of the upper intercostal spaces and apices of the lungs. Such bulging is especially prominent in emphysema because the pulmonary resistance is impaired. The portion of the lung situated above the level of the clavicles shows an increase in volume, so that during a cough in emphysema we frequently notice that large swellings are produced above the clavicles. This phenomenon should not be confounded with the distention of the jugular veins during a cough, which is also oftentimes quite pronounced (p. 146 *et seq.*). If the lung tissue is infiltrated and shrunken, coughing will evidently not produce such an inflation. Hence, a careful inspection of the supraclavicular fossæ sometimes furnishes important conclusions as to the condition of the apices in beginning tuberculosis.

PALPATION, SPHYGMOGRAPHY, AND SPHYGMOMETRY OF THE ARTERIAL PULSE.

EXAMINATION of the arterial pulse is of great diagnostic importance because it informs us of many things, such as the cardiac innervation the force of the heart, the blood-pressure, and the condition of the peripheral arteries, and sometimes suggests the existence of valvular diseases of the heart or of fever. Many individual peculiarities must be considered and various methods employed to recognize them.

The commonest method and the one employed almost exclusively by practitioners is manual palpation of the arterial pulse.

Sphygmography and sphygmomanometry are less used. Auscultation of the arterial pulse is much less important.

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PALPATION OF THE PULSE.

Any superficially placed artery may be utilized for the purpose of palpation; but we nearly always choose the same vessel, so as to have a certain uniform standard, experience, and practice in judging of the pulse. On account of its accessibility, the radial artery is usually selected. It is palpated between the styloid process of the radius, or the tendon of the supinator longus and the tendon of the flexor carpi radialis. An anomalous position of the artery or of the tendons will, of course, necessitate our trying another place. In doubtful or difficult cases the two radials should be compared, so as not to attribute some abnormality really due to local causes to an alteration in the entire circulatory condition. One radial not infrequently seems smaller than the other, while in reality it is merely a small branch which lies in the ordinary place of the radial, and the main trunk takes some abnormal course.

The best method of palpating the pulse is to put the tips of three adjoining fingers of the more adept hand along the artery and to vary the pressure. With every heart beat the artery is found to expand and to lift the fingers. This lifting is what we call the pulse. Palpation with one or two fingers is sufficient to determine the frequency and the rhythm; but we need three fingers for judging the form of the pulse wave and the height of the blood-pressure.

CHARACTER OF THE ARTERIAL WALL.

Palpation of the arterial wall permits us to determine whether arterial sclerosis is present or not, and whether the pulse wave is modified by any factors in the wall itself.

The most important thing to determine is the amount of elasticity —*i. e.*, the rigidity of the arterial tube. This can be most effectually determined by rolling the artery back and forth under the finger and by running the finger along the length of the artery, at the same time being careful to prevent the influence of the blood-current by occluding the artery with another of the fingers. The artery is *soft* and *elastic* in young and healthy individuals; but in *arteriosclerosis* or where the blood-pressure is permanently raised by an increase of the vasomotor tonus (chronic nephritis, lead-poisoning) we can often distinctly appreciate the *increased resistance* of the walls. The tortuous character of the artery so frequent in these conditions is produced by a fresh deposit of histologic elements in its wall. At the same time some arteries may be tortuous without any arteriosclerosis—*e. g.*, the temporal arteries. The deposit of lime in the wall, which is observed in very pronounced cases of arteriosclerosis, can be felt very distinctly as rough, hard irregularities. Although palpation is diagnostically important in determining the existence of arteriosclerosis of the peripheral arteries, it does not necessarily show whether the aorta, coronary arteries, or other deep-seated vessels are normal or not. The arteriosclerotic changes may

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be very unevenly distributed, and the radial artery is one which shows no marked predilection for arteriosclerosis. Therefore, to demonstrate arteriosclerosis we should palpate as many as possible of the superficial arteries. Arteriosclerosis must not be absolutely excluded, even when the results of palpation are negative.

A persistently high-tension pulse is one of the most reliable indications of diffuse arteriosclerosis when the examination shows no other cause for it (nephritis).

CHARACTERS OF THE PULSE.

FREQUENCY OF THE PULSE.

The pulse frequency—*i. e.*, the number of beats in a minute—is estimated by counting the radial pulse. It is advisable to count an irregular pulse for at least one minute and then to repeat, for otherwise the count may not be accurate. If repeated counts furnish different figures, the extremes should be noted.

A great many influences affect pulse frequency, so that it is advisable to estimate the rate under conditions which are as nearly alike as possible, or, in case this cannot be done, to make allowance for the action of these influences in forming an opinion. In sensitive people any mental excitement whatsoever decidedly influences the pulse rate. The physician's entrance is enough to cause a marked rise in the pulse, so that at the bedside it is advisable to delay counting the pulse until after having conversed with the patient for awhile.

Any bodily exertion or movement increases the pulse rate. After running, gymnastics, fencing, or mountain-climbing the rate may be very greatly increased and still be within physiologic bounds. Even the quieter motions in bed, voiding urine, or evacuating the bowels, materially increases the pulse rate in very sensitive or very ill patients. After moderate activity the increase in rate soon subsides, but after prolonged, fatiguing exertion it may persist for some time. The pulse rate, furthermore, depends upon the position of the body. After lying down, sitting up increases the frequency; so does standing up after sitting. This rise may be only transitory, due to the muscular exertion attendant upon the change of position, or to some extent permanent so long as the position is maintained.

Guy found that in fasting, healthy men who had previously rested, the pulse rate was 66 to the minute while prone, 71 while sitting, and 81 when standing.

Digestion of food increases the pulse rate for several hours during the period of digestion, the amount of increase varying according to the quantity of food. The daily variations of the pulse are only partly due to the influence of the meals; for daily variations approximately parallel to the daily variations of temperature are observed even in fasting individuals. They usually amount to only a few beats.

Generally speaking, a high blood-pressure produces a slowing of the pulse, a low blood-pressure an increase in its rate. There are, however, many exceptions to this rule, although it undoubtedly possesses some physiologic importance. Marey

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attributes the increased pulse rate in standing up as contrasted with lying down to the difference in blood-pressure. This, he believes, is much higher in the recumbent posture.

Respiration usually influences the pulse rate; during inspiration it is increased, during expiration diminished.

Coughing or Valsalva's experiment produces a marked increase in the pulse rate. In the latter the increased frequency persists longer than the increased intrathoracic pressure.

The time of life has a very distinct influence upon pulse frequency. Rollet[1] quotes the following figures, collected from various observers, as averages:

Age.	Pulse beats to minute.	Age.	Pulse beats to minute.
End of fetal life	144–133	10th to 15th year	91–76
Newborn and 1st year of life	143–123	20th–60th "	73–69

Vierordt[2] gives the following detailed table for childhood:

Age.	Pulse beats per minute.	Age.	Pulse beats per minute.
1 year	134	7– 8 years	94.9
1–2 years	110	8– 9 "	88.8
2–3 "	108	9–10 "	91.8
3–4 "	108	10–11 "	87.9
4–5 "	103	11–12 "	89.7
5–6 "	98.0	12–13 "	87.9
6–7 "	92.1	13–14 "	86.8

The pulse rate practically diminishes with the advancing years until the age of sixty, after which time it begins to increase again slightly. Sex also has some influence. According to Guy, women average 7 to 8 beats a minute more than men of the same age. In individuals of the same age and sex the pulse rate varies according to the height; it is slower in tall than in short persons.

Sometimes the pulse count at the wrist is less than over the heart. In such an event the latter must be the accurate measure of the heart's frequency, and we naturally conclude that the cardiac power has become affected, so that some of the pulse waves are not transmitted to the peripheral arteries. Such an omission of individual beats makes the pulse sequence irregular, so that the examiner then turns to the heart. If the radial pulse is perfectly regular we should not think of the existence of any difference between it and the heart-beat unless only every other beat was strong enough to be transmitted to the radial artery, such as in the rare condition called pulsus alternans and bigeminus, heart bigeminus, hemisystole, systolia alternans (see later section upon Cardiac Impulse).

The pulse frequency, like the body temperature, can be conveniently represented upon a chart in the form of a curve. The variations of both can thus be very accurately compared at a glance.

Increase of Pulse Frequency; Tachycardia.—*Fever* is one of the most common causes of an accelerated pulse rate. The tempera-

[1] Hermann, *Handbuch der Physiologie*, vol. iv., 1.
[2] H. Vierordt, "Daten und Tabellen," Jena, G. Fischer, 1888.

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ture and the pulse curve in fever usually run parallel. Liebermeister estimated that the pulse frequency was increased by about eight beats for every degree of temperature. So long as pulse and febrile disturbance run parallel in this way, such a harmonious preservation of the functions may be regarded as relatively favorable. On the other hand, the more the acceleration of the pulse exceeds the elevation of temperature the graver the prognosis, because too rapid a pulse rate usually means some serious damage to the circulation, either to the heart or to the vasomotor system. If the patient is resting quietly a pulse of 140 to 160 in fever is always of grave significance. Under various conditions of fever the pulse frequency and the temperature may follow an

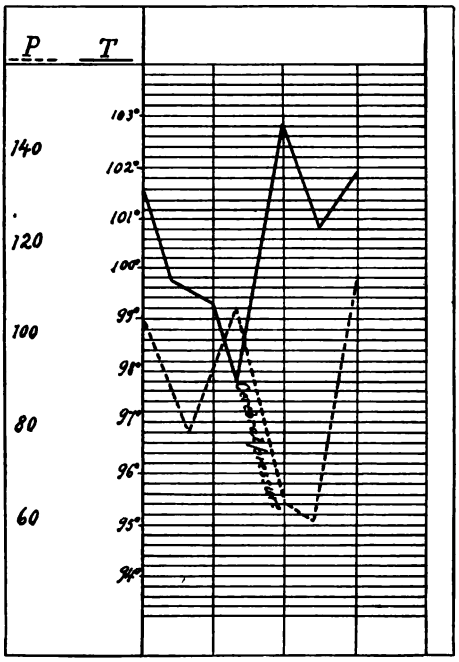

FIG. 33.—Relation of temperature and pulse frequency in tuberculous meningitis.

entirely different course. Such a divergence of the curves is of very great diagnostic importance. A high temperature with a slow pulse is observed chiefly in *febrile brain diseases*, in which the pressure upon the brain is responsible for the slowing of the pulse as in *tubercular meningitis* (Fig. 33); again in a *combination* of a *febrile disease* with a *cardiac disturbance* which causes brachycardia (adiposity, sclerosis of the coronary arteries, myocarditis). The converse, a high pulse rate with an abnormally low temperature, is characteristic of the symptom complex of acute circulatory weakness included in the name *collapse* (see Fig. 32).

There are numerous other exceptions to this rule of parallelism of the two curves, although most of them are less pronounced than the example just cited—*e. g.*, in typhoid fever the pulse frequency is noto-

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riously moderate as compared with the height of the temperature (see Fig. 24). This peculiarity is often serviceable in differentiating typhoid fever from acute miliary tuberculosis or septicopyemia. For in the last two mentioned the pulse rate is almost always accelerated as much as, if not out of proportion to, the rise of temperature. Conversely, in pulmonary tuberculosis a very high pulse rate is frequently observed with only a moderate rise of temperature or even with no rise at all. Yet the rectal is often unexpectedly higher than the mouth temperature. Children usually exhibit a relatively rapid pulse rate with fever.

A rapid pulse rate is, furthermore, found in affections of the heart or of the nerves. Valvular diseases in the stage of disturbed compensation—endocarditis, pericarditis, exophthalmic goiter, nervous palpitation, nervous tachycardia, dislocation of the heart by processes in its vicinity which limit the space—all of these diseases are or may be associated with a more or less considerable increase of the pulse rate, to a certain extent proportional to the severity of the affection. Very imperfect explanation has been found for the cause of the increased pulse rate in these cases. The author considers that the cause of the increased pulse frequency in the clinical picture of so-called "paroxysmal tachycardia" is due to an epileptoid discharge in the neighborhood of the cardiac accelerating nerves, although he realizes that such an opinion is at variance with Martius.

An increased pulse rate also occurs combined with all sorts of pain. Under some conditions not yet understood the reverse sometimes takes place—*i. e.*, a slowing of the pulse (see p. 104). The reflex influence upon the cardiac nerves is the probable cause in both cases.

Atropin and alcohol are the two most important of the numerous poisons which accelerate the rate of the heart.

Not infrequently, under pathologic conditions, the acceleration of the pulse due in a healthy individual to physiologic influences (p. 100 *et seq.*) may reach an excessive degree so far as duration and intensity are concerned. This always points either to some damage to the heart itself or to its nervous system. Thus, in chlorotics or in other weakly individuals even very slight exertion (stair climbing) will greatly increase the pulse rate; and this increase usually is associated with the subjective sense of palpitation of the heart or of dyspnea.

Diminution of the Pulse Rate (Bradycardia; Brachycardia).—A noticeably slow pulse does occur, although rarely, as an individual, non-pathologic peculiarity in a healthy person. A very pronounced slowing of the rate, to as low as 20 or less beats to the minute, is observed pathologically in certain diseases of the heart muscle, especially in the *"fatty" infiltrated heart* and in *sclerosis of the coronary arteries.* A slight degree of retardation is sometimes observed in *compensated aortic stenosis. Cachectic individuals* usually exhibit not only a low temperature but also a slow pulse rate (carcinoma of esophagus, etc.). Similar results are observed in convalescence.

A temporary slowing of the pulse (to a little below normal) is often found in acute febrile diseases after the crisis. When, despite the criti-

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cal drop of temperature, the pulse remains high, there is always reason to suspect a "pseudocrisis" (see Fig. 23). In certain painful affections (*e. g.*, gall-stone colic, lead-colic) a slowing of the pulse is more frequently observed than an increase. Icterus often produces retardation, due to the toxic action of the biliary salts. This, however, usually disappears when the jaundice is prolonged, either because the heart becomes accustomed to the intoxication, because the production of the biliary salts is diminished, or because their elimination is more complete. The slowing of the pulse in acute cerebral pressure is of particular diagnostic importance (meningitis (Fig. 33), fracture of the skull). Chronic cerebral pressure usually does not cause any slowing of the pulse unless acute exacerbations occur. *Shock* sometimes causes an excessive slowing of the pulse. Rapid emptying of peritoneal or pleuritic effusions sometimes diminishes the pulse rate.[1] Some drugs (digitalis, etc.) produce a slowing of the pulse.

PULSE RHYTHM.

Under normal conditions the pulse is regular or rhythmic—*i. e.*, the individual pulse waves follow each other at equal intervals of time. Only slight transitory deviations from such regularity come within physiologic limits, and then principally under conditions which modify the pulse frequency as well. More pronounced irregularities of the sequence of beats, or *arrhythmia*, are probably all pathologic, and indicate either some distinct lesion of the heart or some purely functional disturbance of its activity, such as occurs in all sorts of conditions with or without cardiac weakness.

We may differentiate between an *absolutely irregular pulse* (pulsus irregularis), which is usually at the same time much increased in rate (delirium cordis), and *a partially irregular pulse*. In one type of the latter the irregularities are uneven—*i. e.*, the intervals between the beats are sometimes shortened and sometimes lengthened without any sign of regularity. In the other type of the *partially irregular pulse* the irregularities are periodic—*i. e.*, they follow each other at regular and definite intervals.

Irregularities of rhythm are nearly always associated with inequality of the individual pulse waves—*i. e.*, a pulsus irregularis is at the same time a pulsus inequalis. It is therefore advisable to defer the consideration of the various types of irregular pulse until after we have studied and understood the differences in quality of the individual beat. (See Sphygmography, p. 122 *et seq.*, and p. 130 *et seq.*).

CHARACTERS OF THE INDIVIDUAL PULSE BEAT.

The proficiency which different observers attain in the determination of the quality of the individual beat by palpation varies decidedly with their practice and skill. He who is less adept may obtain more accurate

[1] Much more frequently a rapid weak pulse is the immediate result of tapping the chest or abdomen; the retardation follows later.—ED.

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information by employing sphygmography or sphygmomanometry. In addition to the regularity, the qualities which every physician should recognize by palpation without any especial instrument are: the size, the celerity, the tension, and dicrotism.

Size of the Pulse; Pulse Volume.—By size we mean the extent of the excursion which the arterial wall makes under the influence of the pulse wave. With a large, full pulse (pulsus magnus) this excursion is considerable; in a small, weak pulse (pulsus parvus) the excursion is small. (As to the significance of and the alterations in the volume of the pulse and the variations in the size of the individual waves, see Sphygmography, p. 124 *et seq.*)

What we designate as the pulse volume Marey calls its strength, and with a certain amount of justice; because, of course, the vital power of the rise corresponds to the size of the pulse wave—*i. e.*, to the amount of matter set in motion. This corresponds to the terminology employed in describing wave motion, where the strength of a vibration is measured by its amplitude. For clinical purposes, however, the author prefers the term "volume," because the word "strength" would often suggest that we were expressing a certain opinion about the heart's activity or power, and this is by no means always the case, as we shall see later (see Sphygmography).

Celerity of the Pulse.—By celerity is meant the rapidity of the rise and subsidence of the individual pulse wave. A quick pulse (pulsus celer) is one whose wave rises quickly and descends quickly; a sluggish pulse (pulsus tardus) is one whose wave rises slowly and descends slowly. Upon the fingers a *pulsus celer* produces the impression of a quick, sharp rap. This peculiarity is appreciated most distinctly when the pulse wave is at the same time of considerable volume. Therefore, what is often called a pulsus celer is generally a full pulse as well. The pulse of aortic insufficiency is the most striking as well as the commonest example of a pulsus celer. It very frequently can be appreciated even by the eye as a very striking pulsation of the artery, and the diagnosis of aortic insufficiency made correctly at a distance. The rapidity of the ascent and that of descent of the pulse wave do not take an equal part in producing this "rapping" sensation. For although the rapidity of the rise may be recognized without any particular training, it requires considerable practice to recognize the quickness of the drop. Von Frey claims that the rapidity of the descent of the pulse wave can be estimated only by means of the sphygmograph and not by palpation. But the author believes that an expert physician can perfectly well differentiate between a pulse whose sharp, bounding quality is due only to a rapid onset of the wave and a pulse with a rapid descent as well as ascent. The terms "pulsus celer" and "pulsus tardus" are especially well adapted to the cases in which both limbs of the pulse wave are either steep or blunt. Where either the ascent or the descent alone is rapid or slow this peculiarity should be specified. Thus, a pulse may be *celer* in its ascent and *tardus* in its descent. Such an application of the terms "celer" and "tardus" to the same pulse is no contradiction. These conditions are explained more fully upon p. 124 by Sphygmography. (Figs. 47–51.)

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Pulse Tension.—Consideration of Blood-pressure.—We consider under the term "pulse tension" the qualities of the individual pulse wave which show the amount of pressure in the arteries. The difficulty is, however, that the term blood-pressure may be understood in quite different ways. The physiologist ordinarily uses the word to mean the average or mean blood-pressure as estimated by the usual manometers. But the maximum or systolic and the minimum or diastolic blood pressure must also be considered. In a healthy man or animal the variations of blood-pressure from the average are very slight, and so we ordinarily speak of "blood-pressure" without further remark when we strictly mean average blood-pressure. We do not know, however, whether the systolic variations in pressure under pathologic conditions are so very slight. Sphygmographic and sphygmomanometric observations (see later) seem to show that they are considerable. Therefore, under pathologic conditions, the systolic or maximum, the average or mean, and finally the diastolic or minimum pressure should be considered separately. The writer believes that many apparent contradictions in palpatory, sphygmographic, and manometric estimations of pressure (see p. 135) can be explained by a disregard of such conditions. A more distinct and uniform conception of the terms expressing the pulse tension is therefore essential, and these terms should be clinically correlative to blood-pressure. Just as we distinguish between a maximum, middle, and minimum blood-pressure, so should we differentiate between a maximum or systolic, an average or mean, and a minimum or diastolic tension of the arteries. The ordinary definition that tension is the measure of the amount of finger pressure which is necessary to compress the artery is not accurate, and is, therefore, inadequate to teach us how to palpate pulses correctly. It is better to define the maximum tension of an artery as the amount of force which is necessary to prevent the transmission of the pulse wave to the periphery,[1] the minimum tension as the power necessary to compress an artery during cardiac diastole, and the average or mean tension as a certain average amount of power which will suffice to close the artery between systole and diastole. A disregard of the various meanings of the term arterial tension thus explains the variations in the description of one and the same pulse, one observer calling the tension high, another calling it low.

As the manner of palpating an artery varies, so do conclusions as to the pressure conditions in the artery differ. Usually (although only partly justifiably) most importance is attached to the maximum or systolic tension of the artery. This is determined ("dynamic" procedure of taking the pulse) by palpating the artery with three adjoining fingers along the long axis of the vessel. The distal finger compresses the artery so that no recurrent pulse wave can reach the vessel from the periphery. The proximal finger exerts a gradually increasing pressure upon the artery until the middle finger, which should rest quite gently upon the vessel, can no longer feel the wave. The power used is the measure of the (heart)

[1] See the following pages for the doubts about identifying this exercise of power with the systolic pressure.

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systolic or maximum tension of the artery. This procedure is imitated by von Basch's method of sphygmography (p. 134 *est eq.*). The physical accuracy of his method of determining the systolic pressure—*i. e.*, the correctness of identifying the amount of pressure employed with the systolic pressure—is questionable (p. 135). The same doubt applies to this dynamic method of palpation, for the reason that, besides the systolic increase of pressure, the vital energy of the pulse wave has an important influence upon the result obtained (p. 135 *et seq.*). This vital energy is closely dependent upon the *size* of the pulse wave (p. 135 *et seq.*), and the size in turn by no means runs parallel with the blood-pressure; in fact, it is often inversely proportional to the latter. We have not only the systolic blood-pressure to overcome in the procedure, but, generally speaking, a much greater pressure, because the recoil of the pulse wave at the compressed point of the artery acts very much as a water-ram. On account of the *size* of the pulse wave, the excess of energy employed above the systolic pressure is oftentimes greatest with a low blood-pressure. Consequently this dynamic method is frequently responsible for questionable results, for it is often hard to compress an artery if the pulse is full (large), even though the tension is low, while conversely, a small (weak) pulse may frequently be easily compressed even if the blood-pressure is high.

The following "static" method for determining the minimum or diastolic arterial tension is probably more reliable. If the fingers exert but slight pressure in palpating the artery we notice that the appreciable pulsatory excursions of the artery are usually small, and that they increase as soon as the pressure is increased until a maximum excursion is reached. After this point still greater pressure again diminishes the size of the pulse. The amount of pressure necessary to produce a maximum excursion corresponds, in all probability, to the minimum or diastolic pressure in the artery. This may be explained as follows: The reason why the excursion of the artery increases as we increase the pressure upon its wall is evidently because a part of the pulsatory increase in pressure is absorbed by the tense arterial wall when the artery is not compressed; whereas, if we remove the tension from the arterial wall by contrapressure, the pulse wave will be transmitted to the palpating finger with a minimum amount of loss. The greatest excursion will evidently coincide with the complete relaxation of the arterial wall at the onset of the pulse wave—*i. e.*, when the pressure exerted corresponds to the minimum pressure of the artery. Besides this, when pressure is brought to bear upon the artery from without which exactly corresponds to the minimum arterial pressure, or, more strictly speaking, is the slightest amount in excess of it, the artery will then be occluded at the instant of the arrival of the pulse wave, and, of course, the latter will back up considerably from reflections, so that the excursion of the wall will be increased in this way too. Any compression above this point will, of course, diminish the excursion again, since the pulse wave will no longer be capable of overcoming the resistance of the pressure fingers. The digital pressure exerted to produce the greatest

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pulse excursion possible by this method is considered as the measure of the minimum arterial pressure. It is important to estimate the size of the arterial excursion, and not its strength or force.

Beside these unavoidable inaccuracies we can readily appreciate the fact that an especial skill in appreciating the relations of pressure accrues to the careful clinician only after years of practice and a long experience in contrasting and remembering differences in pressure. Another difficulty is that, according to Pascal's law (law of hydraulic pressure), the amount of pressure necessary to compress an artery is proportional not only to the arterial pressure, but also to the diameter of the artery. On account of such varied and manifold difficulties in judging arterial tension, it is advisable to supplement palpation by sphygmography (p. 129 *et seq.*).

Custom has come to employ the expression "hard" or "tense" pulse (pulsus durus) as synonymous with increased arterial tension, and "soft" or "relaxed" pulse (pulsus mollis) with diminished tension. Of course, we must not confuse these with the same terms applied to qualities of the arterial wall (softness *versus* rigidity of the artery) (p. 99). Again, these terms may at one time be applied to the systolic, at another to the diastolic, pressure. In nephritis and arteriosclerosis the diastolic as well as the systolic pressure is ordinarily increased; in fever usually the diastolic is low while the systolic is more frequently high; *in disturbances of compensation* systolic and diastolic pressure are both low.

Dicrotic Pulse.—We speak of a pulse as being dicrotic when a second wave follows immediately after the principal rise. (The nature of this will be discussed under Sphygmography, p. 113 *et seq.* and p. 129 *et seq.*) A dicrotic pulse furnishes a certain sense of after beating to the palpating finger. It is usually associated with diminution of the minimum pressure. Both the primary and the secondary wave are felt to be greatest when the palpating finger presses only slightly; therefore a dicrotic pulse is best appreciated with gentle pressure. This can be readily understood from the explanation given of the excursion of the arteries (p. 106 *et seq.*). The beginner is apt to employ too great pressure to recognize readily such a condition.

Combined Qualities of the Pulse.—We have a number of terms which refer to a pulse which comprises two or more of the qualities already described. Of these we shall mention only those in most common use: pulsus fortis, strong pulse = large + tense (see also p. 105); pulsus plenus, full pulse = large and medium hard; pulsus debilis s. inanis, weak or feeble pulse = small + weak; pulsus undosus = large + soft; pulsus serratus = large + tense + rapid; pulsus vibrans = very large + very tense. This name is applied because the so-called elasticity elevations (see p. 116) may be distinctly appreciated by palpation. These terms are fitting enough, but rather unnecessary. Instead of employing them, it is better for a beginner to mention the individual qualities one after another. The Latin terms are sometimes combined. For instance, we speak of a pulsus tardodicrotus or a pulsus magnodurus, etc. Such

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combination terms are practical enough in themselves, but some of them are not sufficiently clear, considering the double meaning of the terms "celerity" and "tension" (see above). In general a detailed description is better than an attempt to describe the pulse by one word. There are some terms employed to characterize peculiarities of a sequence of beats and not of individual beats. These terms as well as further details about pulse peculiarities will be described in connection with the Sphygmographic Curves (p. 122 *et seq.*).

SPHYGMOGRAPHY.

Sphygmography is the method of registering the pulse wave of some peripheral vessel, generally the radial artery, upon a moving surface (usually smoked paper) by means of a special instrument, the sphygmograph.

Vierordt constructed the first sphygmograph, and since then a great many improved instruments have appeared. Most of them, however, have retained Vierordt's principle of transmission by means of a lever. The best known and most frequently employed sphygmographs are: Marey's, for a long time the only one used clinically; Landois', Sommerbrodt's, Riegel's, Dudgeon's, Jaquet's, and v. Frey's. Dud-

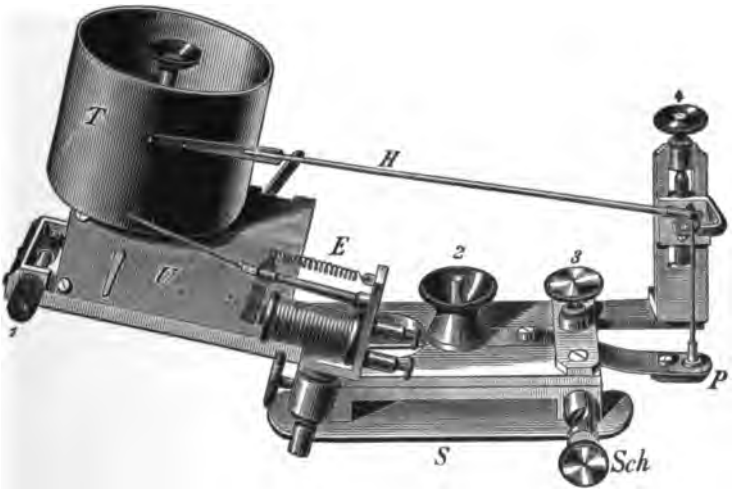

FIG. 34.—v. Frey's sphygmograph.

geon's apparatus has been a favorite in recent years, because with it curves of considerable excursion are easily traced. Jaquet's is an improvement upon this pattern, including a really excellent mechanism for marking intervals of time. v. Frey's sphygmograph,[1] also a very excellent instrument, is depicted in Fig. 34.

It possesses this advantage over other instruments: the motions of the pulse are transmitted to the writing-lever as simply as possible.

[1] v. Frey, *Die Untersuchung des Pulses*, Berlin, 1892, J. Springer.

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The method of employing this instrument is as follows: The most superficial part of the radial artery is marked upon the wrist with a skin-pencil; and the apparatus is adjusted so that the "pelotte" impinges exactly over the indicated artery, and with the drum toward the elbow. It is then fastened to the forearm by means of the carrier or slide, *S*, with a strap. The carrier (Fig. 35) is fastened separately with the hooks that are attached for this purpose. The screw *Sch* serves to fix the strap. Loosening screw 2 makes it possible to move the entire apparatus upon the "carrier" This is very convenient for the purpose of adjusting the "pelotte" more accurately. By means of screw 3 we attempt to regulate the amount of pressure upon the pad until the lever needle begins to make excursions. The apparatus is then firmly fixed in place by tightening screw 2.

The drum has, of course, been removed from the apparatus before this, covered with wax paper, smoked[1] by revolving over a flame, and then replaced upon the clock mechanism, *U*. By turning key 1 the

FIG. 35.—The carrier or slide of v. Frey's sphygmograph.

drum can be moved so that the bent point of the registering-needle rests lightly upon the smoked paper. The height of its excursion and, hence, of the curve can be regulated by turning screw 3, which controls the tension of the "pelotte." The clockwork is then started by means of the lever which is visible behind the writing-lever *H*, to the right of the drum. The drum revolves and the bent point of the needle registers the sphygmographic curve upon the smoked drum. The drum is then taken off again, the paper carefully cut off, separated from the drum, and fixed in shellac or in a 10 per cent. solution of dammar balsam in benzin. The part *E*, which has not yet been described, is a small electromagnet with a registering-needle attached to the anchor, so that it registers upon the drum. When connected with an electric current, intervals of time or signals may be indicated in the same way as is customary in physiologic experiments. When it is not being used it may be disconnected.

v. Frey has recently modified his sphygmograph; and, it would seem, very advantageously. The chief improvement is the substitution of a delicate metal

[1] A pointed gas flame is perhaps the best source for smoking, or a kerosene lamp, or a piece of burning camphor. We must be careful not to smoke too thickly, because the friction between the registering-needle and the drum would be so great that the curve would be disturbed.

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spring for the joint connections, so that the motions of the registering-needle are less angular. Besides this, the drum is connected with a Jaquet's chronometer works, so that the time intervals are pictured. The curve may, of course, be registered by means of air transmissions upon a kymographion placed at some distance.

Jaquet's sphygmograph[1] (Fig. 36) consists of a metal frame, *Dp*, to which is sewed a cuff, *B,B*, for attaching to the wrist. The sphygmograph proper, *Ar*, is attached to the frame. The window cut out of the frame is to be applied accurately along the radial artery (previously marked out with a pencil), and the cuff then strapped around the wrist quite tightly. The sphygmograph proper is set into the frame by hooking into the hinge, *p;* and then the connection of the two parts is effected by pressing down at *r* and tightening the screw *m*. The pulse-registering apparatus consists of a short, broad spring, which presses upon the artery and transmits its movements to the registering-needle by means of the lever system *ef*. The screw *m* also serves to adjust the registering-needle at the desired height upon the smoked strip of paper. By screwing it down the spring *d* is pressed against the artery. The screw *c* is connected with an "eccentric" contrived to increase or diminish the pressure upon the spring. The amount of pressure can be determined by noting the position of the figures upon the screw. With this mechanism the instrument can be adjusted with practically equal pressure in each case, and taken away from the frame and reapplied in the same case without alteration of the pressure. Jaquet's instrument, like Dudgeon's, writes upon a flat and not upon a curved surface, as most other sphygmographs do. Another advantage consists in the very slight and constant amount of friction between the writing needle and the paper, so that there is very little trouble in adjusting the registering part of the apparatus. The paper is horizontal; it need not be especially strong; and it may easily be 40 or 50 cm. long. The little box *a* contains the clockwork which moves the strip of paper, *p*. This is started by pressing down on lever 6. The rate that the paper moves may be increased from 1 to 4 cm. in a second by altering the position of lever *a*. The slower motion produces a curve which presents a better general idea; the more rapid motion, one which may be more accurately analyzed. This rate of motion may be altered while the sphygmograph is in action. The box *a* also contains a stop-watch connected with the time-registering mechanism, *S*. The latter consists of a small pen which registers a mark upon the margin of the smoked ribbon every fifth of a second (Fig. 39).

The old style of the Jaquet sphygmograph, illustrated in Fig. 36, has the disadvantage that the individual parts of the pulse-registering mechanism are not connected firmly enough to insure their constant contact with every change in the rate of motion. At *e* and *d* the movements of the lever system are transmitted by loose joints. This is not a disadvantage when the excursions are slow, since the parts are held in contact by the metallic ball which is visible at the top of the apparatus. When the excursions are rapid (increased frequency of the pulse, great

[1] Described in *Zeit. f. Biol.*, vol. xxviii., N. F. x. Manufactured by Schüle, Mechanie to Physiologic Institute of Basel.

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elevation of the pulse), however, the oscillations of this mechanism may give rise to errors. These consist particularly of the appearance of artificial elevations in the pulse curve, dependent entirely upon the apparatus. One part of the lever system may advance independently until it meets with resistance; then, at the moment of the occurrence of this resistance, there is a jolt of the writing-lever, which appears as an artificial elevation in the curve, with no existence whatever in the pulse wave. In addition to the occurrence of these artificial elevations,

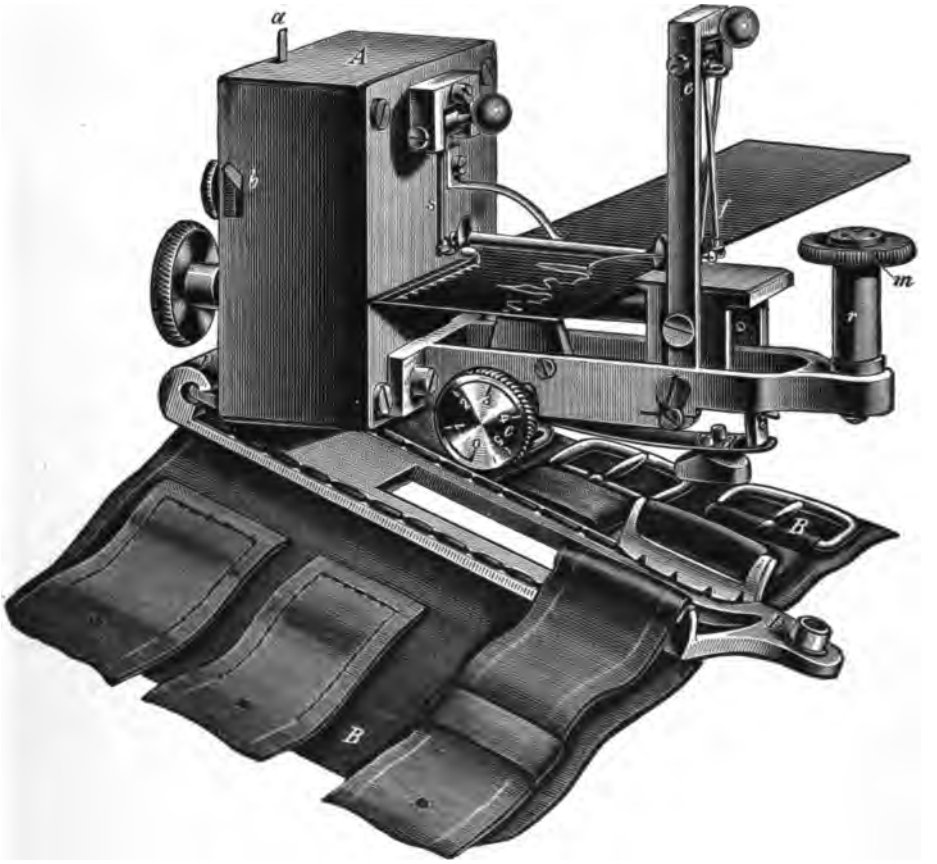

FIG. 36.—Jaquet's sphygmochronograph (old style).

the oscillation dependent upon the defective connections of the parts of the lever system also causes an increase in the height of the curve. As we shall subsequently learn (p. 125), it is difficult under any circumstances to gain an idea of the volume of the pulse from the height of the curve, and this error of oscillation renders the employment of the sphygmograph for this purpose still more questionable. A further source of error is the fact that the rate of motion of the needle at the moment when the individual parts of the lever system are not in contact is

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dependent upon the weight of the small metallic ball. Jaquet himself has criticised these faults of his apparatus, the lever system of which he copied from the Dudgeon sphygmograph, and has recently attempted to overcome them by a rearrangement of the recording mechanism, of which he gives the following description,[1] aided by the accompanying diagrammatic drawing (Fig. 37): "The movements of the needle *a* are transmitted to the bent lever *c c* by means of a wedge-shaped blade, *b*. The upper portion of the bent lever is marked at *s* by a carefully made screw-thread which works in a corresponding cog-wheel, bearing the writing-lever. The contact between the screw-thread and the cog-wheel is insured by a spring (*e*), which constantly presses the lever *c c* against the cog-wheel. In addition to this the bent lever *c c* does not turn upon an axis passing through its angle, as in the old instrument, but about the point *f*, and the axis of rotation is replaced by a short, flat watch spring, which insures the contact between the lever *c* and the edge *b*. This spring is so short that individual oscillations are not to be feared, since the length of such an oscillation is less than $\frac{1}{500}$ of a second, and is consequently trivial in comparison to the velocity of the pulse-registering mechanism. This spring also renders unnecessary the eccentric of the old instrument, since its tension is sufficient to overcome the resistance which the stretched skin offers to contact with the pulsating artery. The ratio of transmission in the short arm of the lever *c c* is 1 : 2, and that between the cog-wheel and the writing-lever 1 : 50, so that the total enlargement amounts to 1 : 100."

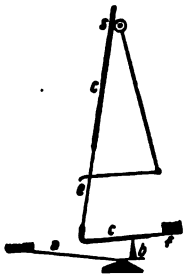

FIG. 37.—Diagram to explain the improved mechanism in the registering-lever of Jaquet's sphygmochronograph.

An artificial pulse has been constructed by Langendorff, upon a principle first employed by Donders, in which a revolving eccentric transmits to the pin of the sphygmograph excursions similar to those of an arterial pulse. Jaquet, by the use of this mechanism, found that his modified sphygmograph records the movements of the pulse accurately and without oscillation for all of the velocities usually encountered in sphygmography.

In spite of the marked improvements obtained by these changes, it seems to the writer that the construction of the modified Jaquet sphygmograph is still open to some objections, and particularly because there is not a firm connection between the blade *b* and the lever arm *c* (Fig. 37); if the velocity is very great or the pulse rate very rapid, the parts *c* and *b* may become separated, and thus still produce artificial elevations in the curve. The oscillating pad is a further disadvantage of the old instrument which has not been overcome in the new modification. In both the old and the recent models this pad is connected to the spring *a b* by a loose joint. This naturally simplifies the application of the spring to the artery, but from the lack of a firm connection it may also produce distortions of the curve. In order to obviate these faults the author had mechanic Schüle, who executed the improvements

[1] *Münch. med. Woch.*, 1902, No. 2.

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in Jaquet's modified sphygmograph, attach the pad firmly to the spring, as in Marey's and v. Frey's sphygmographs, and also connect the arm *f c* of the bent lever with the blade *b* by a small wire stirrup, which passes over the lever from the profile of the blade, so that any separation of the blade and the lever is rendered impossible. This mechanism was recommended to the author by mechanic Schüle; it acts like a hinge joint, but possesses none of its disadvantages. By these modifications the entire lever system has been united into a firm whole, in which no displacement is possible without implicating the entire system, and which must consequently accurately represent the movements of the arterial wall, disregarding the possibility of the oscillation of the entire system, a condition that the author has been able to exclude, for the velocities ordinarily encountered in sphygmography, by tests with Donder's artificial pulse. The jointed connection of the edge with the bent lever has the additional advantage that we may employ the eccentric of the original apparatus, which the author sorely missed in the improved Jaquet sphygmograph, since it not only greatly facilitated the application of the sphygmograph and rendered possible the necessary variations in the tension of the spring, but also permitted us to obtain in every pulse-tracing exactly the same amount of tension of the spring with the same position of the pencil. The writer believes that Jaquet's sphygmograph, with these different improvements, now answers all reasonable requirements. It, of course, produces lower curves than the old model, on account of the suppression of oscillation. Mechanic Schüle, of the Physiologic Institute at Basel, adds the described improvements to the old Jaquet sphygmograph for 28 to 35 francs, according to the condition of the instrument, and also furnishes the modified sphygmograph at the original price of 165 francs.

Compare p. 123 *et seq.* in regard to the degree of tension which is desirable upon the spring.

EXPLANATION OF A NORMAL PULSE CURVE; FACTORS WHICH INFLUENCE ITS FORM.

The curves obtained with the good modern sphygmographs usually correspond quite uniformly. Fig. 38 represents a normal pulse curve of the radial, the artery which is commonly selected. This illustrates what we have already learned from palpation, that the pulse wave is composed of a steep ascending and a rather slanting descending limb. Palpation alone might lead us to believe that the ascending and descending limbs of the wave present smooth lines, but sphygmographic tracings show a number of small elevations in the descending limb (so-called catacrotic elevations). Similar irregularities which may appear under pathologic conditions in the ascending limb are termed *anacrotic* elevations. A pulse with catacrotic elevations is termed *catacrotic;* one with anacrotic elevations, *anacrotic.*

The normal pulse has usually three distinct catacrotic elevations, and is therefore catatricrotic. The significance of the individual parts of the pulse curve, especially of these elevations, has created much

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discussion. Marey and Landois, and recently Hürthle, v. Frey, and Krehl, have taken the most active part in the dispute.

To understand the pulse curve we must, first of all, avoid confusing the progressive motion of the blood with the wave motion. This difference is very clearly illustrated by E. H. Weber's well-known expression, "Unda non est materia progrediens sed forma materiae progrediens."

In palpating a pulse or in employing a sphygmograph we study exclusively the wave motion of the blood. Of course, the wave motion has some connection with the conditions of the blood-current, but only indirectly. The pulse of a peripheral artery is a wave motion which has reached this vessel by transmission to the periphery of the primary wave arising in the aorta, long before the blood whose impulse produced the primary aortic wave has reached the artery. The rate of transmission of the wave motion of the blood is quite rapid, according to E. H. Weber about 9 m. a second. As we should expect from what has just been said, an aortic curve (from an animal) corresponds very closely to the human radial curve. It rises quickly, and falls off gradually with secondary waves and depressions. To understand the radial curve thoroughly, it is well to begin by analyzing the aortic curve. The wave

FIG. 38.—The normal radial pulse curve (Riegel).

motion in the aorta is evidently due to the fact that during the "expulsion period" of systole the blood-content of the aorta is increased. Afterward, when the aortic valves have closed, this increase is carried toward the periphery by the increased tension of the aortic wall. Thus the ascending limb of the aortic curve evidently corresponds in general to the so-called "expulsion time"—*i. e.*, the period during which blood flows from the heart into the aorta. The summit of the pulse curve, however, does not correspond exactly to the end of the "expulsion time"—*i. e.*, to the closure of the semilunar valves. On the contrary, the "expulsion time" is probably extended over into a part of the descending arm of the curve, because the flow of blood into the aorta is being diminished, so that the aorta is being partially emptied toward the periphery all the time. The end of systole is, then, not marked in a pulse curve, but lies in the descending limb of the curve, somewhere near the summit. The descending limb of the curve thus includes this remnant of the "expulsion time" plus the whole period during which the semilunar valves of the aorta are closed—*i. e.*, that part of systole which lasts after the close of the semilunar valves (Martius "persisting interval"); the whole of diastole; and the so-called "closure time" of

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systole. Hence, the descending limb is more than diastolic; but for the sake of brevity it may be called diastolic.

The secondary elevation of the descending limb, which is designated *a* in Fig. 38, is usually distinguished from the others by being very distinctly marked. If it is still further developed the pulse becomes dicrotic. It is therefore called the "dicrotic elevation or wave," and is pretty generally considered to be a "recoil elevation," coinciding with Landois' theory of its origin. His theory is as follows: At the moment when the primary positive wave leaves the aorta—*i. e.*, when the stretched aortic wall, in virtue of its elasticity, retracts again—this elastic contraction exerts an impulse upon the column of blood. This impulse strikes the closed semilunar valves, is reflected back, and passes through the aorta centrifugally to all the peripheral vessels in the form of a second positive pressure wave.

In cases where the duration of each pulse wave is sufficiently long for a complete formation of the pulse curve, the recoil wave may, in its turn, produce another second recoil wave. This second recoil elevation may be recognized by the fact that it follows about as quickly after the recoil elevation as the latter did after the primary wave.

Landois considers that *b* and *c* (Fig. 38), which are smaller elevations, are elasticity elevations—*i. e.*, due to individual vibrations of the arterial wall which are not transmitted from the blood-column to the arterial wall, but, conversely, from the arterial wall to the blood-column. (See p. 118 *et seq.* for a criticism of this conception of the secondary elevations.)

From experiments with rubber tubes Landois[1] states the following laws about both kinds of secondary elevations:

1. The further the artery is from the heart, the later the recoil elevation appears in the diastolic portion of the curve.
2. In the same artery, the further from the heart we apply the sphygmograph, the less pronounced is the recoil elevation.
3. The recoil elevation is so much the more pronounced at the heart the shorter (sharper) the primary wave, and *vice versâ*. The duration of the primary wave being equal, one of large volume produces a stronger recoil wave than one of small volume. If, however, such a large voluminous wave persists for some time, while a small wave lasts only a short time, the latter will produce the larger recoil elevation. The deciding factor is thus always the brevity—*i. e.*, the celerity of the primary wave.
4. Other things being equal, the recoil elevation is larger the lower the mean arterial pressure.
5. The further from the heart the examined artery is, the more marked are the elasticity elevations in the descending limb of the curve.
6. An accentuation of the mean pressure in the artery will increase the number of the elasticity elevations upon the descending limb and at the same time bring them nearer the summit of the curve.
7. When the mean blood-pressure is very low, the elasticity elevations disappear entirely.
8. In diseases of the vessels which affect or destroy the elasticity of the artery, the elasticity elevations are either much diminished or else disappear entirely.

The following assertions may be ventured in regard to the varying shapes of

[1] *Die Lehre vom Arterienpuls*, 1872.

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the entire curve. They are based partly upon experimental investigations by Marey, Landois, and others with tubes, and partly upon clinical observations :

1. Other things being equal, the curve is lower the higher the mean blood-pressure, and *vice versâ*. This can easily be understood, because under a high blood-pressure the arterial wall is already so tense even during diastole that the increase in pressure during systole can produce but a very small excursion. When, on the contrary, the pressure is low, the artery easily yields to the wave. Of course, this does not apply to those cases where the high pressure is produced by a large systole (left-sided cardiac dilatation and hypertrophy in compensated heart affections), nor where a low pressure is due to a small systole (disturbances of compensation).

2. Other things being equal (the same blood-pressure), the pulse curve is higher the larger the systole, and *vice versâ*.

3. Other things being equal (equal systole and equal blood-pressure), the ascending limb of the curve is steeper the quicker systole takes place.

4. Other things being equal, a low mean blood-pressure produces both a steep ascent and a steep descent—*i. e.*, a pointed curve (celerity of curve *in toto*). Conversely, a high blood-pressure produces a slanting rise and gradual descent (tardiness of curve).

5. Other things being equal, rigid arterial walls (like high blood-pressure) produce low curves with slanting ascent and gradual descent (tardiness). On the contrary, delicate elastic arteries (like low blood-pressure) produce curves with steep ascents and descents (celerity).[1]

FIG. 39.—Reduction of a sphygmogram to its simplest form. The above curve was taken with a Jaquet's instrument, the four waves at the left while the paper was moving slowly; the three at the right while moving rapidly. The time intervals are shown in the notched line above (0.2 seconds).

As a matter of clinical experience, however, it must be acknowledged that only the descending limb of the curve is affected by either arterial rigidity or blood-pressure, for the great working capacity of the heart is sufficient to make the rise of the curve steep even with rigid arteries and with a high blood-pressure. All these statements possess more theoretic than diagnostic interest.

Anacrotic elevations occur only under pathologic conditions. Landois has come to the following conclusions concerning them :

Anacrotic elevations—*i. e.*, secondary elevations in the ascending limb of the curve (Fig. 49)—are elasticity elevations. They depend upon similar influences to those which cause the ordinary elasticity elevations of the descending limb. The reason that anacrotic elasticity elevations are so rarely observed is because, as a rule, the ascending limb is so steep that elasticity elevations could not be reproduced in it. Hence, all the factors which tend to retard the rise of the curve are capable of producing anacrotic elevations, especially when these factors referred to also favor the formation of elasticity rises (compare above and p. 131).

The shape of the curve is decidedly influenced by the frequency of the cardiac action, because a quick sequence of the chief wave makes a complete formation of secondary waves in the descending limb impossible. At the moment that the systolic rise of a new wave begins, all secondary elevations disappear in the great rise of the new wave; although otherwise they would have developed in the descending

[1] A very good way of judging the real shape of a curve with many elevations is to divide each elevation in half and then join the points by a dotted line, as in Fig. 39.

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limb of the preceding wave. Curves with a slow sequence of beats are therefore generally richer in secondary elevations. An illustration of the action of the frequency of beats upon the pulse wave is the change of a febrile dicrotism of the pulse to hyperdicrotism (compare Fig. 51).

The facts mentioned above may be considered correct. At the same time Landois' explanation of the origin of secondary elevations is not generally accepted. Both v. Frey and Krehl have recently attacked it. These investigators studied the pressure curve by an artificially produced current impulse upon an animal's fresh aorta (left *in situ*). They registered simultaneously the manometric pressure curve from the beginning of the aorta and from the celiac axis. They found by a detailed examination that each individual secondary elevation may be explained by the fact that the primarily produced variations in pressure are reflected, as it were, from the periphery to the center and then back again to the periphery. Under certain conditions they may traverse this path repeatedly in the curve. According to this there would be neither recoil nor elasticity elevations; the so-called waves (even the anacrotic elevations) would be nothing more than centrifugal or centripetal reflections of the principal wave, which interferes with it in various ways as well as with each other. v. Frey and Krehl claim that these elevations, and especially the dicrotic rise in every vascular area, are due to centrifugal and centripetal impulses, but in different and complicated ways. They characterize the dicrotic rise practically as a reflected wave which arises late, and which is therefore very distinctly marked from the main wave; whereas they characterize the so-called elasticity waves which precede the dicrotic rise merely as waves derived by reflection of the main wave from points closer together, so that they are partly confluent with the main wave. Further, they characterize the elasticity elevations which come after the dicrotic rise as waves of reflection, like the latter, but arriving late and becoming less and less distinct toward the end of the curve, because the distance they traverse progressively increases.

It is questionable whether these theories will explain the relationship between the height of the blood-pressure and the so-called elasticity elevations and the dicrotic waves. But v. Frey and Krehl found that an increase of the blood-pressure pushed the reflected waves nearer to the main summit of the curve (*i. e.*, in the sense of time), because the rate of transmission of the waves increases with the blood-pressure. This explains the fact that those reflection waves which are situated nearest to the main summit (which are ordinarily described as elasticity elevations), and which include the anacrotic elevations, occur chiefly when the blood-pressure is high. It also explains the fact that with a low blood-pressure a reflection wave as a so-called dicrotic wave arrives very late and that it is characterized by being very distinctly formed, because at this moment the tension of the arterial wall is low enough for the wall to make quite a marked excursion. So we see that as a matter of fact these relations between blood-pressure and the form of the curve may be explained by the theory of the reflection of the secondary elevation.

INFLUENCE OF BREATHING UPON THE PULSE CURVE.

It has long been known that deep breathing may have some influence upon the pulse curve. This influence is chiefly due to the changes of arterial pressure which occur in the two phases of respiration. The respiratory increase in pressure

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manifests itself in a sphygmographic tracing by an elevation of the whole curve[1] and by an alteration in the shape of the individual beat (the latter corresponds to what was said in 4, 5, and 6, p. 116 *et seq.*)—viz., a diminution of the dicrotic rise, an increase of the elasticity elevations.

Authors differ very materially, however, as to whether the variations in pressure which are evident in a sphygmographic tracing belong to inspiration or to expiration. The reason for such a difference of opinion is that the factors which alter the blood during respiration are manifold; they frequently produce opposing results, and the final effect varies according to the way that the breathing progresses. The most important influence of breathing upon the blood-pressure is probably to be found in the change of diameter of the pulmonary vessels. They become wider during active inspiration and narrower during expiration.[2]

Consequently, so long as the dilating pulmonary vessels receive an excess of blood at the beginning of inspiration, the greater circulation must receive less blood (because the size of the diastole, and with it that of the systole, is diminished), and so the blood-pressure will fall. However, in the second part of inspiration the pulmonary circulation through the dilated pulmonary vessels improves; this in turn favors both diastole and systole, and eventually the greater circulation, and so increases the blood-pressure. During expiration the reverse occurs. The pulmonary vessels become narrower; they therefore empty a part of their blood into the greater circulation; the diastole and the systole of the left heart becomes fuller and the pressure increases. As soon, however, as the pulmonary vessels have become empty, the increased resistance in the lung will be felt as a factor in diminishing the diastole of the left side and the pressure in the general circulation will be reduced.

Therefore, under these conditions blood-pressure falls during the first half and increases during the second half of inspiration; whereas, conversely, pressure increases during the first half and diminishes during the second half of expiration. Hence, a maximum of pressure occurs at the beginning of expiration; a minimum, at the beginning of inspiration. These rules, however, apply only to very slow and deep breathing. Only the initial effect of the change of diameter of the pulmonary vessels is felt during rapid breathing—viz., expiration increases the pressure, inspiration diminishes it. Hence, respiration influences blood-pressure in two opposite ways, according to its rapidity. And especially in pathologic cases, where the breathing is abnormal, it is evident that the two types may be merged beyond recognition, so that we are unable to formulate any fixed rule for the relation of the sphygmogram to the respiratory phases. This is one of the causes for the diversity of opinions found in literature. As a matter of fact, we have considered only the most important factor by which respiration affects the arterial pressure—namely, the varying diameter of the pulmonary vessels, whereas in reality the conditions are much more complicated; for example, we might consider the influence of the varying intrathoracic pressure upon the heart and upon the great intrathoracic vessels, the effect of varying intra-abdominal pressure upon the vessels of the abdominal cavity, which changes with respiration, and further the changes of the vasomotor tonus synchronous with respiration, etc. Nevertheless, it may be stated, as a rule, that in the sphygmographic tracing (Fig. 40) deep and slow breathing increases the pressure during inspiration and diminishes it during expiration; whereas deep and rapid breathing produces the reverse effect. The pulse frequency is often accelerated during inspiration. Corresponding to the mode of origin of the variations in pressure, the individual beats which coincide with the increase in pressure are greater than those which correspond to its decrease.[3]

[1] This sign is, however, ambiguous, because the pelotte of the sphygmograph rests not only upon the artery, but also upon the venæ comites, and, of course, any increased dilatation of the latter will also lift the registering-lever.

[2] The conditions differ in the case of the artificial respiration employed in animal experiments.

[3] For further information see Tigerstedt, *Lehrbuch der Physiologie des Kreislaufes*, 1893, p. 453 *et seq.*

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It should also be noted that the lower the arterial pressure, the more decided are the effects of respiratory influence upon the pulse curve, since thoracic aspiration naturally causes a greater increase of arterial pressure when the arteries are comparatively empty than when they are full. Such imperfect filling of the arteries generally, but not always, coincides with low pressure. Upon the other hand, with a low arterial pressure the arteries may be even abnormally distended—*e. g.*, when the low pressure depends upon dilatation of the arteries with a good volume of circulating blood. Arterial pressure in itself is consequently not responsible for the occurrence of pronounced respiratory variations in the pulse curve, but for the volume of blood contained in the arteries. To this extent the occurrence of marked inspiratory variations in the pulse curve possesses a certain interest in our consideration of circulatory conditions.

The influence of respiration upon the pulse curve is naturally more pronounced if the respiration produces great variations in intrathoracic pressure. As a result

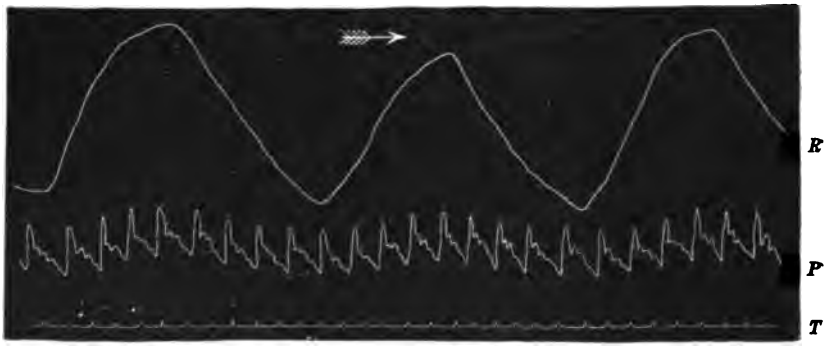

FIG. 40.—The influence of deep and slow breathing upon a normal pulse curve: *R*, Respiration curve, inspiration above; *P*, pulse curve; *T*, time curve (intervals = 0.5 seconds) (Rollet).

of this we observe particularly pronounced variations of the pulse curve in stenoses of the air passages and in all conditions, like pneumonia and pleurisy, in which the equalization of the intrathoracic and extrathoracic air pressures is disturbed.

Respiration exerts a particularly pathologic influence upon the pulse curve when, as the result of intrathoracic adhesions (particularly in the mediastinum), inspiration or expiration exerts an abnormal traction or pressure upon the veins leading to the heart, by which the blood is more or less withheld from the vessels during one of the respiratory phases (pulsus paradoxicus, cicatricial mediastinitis, see p. 126 *et seq.*).

Under normal conditions the influence of respiration upon the pulse curve is slight and scarcely perceptible even in the sphygmogram, so that when marked inspiratory variations are present in the pulse curve we should look for one of the above-mentioned pathologic factors.

Superficial breathing has no especial influence upon the sphygmogram.

OTHER FACTORS WHICH INFLUENCE THE PULSE CURVE.

1. Diminution of the amount of blood. This produces a diminution and a delay in the elasticity elevations and a decided prominence of the recoil elevation. Venesection for therapeutic purposes may sometimes be sufficient to accomplish this effect.

2. An intermitting cardiac activity diminishes the arterial pressure during the intermission of the pulse. Consequently the pulse wave following upon the pause presents signs of diminished pressure—*i. e.*, the elasticity elevations are weaker and delayed and there is dicrotism.

3. Elevating an extremity diminishes, depressing it increases, the arterial

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pressure in the part. Consequently the sphygmogram of an elevated area shows more distinct dicrotism and slighter elasticity elevations than that of a horizontal or dependent area.

4. Compressing the larger vascular trunks produces an increase of pressure in the other pulsating vessels. This is shown in a well-known way by the sphygmogram.

5. An interference with the venous flow from an extremity acts in an opposite way. The arterial pressure in the supplying artery increases, and its sphygmogram is correspondingly altered. The rise of the curve as a whole is, however, partly due to the distention of the congested veins (see p. 118).

We are indebted to Landois and Marey for most of these statements.

DIAGNOSTIC SIGNIFICANCE OF THE PULSE CURVE.

At first observers were naturally inclined to overestimate the value of sphygmographic tracings for the diagnosis of pathologic conditions, but to-day most clinicians have gone to the opposite extreme. As a matter of fact, sphygmography is more than an interesting amusement; it is really a valuable help in the interpretation of circulatory changes. But the factors which influence its shape are so numerous that many different circulatory conditions may be responsible for an identical sphygmogram; so that even in a cardiac case we are not justified in making a diagnosis merely from the type of the curve. Even the curve of aortic insufficiency, one of the most characteristic of all curves, cannot be considered as pathognomonic of this affection; exactly the same type may occur without any valvular mischief. Still, the sphygmogram is valuable in the diagnosis of many conditions, provided that other symptoms are given their proper value, and provided that the clinician possesses considerable technical skill in making the tracings and a very accurate knowledge of the significance of the sphygmographic curve and of the way it may be modified by various factors. Such a knowledge can only be obtained by studying physiologic works.[1]

One of the main objections to making use of the sphygmogram for diagnostic purposes has been that all the variations in the curve attributed upon p. 116 *et seq.* to the arterial tension—*i. e.*, to the general blood-pressure—can be produced by local changes in the vasomotor tone of the artery examined with a uniform blood-pressure. Of course, if this objection were correct, sphygmography would practically be relegated to a useless place. Mosso is chiefly responsible for this objection. He found that the pulse curve could be changed by local thermic application—*e. g.*, the local application of heat to the arm dilates the vessels and produces a pulse curve characteristic of low tension, while local cold produces a curve characteristic of high tension. But a closer examination of Mosso's curves shows that such changes are quantitatively quite insignificant, and apply much more to the height of the curve than to its shape. Variations in the pressure acting upon the vessels

[1] We mention especially Landois, *Die Lehre vom Arterienpuls*, 1872; Marey, *La Circulation du Sang*, 1881; Grashey, *Die Wellenbewegung elastischer Röhren und der Arterienpuls des Menschen*, Leipzig, F. C. W. Vogel, 1881; v. Frey, *Die Untersuchung des Pulses*, Berlin, J. Springer, 1892; Mosso, *Die Diagnostik des Pulses in Bezug auf die localen Veränderungen desselben*, Leipzig, Veit & Co., 1879.

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from without produce but an insignificant effect upon the shape of the pulse curve. This Mosso easily determined in his investigations, which were made with a water sphygmograph. The writer has become convinced that it is extremely difficult to alter very materially the shape of the individual beats by such local thermic influences, either in the sphygmogram or in the tachogram.[1] Besides, where the changes are pronounced, it is not possible to exclude the effect upon the general blood-pressure which can be produced reflexly by the local application of heat or cold. In fact, where the greatest alterations were observed the patients reacted to the thermic changes with sensations of heat and cold. The author has never been able to change a high tension to a dicrotic pulse, or the reverse, by purely local influences of so moderate a degree that a general influence could at the same time be excluded. The general character of the pulse curve remains the same even after a more extensive application of heat or cold to the arm. The difficulty in influencing the character of the curve is well illustrated by making tracings with a different amount of pressure upon the spring. Although the height of the curve as a whole, and that of the secondary curves, will be influenced, neither the general shape of the curve nor the number and position of the secondary elevations will be effected in the least. This remains true even if the artery is almost compressed at the periphery, and such a compression would apparently be quite similar to the effect of a sharp vasomotor contraction there. This fact convinced v. Frey that the best way to employ the sphygmograph was with a very strong pressure of the spring, thus avoiding any excessive swinging of the registering lever. The more frequently and carefully one employs the sphygmograph, the more one is convinced that it registers the condition of the general—*i. e.*, the aortic—circulation, and that it is but slightly influenced by local vasomotor influences. The sphygmogram is, first of all—and herein lies its clinical value—the expression of the form which the pulse wave assumes under the circulatory conditions of pressure and resistance in the aorta and its large branches. Another thing worth remembering is this, that excessive vasomotor conditions, such as Mosso employed (local baths, violent muscular motions, etc.), rarely affect the ordinary radial heat. If they did we should notice other evidences of vasomotor action, such as an increased temperature or redness of the skin. The radial vasomotor tonus of an individual ordinarily clothed and reasonably quiet in all probability varies but little from the average value, which depends upon the condition of the general circulation.

One practical difficulty with the sphygmograph is that in one case, even under physiologic conditions, the pulse curve may present several peculiarities which in another case would signify some pathologic variations. Hence, a sphygmogram is more practically useful in following the circulatory conditions in the same patient—*i. e.*, for what might be termed functional diagnosis of circulatory disturbances—than for ordinary diag-

[1] E. Balli, *Ueber den Einfluss von Erwämung und Abkühlung der Haut auf das Flammentachogramm*, J. A. D., Bern, 1896.

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nostic purposes. It is of decided assistance in studying more closely the therapeutic action of certain measures upon the circulation and of determining the more rational plan of treatment in a given case. Jaquet's sphygmograph is especially suitable for this purpose, not only because an accurate time-measuring apparatus is attached, but also because the amount of pressure upon the spring can be reproduced each time exactly the same. Another advantage is that the examiner can make at each trial five different curves (corresponding to the five degrees of tension in the spring, and so compare them with five others). This will prevent misjudging rather insignificant local variations of vasomotor tension. The increased pressure on the spring evidently has the same effect on the circulation of the hand as a marked increase of tonus of the radial artery. After a little practice one can take five such tracings very rapidly. There is still quite a difference of opinion whether high or low curves present the more correct picture. Since the modern instruments do not permit much swinging, the high curves seem rather more suitable, because they show that the sphygmograph was very perfectly applied, and because their results are perhaps better for comparison in different patients as well as in the same patient. It is evident that the excursions of the registering-needle will be the highest when the tension upon the spring either just balances or is but very slightly in excess of the minimum (cardiac diastolic) pressure in the artery (see p. 107). On the one hand, this is because the transmission of the systolic increase of pressure to the sphygmograph is not interfered with by the relaxed arterial wall; and, on the other hand, because with such an amount of pressure the artery will be closed during the passage of the trough of the pulse wave, so that the next wave will back up considerably, and consequently the rise will be magnified, just as with a hydraulic press. Comparing the various pulse curves with each other, especially under those well-definable conditions, is really very valuable.

FREQUENCY OF THE PULSE IN THE SPHYGMOGRAM.

With Jaquet's and with v. Frey's latest sphygmograph, both of which are furnished with a time-marking apparatus, we can estimate the frequency of the pulse very accurately merely by the sphygmographic tracing.

RHYTHM OF THE PULSE.

A pulse-tracing shows the pulse rhythm at a glance, and much more accurately than it can be described. A *regular* pulse is one in which the individual waves follow each other at exactly equal intervals of time. Where this is not the case the pulse is termed *irregular*. If the irregularity is complete we call it a *pulsus irregularis*, without further comment. A *pulsus intermittens* is one in which, after a regular number of waves, one beat from time to time is omitted. A *pulsus intercidens* is one in which a small beat is inserted into a regular sequence of beats. The so-called *bigeminus* (Fig. 41) and *trigeminus* (Fig. 42) represent periodically irregular pulses. In the former 2 beats, and in the latter 3 beats, are grouped together and separated from the preceding and

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following beats by a somewhat longer interval. Their curves usually do not reach the base line between the groups of 2 or 3 beats, so that the sphygmogram looks like a two- or three-peaked curve.

The clinical significance of the periodically irregular pulse (see p. 104) is that of a slight degree of irregularity. We have no accurate information as to the mode of origin of the various irregular rhythms.

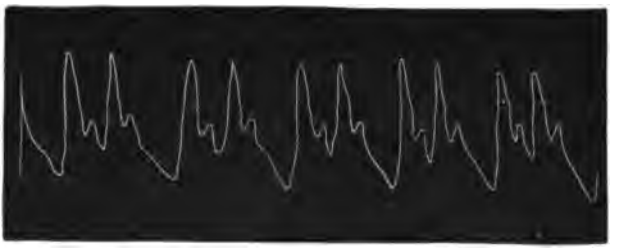

FIG. 41.—Pulsus bigeminus æqualis (Riegel).

[In an exhaustive recent work by Wenckebach[1] a masterful demonstrative explanation is given of many of the most puzzling of heart symptoms. The various types of arrhythmia are considered. It may only be said in this place that he accepts without reservation the myogenic theory of the heart's action as opposed to the neurogenic—*i. e.*, that the ordinary rhythm of the heart is independent of its nervous connections, the muscle fibers possessing all the attributes necessary for the rhythmic production of the heart's work.

FIG. 42.—Pulsus trigeminus æqualis (Riegel).

The editors earnestly recommend a study of the work to all interested in heart studies. A slight criticism may be made that Wenckebach does not give the credit due to English observers in the same field of work (see the Appendix).—ED.]

(Compare "the practical examples" (p. 133) with regard to the possibility of differentiating a "sufficient" from an "insufficient" heart by the irregularity in the sphygmogram.)

VOLUME OF THE PULSE IN THE SPHYGMOGRAM.

According to our definition on p. 105, the volume of the pulse is represented by the height of the primary curve summit above the base of the curve. Other things being equal, it is evident that the volume

[1] *Die Arhythmie als Ausdruck bestimmter Funktionsstörungen des Herzens*, by K. F. Wenckebach, Gröningen, Leipzig, 1903.

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of the pulse depends upon the amount of blood which is thrown into the artery during systole. If this systolic amount of blood is equal, the volume of the pulse then depends upon the facility with which the arterial wall gives way to the wave motion of the blood—*i. e.*, on the one hand, upon the passive tension of the artery as determined by the blood-pressure, and, on the other hand, upon the active tension of the muscularis of the artery. The volume of the pulse—*i. e.*, the height of the sphygmographic curve—also depends very decidedly upon the size of the surface of the artery (according to Pascal's law of the hydraulic press). Provided the systole of the left heart is equal and the blood-pressure is equal, the larger the diameter of the radial artery the higher the spring of the sphygmograph will be raised, and hence the larger the pulse. The frequency of the pulse also influences the height of the sphygmogram. If the pulse is rapid a part of the descending limb of the curve will be cut off by the following wave. So many factors influence the volume of the pulse in ways which we cannot perfectly determine from the appearance of the curve itself, that the mere volume of the pulse is of very uncertain diagnostic significance. In the following cases, provided the tracings compared are all of a maximum height, the volume of the pulse is of some significance:

1. When the size of the individual beat varies in any one pulse curve we may be sure that the larger beat corresponds to a larger expulsion—*i. e.*, is preceded by a more complete diastole of the heart—and that the smaller beat corresponds to a smaller expulsion.

2. If the same patient's pulse becomes fuller and of higher tension when examined with the same sphygmograph (secondary rises nearer the chief elevation, p. 116, 6, 128 *et seq.*), then we are justified in assuming that a greater systolic expulsion is the cause.

FIG. 43.—Pulsus alternans (Eichhorst).

On the other hand, if the full pulse is at the same time of diminished tension, then its increased size may depend upon the flaccidity of the arterial tube—*i. e.*, upon diminished blood-pressure.

The following terms refer to the size of the pulse: *Pulsus equalis* and *inequalis* (the latter is usually a pulsus irregularis as well); *pulsus inequalis periodicus.* The most interesting types of the latter are the *pulsus alternans* (Fig. 43), *pulsus bigeminus alternans* (Fig. 44), and *pulsus paradoxus* (Fig. 45).

Inequality of the pulse is of similar clinical importance as irregularity (see p. 104), and is usually found combined with it. Periodic

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inequality means the same thing as a slight degree of inequality. The *pulsus paradoxus*, a poorly chosen name, was first described by Griesinger,[1] and later by Kussmaul, as a constant symptom of indurative mediastinitis. Its peculiarity is that during inspiration the pulse becomes feeble or else cannot be felt at all. Kussmaul explains it as due to an inspiratory pull upon the veins leading to the heart by the intrathoracic adhesions. From the explanation given at p. 118 *et seq.* it follows that this symptom can have no pathognomonic importance in the diagnosis of indurative mediastinitis. From the physiologic significance of

FIG. 44.—Pulsus bigeminus alternans (Eichhorst).

the phenomenon as there explained, it is not surprising that it has been observed also in pericarditis, in valvular disease, in weak heart, in pneumonia, in pleurisy, and in stenoses of the air passages (laryngeal and tracheal stenoses, obliterative bronchitis). Even when the heart and lungs are absolutely normal, it is possible that respiration may produce some evident effect upon the pulse, although this is not usually the case, as we have previously pointed out on p. 120.

It seems to the writer that the pulsus paradoxus is of value in the diagnosis of indurative pericarditis only when there is a concomitant inspiratory engorgement of the jugular veins, a symptom which indicates a stenosis of the jugular veins during inspiration.

FIG. 45.—Pulsus paradoxus: *E*, Beginning of expiration; *J*, beginning of inspiration (Kussmaul).

The typical pulsus paradoxus (Fig. 45) is copied from Kussmaul's original communication. It especially seems to the writer to possess no diagnostic importance whatsoever. As is easily seen from the figure, the pulsus paradoxus is just twice as frequent as the respirations, so that expiration always coincides with one beat and inspiration with the other. If the breathing is energetic this relation will naturally emphasize the physiologic factors described on p. 118. As a matter of fact the breathing is rapid, so that under the circumstances, according to p. 119, even if the circulation is normal, the pulse shows more or less distinctly an inspiratory decrease or even a disappearance. Besides, from what

[1] Widenmann, *Beitrag zur Diagnose der Mediastinitis*, Tübingen, 1856.

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has been mentioned on p. 119, it is evident that under certain circumstances, and practically independent of the frequency of the respiration, a noticeable diminution or even an omission of the pulse may be observed even during expiration. Such a phenomenon could just as well be called a "pulsus paradoxus," and it certainly has just as little significance in the diagnosis of any definite disease.

Although all these phenomena of an alteration in the pulse from respiratory influences coincide with the occurrence of a low arterial pressure (p. 120), we are not justified in diagnosing any one definite disease, such as pericarditis or mediastinitis, but merely a deficient general peripheral circulation.

CELERITY OF THE PULSE IN THE SPHYGMOGRAM.

The celerity of a pulse is a quality which we can appreciate by palpation (p. 105). It is also plainly represented in the sphygmographic curve. Both ascending and descending limbs of a *pulsus celer* are steep and the summit is sharp. On the contrary, both limbs of a true *pulsus tardus* are sloping; the curve is flat and the summit blunt (Fig. 47).

FIG. 46.—Pulsus celer in aortic insufficiency (Riegel).

Either limb of the curve may exhibit the signs of celerity or tardiness, while the other limb either is not affected or may exhibit the opposite condition. In this case our description must include the character of each limb. For the method of determining by construction the shape of the general curve, or of the main summit in the case of polycrotic

FIG. 47.—Pulsus tardus in aortic stenosis (Strümpell).

curves, see Fig. 39. The ascending limb of aortic insufficiency (Fig. 46) is steep because a large mass of blood is suddenly expelled from the dilated left ventricle into the aorta; and the descending limb is also steep because, on account of regurgitation of the blood into the left ventricle, the negative stage of the wave is introduced abnormally

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rapidly. Conversely, the ascending and descending limbs of aortic stenosis are oblique; it is a typical *pulsus tardus* (Fig. 47). Here the rise as well as the drop of the curve takes place slowly because the systolic impulse is deadened and prolonged at the seat of stenosis. In relaxed vessels—*i. e.*, in fever—the rise as well as the drop is usually quick (see p. 116). The dicrotic febrile pulse is therefore usually a *pulsus celer* (Fig. 51). With high arterial tension the descending limb of the curve is usually quite gradual and oblique (p. 116). The rise is generally rapid and steep on account of the energetic cardiac action.

FIG. 48.—Senile pulse in arteriosclerosis (Riegel).

This is the type of pulse which is usually observed in old people with arteriosclerosis (Fig. 48). Nevertheless a very decided degree of arterio-

FIG. 49.—Pulse in advanced arteriosclerosis: Slow ascent and descent of the wave; anacrotism—only 32 beats to the minute (taken with Jaquet's instrument).

sclerosis (Fig. 49) is generally accompanied by a gradual rise. In nephritis (Fig. 50) the rise is steep and the descent slow, but the pulse

FIG. 50.—Pulse in chronic nephritis.

differs from that of arteriosclerosis by the great number of secondary elevations.

Although the general shape of the sphygmogram gives some idea of the arterial pressure and the way the blood flows into the arteries, and from them to the periphery, yet neither the descent nor the ascent of the curve is absolutely diagnostic. In the first place, the height of the curve influences the slant of the descending limb; and, as a rule, we are unable to interpret the significance of variations in the height (p. 125). Again, the frequency of the pulse decidedly modifies the slant

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of the descending limb, because if a pulse is rapid a portion of its descending limb is simply cut off by the following wave, so that the height of the curve seems lessened. Even in aortic insufficiency the value of the slant of the descending limb is rather more for demonstration than for diagnosis. The slant of the ascending limb is almost as variable. The steepness of its ascent does not, in itself alone, give us accurate information concerning the quickness of the rise of the pulse wave. The only really accurate way of determining the celerity of the rise of the ascending limb, and this is of decided importance in pronounced arteriosclerosis or in aortic stenosis, is to measure the length of the time-registering line between the base of the rise and its summit. We must, of course, remember that this time interval should not be confounded with the duration of the cardiac systole, for systole (p. 115) extends beyond the main elevation. It should be observed that as a result of the fact that the duration of the ascent of the pulse curve does not correspond with the duration of the delivery of the blood into the aorta, the demonstration of the characteristic pulse of aortic stenosis is frequently unsuccessful, since the duration of the ascending limb of the curve is shortened by the rapid flowing of the blood toward the periphery. The pulse simply seems small, but does not present the typical characteristics of the pulsus tardus. To the best of the writer's knowledge, attention has not been previously directed to this evident explanation of the frequent absence of the pulsus tardus in aortic stenosis.

TENSION OF THE PULSE IN THE SPHYGMOGRAM (POLYCROTISM; DICROTISM; ANACROTISM).

The secondary elevations of the descending limb (p. 116 *et seq.*) are most significant in attempting to estimate from the pulse-tracing the tension of the pulse—*i. e.*, the mean blood-pressure. Elasticity elevations which are pronounced or which arise early in the curve [according to v. Frey's and Krehl's conception (p. 118) the early reflex waxes—*i. e.*, secondary elevations near the summit] or anacrotic elevations (p. 117) generally indicate a *high* mean blood-pressure. The converse—*i. e.*, the so-called dicrotic wave (p. 116)—is usually more pronounced with a *low* mean blood-pressure (p. 116). Where none of the secondary elevations, either from their size or their position, can be considered as a dicrotic wave, we are justified in assuming that numerous and pronounced secondary elevations are more in favor of a high pressure; because, according to Landois, these numerous elevations should be regarded as elasticity elevations, and according to v. Frey's and Krehl's idea, powerful and, at least in part, early reflex waves.

Compare 116 *et seq.* and 122 in regard to the general form and size of the pulse under variable mean pressure.

The type of the pulse curve with varying arterial pressure may be represented as follows:

1. *Normal Pressure.*—Both the elasticity elevations and the dicrotic wave, moderately developed; the latter, however, only slightly different from the elasticity elevations (see Fig. 38).

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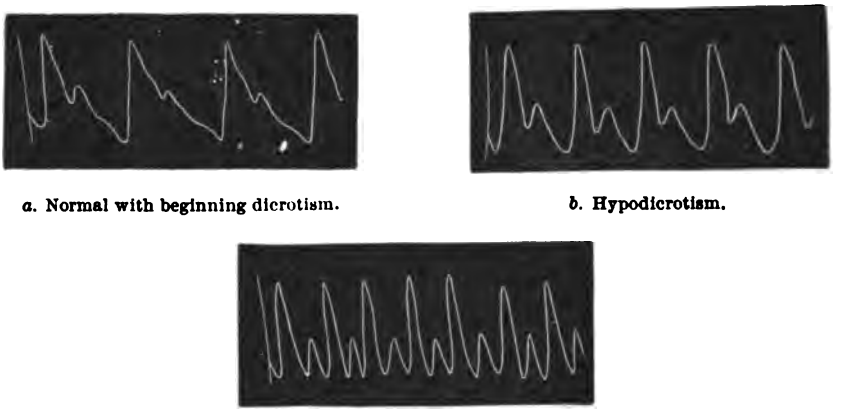
2. *Low Pressure.*—Elasticity elevations disappear, dicrotism increases, and eventually becomes transformed into monocrotism; the height of the pulse is increased—*i. e.*, it becomes a pulsus celer, except when the low pressure is due to a weak, slight systole (Fig. 51, *b*, *c*, *d*, *e*).

3. *High Pressure.*—Elasticity elevations increase in size and number; they are situated nearer the summit (Figs. 45 and 52) or even on the ascending limb of the curve (anacrotic); the descending limb, and in rare cases the ascending limb, are oblique (tardus); the height of the curve is generally low except when the high pressure is due not to increased resistance alone, but to a strong, full systole as well. If the



a. Normal with beginning dicrotism. *b*. Hypodicrotism.

c. Dicrotism.

d. Hyperdicrotism. *e*. Monocrotism.

FIG. 51.—Increasing dicrotism finally transformed into monocrotism (Riegel).

blood-pressure is very high, and especially if it is due to arteriosclerotic resistance, not only the dicrotic rise disappears, but even the elasticity elevations fade away more or less completely, because the arterial wall has become stiffened, either on account of the firm contractions of its muscularis or on account of the anteriosclerotic changes. A monocrotic pulse finally results. Fig. 49 represents such a slow, low, and almost monocrotic sphygmogram; but there still remain some indications of elasticity elevations visible especially in the ascending limb (anacrotism).

In this schematic grouping the relation of the size and the celerity of the pulse curve to the blood-pressure has only a theoretic significance,

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because the qualities of size and celerity in the sphygmogram (pp. 124 and 127) possess only slight diagnostic value.

Furthermore, merely counting the elasticity elevations is not always sufficient to determine the height of the blood-pressure, because the pulse frequency always plays a part. With a frequent pulse the curve is not completely developed; so that a part of the descending limb and the elasticity elevations contained in it will not show.

The development of a dicrotic wave is best studied in a febrile pulse, because dicrotism usually goes hand in hand with a diminution of the vascular tension. The higher the fever, the more fully the dicrotic wave is developed, and the further distant is it from the main summit of the curve (Fig. 51, *a*, *b*, *c*, *d*). The individual steps of this change have received separate names. If the dicrotic elevation begins before the descending limb reaches the base of the curve, the pulse is called *hypodicrotic* (Fig. 51, *b*). It is strictly *dicrotic* when the dicrotic wave starts only after the descending limb reaches the base line. If the dicrotic wave occurs still later —*i. e.*, in the ascending limb of the following wave—the pulse is then termed "hyperdicrotic" (Fig. 51, *d*). This peculiarity may arise either because the dicrotic wave is retarded or because the following wave, coming so rapidly, cuts off the descending limb of the dicrotic wave. If in Fig. 51, *c*, the ordinary dicrotic pulse, we imagine that the individual beats follow more closely one after the other, then "hyperdicrotism" results (Fig. 51, *d*). If the dicrotic wave is still further retarded or, what amounts to the same thing, if the rate of the pulse is still further increased, a monocrotic wave results (Fig. 51, *e*). This monocrotism with relaxed vessels and low pressure differs from the monocrotism of high pressure (Fig. 49). In the latter the tense or rigid arterial wall cannot produce any considerable secondary elevations. The pulse wave is here usually low and the pulse very slow. Typical examples of high-tension pulse curves are the arteriosclerotic pulse (Fig. 49), the nephritic pulse (Fig. 45), and the tense pulse in lead colic (Fig. 52).

Anacrotic elevations (p. 118) probably occur only with high blood-pressure.

FIG. 52.—Tense pulse in lead colic: Some of the waves anacrotic (Riegel).

Fig. 52 shows anacrotism in some of the summits, and it is also suggested in the curve of Fig. 49. Rounded summits (Fig. 48) probably signify anacrotism (consisting of several anacrotic elevations). The rigidity of the artery prevents it from being distinctly indicated. Similarly, rounded tops which slant off toward the descending limb should probably be imagined as confluent catacrotic elasticity elevations. Sometimes the rounded top is flattened like a plateau. But the significance is probably not changed.

SPECIFIC SPHYGMOGRAMS.

When the sphygmograph was first employed, it was believed that pathognomonic pulse curves would be found to characterize certain affections, especially cardiac cases. But such a belief has not been justified. Not even the curve of aortic insufficiency can be considered specific of this disease, for in fever and in exophthalmic goiter, without any leak at the aortic valve, a very pronounced pulsus celer is often observed. The pulse curve of mitral lesions is less suggestive or certain, although in some instances it is of decided assistance in the diagnosis. A serious

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error was formerly made in the attempt to find characteristic signs in the curve during the time of disturbed compensation. This is evidently the least favorable time, because if compensation is affected to any extent the pulse will always be a small pulse and of low tension; and such a condition might be due to the valve lesion alone, without any disturbance of compensation. v. Noorden[1] has particularly emphasized the necessity of utilizing well-compensated cases in order to obtain characteristic curves, and believes that compensated mitral stenosis exhibits

FIG. 53.—Tense pulse in compensated mitral stenosis (v. Noorden).

a high-tension pulse, whereas compensated mitral insufficiency exhibits a low-tension pulse (Figs. 53 and 54).

v. Noorden accounts for this phenomenon by assuming that in mitral stenosis an increased arterial tone aids in maintaining the compensation. The arterial

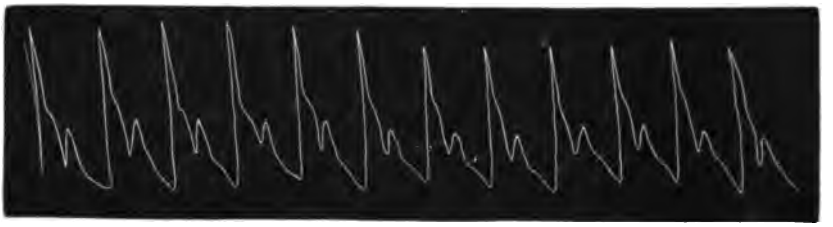

FIG. 54.—Lack of tension of the pulse in compensated mitral insufficiency (v. Noorden).

system is thus sufficiently filled and the pressure preserved despite the small systole. On the contrary, in mitral insufficiency the compensation is favored by vasomotor relaxation of the vessels, which diminishes the resistance in the arterial system. In this way a much larger proportion of the left ventricular contents can be utilized by the circulation, and a correspondingly smaller portion returns into the left auricle. These peculiarities are, the writer believes, more simply explained as follows: In mitral stenosis there is no reason for any diminution of the arterial pressure; whereas in pronounced mitral insufficiency a high arterial pressure cannot possibly occur because of the regurgitation of the blood into the left auricle. If the circulation should be approximately normal in such a marked degree of mitral insufficiency, in spite of the low arterial pressure, it is an indication that the vasomotor tone of the general circulation is lowered, a fact which has a compensatory significance, and to this extent v. Noorden is correct.

A FEW PRACTICAL EXAMPLES TO ILLUSTRATE THE APPLICATION OF THE SPHYGMOGRAPH.

The deductions which may be made from the sphygmogram as to the condition of the circulation are well illustrated in Fig. 55, *a* and *b*, and Fig. 56 *a*, and *b*. They represent tracings taken (*a*) from a case of disturbed compensation and (*b*) from the same case after compensation has been re-established by the employment of digitalis.

The comparison of curves in Fig. 55, *a*, and Fig. 57 is very interesting from a

[1] *Charité-Annalen*, 15 Jahrgang.

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diagnostic standpoint. Both curves show an irregular pulse, but with these differences: The curve in Fig. 57 in general, and the small interposed beats, represent a pulse of high tension, whereas the pointed single elevations of the curve in Fig. 55, *a*, point in general to a low degree of tension. The arterial pressure sinks quickly, especially in the little beats; this is evident from the dicrotism and from the depression of the

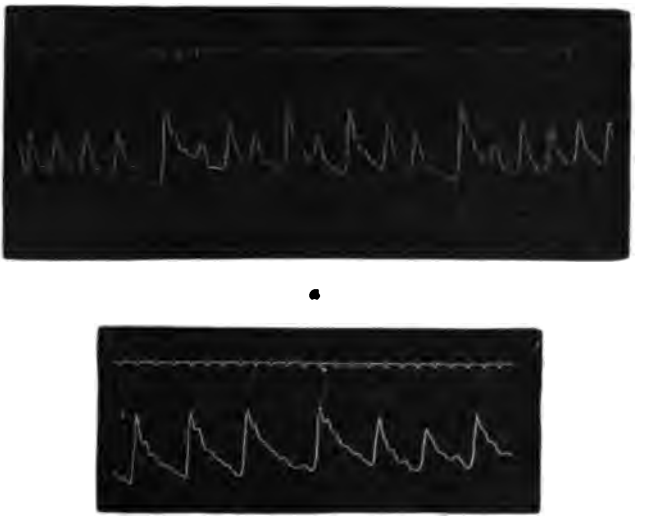

FIG. 55.—Sphygmogram from a patient with mitral insufficiency, to demonstrate the action of digitalis: *a*, Before the administration of digitalis—circulation decidedly affected, radial pulse 128, cardiac beats 172; *b*, after the employment of digitalis—circulation practically normal.

base of the curve. The latter fact alone points to an insufficient systole as compared with the curve in Fig. 57, where the cardiac apparatus is evidently sufficient.

The type of irregularity illustrates another difference between the two curves. In Fig. 55, *a*, the size of the individual pulse wave certainly in some places seems to be independent of the size of the preceding interval, whereas in Fig. 57 the size

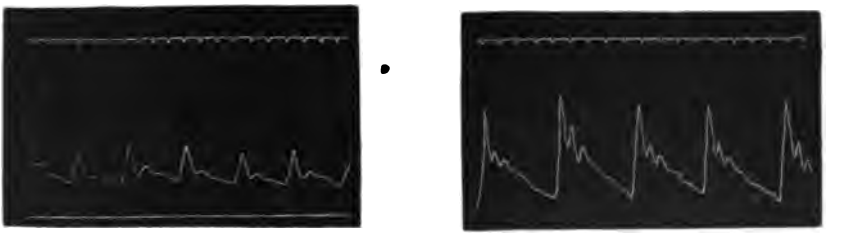

FIG. 56.—Sphygmogram from a patient with emphysema and cardiac dilatation, to demonstrate the action of digitalis: *a*, Before the administration of digitalis—circulation decidedly affected, pulse about 100; *b*, after 2 gm. of digitalis—circulation normal, pulse 70.

of the individual beat is directly proportional to the duration of the preceding interval. The first type of irregularity, according to my experience, always points to cardiac insufficiency of a kind that can be helped by employing digitalis. This drug will improve the flow of blood through the cardiac muscle itself, and so help the disturbed innervation which is the cause of the arrhythmic condition.

The irregularity represented in Fig. 57 is quite different. Here the difference

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in volume of the beats is a direct sequence of the irregularity. Because the left ventricular diastolic filling is brief, the succeeding pulse must be smaller, even if the cardiac power is entirely sufficient. This type of irregularity, therefore, does

FIG. 57.—Arrhythmic sphygmogram in cardiac sufficiency.

not point to cardiac insufficiency, and so in itself presents no indication for the use of digitalis. The size and the high tension of the individual pulse wave evident in Fig. 57 would be another reason for the uselessness of digitalis in this case.

SPHYGMOMANOMETRY (TONOMETRY).

The sphygmograph furnishes only relative information of the height of the arterial pressure; hence v. Basch[1] attempted to construct an instrument to measure the arterial pressure upon the intact human body. He had succeeded, approximately, with the sphygmomanometer. Potain has since modified v. Basch's more modern instrument.

v. Basch attempted to copy the method of estimating pulse "tension" with the finger (p. 106) by measuring instrumentally the amount of pressure required to suppress the pulse wave. Waldenburg and Talma had attempted to obliterate the pulse in the artery by compressing the artery by weights or springs. The size of the surface pressed upon was neglected in these experiments, so that their results are of no particular value. To obviate this difficulty, v. Basch employed a so-called air pelotte—*i. e.*, a thin rubber membrane tied like a bladder over a metal cylinder, filled with air and connected with a manometer. With this mechanism the size of the surface pressed upon makes no difference; the manometer reading will always be the same. For, according to hydrostatic laws, the amount of pressure indicated by the manometer is that exerted upon each point of the surface of the pelotte.

v. Basch has recently made a practical improvement in the shape of the pelotte. Fig. 58 represents the instrument in its new form: *A*, the manometer;

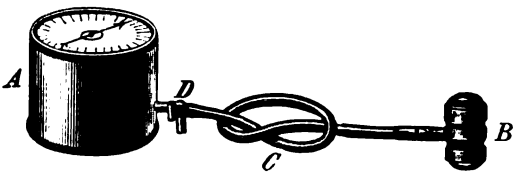

FIG. 58.—v. Basch's sphygmomanometer.

B, the pelotte; *C*, the connecting tube. By means of the cock, *D*, the entire system is filled with air under a low pressure,[2] so that the pelotte is but slightly tense and the two closing membranes bulge a trifle.

[1] *Berlin. klin. Woch.*, 1887; *Arch. de physiologie*, 1889, Ser. 5, vol. i., p. 556, and vol. ii., p. 300.

[2] v. Basch formerly filled the cylinder with water. Air was first employed by Potain, and has the advantage of doing away with hydrostatic pressure, and so enabling us to disregard the level of the manometer in reference to the pelotte.

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The employment of this instrument is very simple. The course of the artery to be examined is first marked upon the skin; one of the bulging membranes of the pelotte is placed upon the artery, and the manometer is laid upon the bed at the side of the patient. The examiner then attempts to estimate the amount of pressure (read upon the manometer scale) necessary to obliterate the pulse peripheral to the pelotte. This he does by palpating with one finger, while with another he prevents any anastomotic pulse from entering the artery. This method will determine the arterial pressure at least approximately; perhaps more accurately if the manometer is read at the moment when the pulse reappears after the pressure has been gradually diminished. The method is, of course, entirely subjective, as its accuracy must depend upon the examiner's sense of touch. To obviate this difficulty, v. Basch has attempted to substitute the sense of sight. A rubber band is rather loosely placed over the radial artery and a small pin stuck in it, so that the excursions of the pin are plainly visible. If the pin stops moving the pulse is considered absent. According to the writer's experience, this device is not always successful.

Two factors combine to prevent this method from accurately measuring the true arterial pressure. In the first place, the wall of even an empty artery remains open, so that a certain, though small, amount of the pressure exerted represents that employed to compress the arterial tube; and, in the second place, the tissues covering the artery must interfere to a slight extent with the accurate application of the pelotte, so that the instrument must register a somewhat higher pressure than actually exists.

v. Basch's experiments, however, show that both these factors combined cannot cause a deviation of the real values from those recorded of more than about 10 to 15 mm. Tigerstedt[1] gave a very unfavorable opinion of the value of this method, because he found a much greater deviation. In the previous editions of this book the author shared this view, but feels obliged to withdraw his criticism since he has used the improved air pelotte as illustrated in Fig. 58. As a matter of fact, the results given by the old pelotte were not very reliable, for reasons which cannot be here given in detail. The temporal artery is the most conveniently situated for this measurement; the radial next, provided that it is compressible against the lower end of the radius. In the former v. Basch found that the pressure varied from 90 to 120 mm. Hg.; in the latter, from 110 to 160. The author's own estimations at the radial have usually been from 160 mm. upward. Potain's investigations (*loc. cit.*) show that these figures correspond to the maximum pressure variations during a pulse wave—*i. e.*, to the systolic pressure.

Another reason to doubt the accuracy of this method is the following: Since dynamic processes—that is, moving masses—are chiefly concerned, we must consider the vital power of the pulse wave according to the laws of the hydraulic press. Now, it is well known that when a current is compressed by some obstruction, a far greater force is developed above the point of resistance at the moment of compression than the corresponding amount of lateral pressure of an unresisted flowing fluid. This arises from the transformation of the pressure of velocity into lateral pressure. The hydraulic press or ram depends upon this principle. Consequently a large pulse wave, even with low arterial pressure, will force its way through under the compressing pelotte, owing to the greater amount of "energy," while a smaller pulse wave, even with higher arterial tension, will be completely obstructed, because the latter possesses less energy. Again, the portion of the wave which passes through under the pelotte will be felt longer if the pulse is large than if it is small and hard to feel, even though of high tension. This objection agrees with Potain's results (*loc. cit.*), according to which the values found with the v. Basch instrument correspond to the maximum (systolic) pressure, but do not furnish any information of the mean or of the minimum pressure. This only means that the manometric values depend upon the potential energy of the individual pulse wave. With this proviso the method may be of some clinical use, but we must not imagine that the values thus determined correspond with the read-

[1] *Lehrbuch der Physiologie des Kreislaufes*, Leipzig, 1893.

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ings of a mercury manometer connected directly with an artery. It is commonly supposed that these systolic values, to be sure, are not so very different from the mean arterial pressure. But although the well-known kymographion curves with the mercury manometer seem to show that the systolic pressure varies but little from the mean pressure, at the same time these curves are not at all applicable for estimating the extent of pressure variations, because the inertia of the mercury column prevents its registering the variations correctly. As a matter of fact, we know very little about normal variations of blood-pressure in man and animals, and still less about the size they may attain in pathologic conditions. In fever, for example, on account of the large, bounding pulse waves, v. Basch's sphygmomanometer may indicate high figures, although the sphygmographic curve shows relaxation of the vessels and a low (mean) arterial pressure. Such peculiar contradictions, which have thus far been attributed entirely to the fault in the sphygmogram and never to the really much less reliable sphygmomanometer, can only be explained by assuming that under these conditions there exists a very much greater difference between minimum and maximum or between mean and maximum pressure in the arteries than is generally accepted. The writer believes that the mean arterial pressure in fever is probably low, corresponding to the shape of the sphygmogram, in spite of the fact that v. Basch's instrument indicates high values, on account of the large and powerful systole in consequence of free flow of blood through the relaxed vessels. Should these views be confirmed, the clinical value of sphygmography will be increased at the expense of sphygmomanometry. At all events, the writer recommends the greatest caution in the clinical use of v. Basch's instrument. (See p. 106 for the real significance of the term arterial tension or blood-pressure.)

FIG. 59.—Sahli's pocket mercury manometer.

The author has recently made what he believes to be an improvement in v. Basch's instrument by increasing the diameter of the air pelotte from scarcely 2 cm. to 3–4 cm. This greatly aids us in securing pneumatic pressure upon the artery; while with the small pelotte of v. Basch there is danger of compressing the vessel by the resistant portion of the caoutchouc near the rim of the pelotte or by the rim itself.

As it has been my experience that the metal manometer of the shops is not very accurately graduated, and that, although it may be correct originally, the values of the scale gradually change, I have constructed a pocket mercury manometer, which may be easily transported, is absolutely correct, and has the additional advantage of being cheap.[1] This manometer may also be employed for any other manometric method, in the Riva-Rocci sphygmomanometer, for example (see p. 137, *et seq.*), for the measurement of the pressure of pleural exudates, and, in virtue of its fine caliber, for the measurement of the pressure of the cerebrospinal fluid in lumbar puncture. (See section upon Exploratory Puncture.) The instrument is illustrated in Fig. 59, and consists simply of a U-shaped manometer in which one branch can be elongated by inserting a glass tube, the connection being accurately ground. The caliber is sufficiently large to exclude the disturbing element of capillarity, and yet not large enough to require a great quantity of mercury. Owing to the accurate adaptation of the two tubes, it is unnecessary to use the greasy materials usually employed to secure the perfect adjustment of glass

[1] The instrument is manufactured by the optician Büchi, in Bern.

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stop-cocks, and the mercury is consequently kept in much better condition. When the instrument is to be employed, the elongation *a b* is inserted, and the pelotte of v. Basch's instrument (preferably the enlarged form, as described upon page 134; see Fig. 58) is connected with *c* by a tube. The pressure exercised upon the artery by the pelotte is transmitted to the shorter branch of the manometer. The elongated branch is so divided that it gives the pressure directly in centimeters, every half-centimeter being marked as a unit. In such a manometer, as is well known, the pressure is obtained by multiplying the height of the ascending column by two, because the mercury falls exactly as far in one branch as it rises in the other. The manometer must be accurately filled with mercury to the zero mark. The ampulla *e* is for the purpose of preventing the throwing of the mercury out of the shorter branch by a sudden diminution of pressure, and the ampulla *b* acts in a similar manner when the pressure is suddenly increased by careless manipulation of the pelotte. The instrument may be packed and transported by removing the elongating tube *a b*, and plugging the openings *a* and *c* with rubber corks. I formerly employed glass stop-cocks for this purpose, but owing to their fragility and the necessity of using a lubricant to render them air-tight, I have found that they were not well adapted to the purpose. The entire instrument may be packed in a handy, well-cushioned case, which is made large enough to also hold the pelotte for von Basch's instrument.

Since the mean blood-pressure is so important for diagnosis, as well as for determining the efficiency of certain therapeutic agents, it is very fortunate that so many physiologists have recently turned their attention toward devising some simple and accurate method of measuring it. Such methods have been described by Mosso, Hurthle, v. Frey, Riva-Rocci, and Gärtner.

The Riva-Rocci and the Gärtner instruments are the only ones which have proved to be practically valuable. The detailed descriptions of the others in previous editions of this book have consequently been omitted in the present one.

The principle of Riva-Rocci's sphygmomanometer[1] is the same as that of v. Basch's. It measures the amount of pressure necessary to obliterate the pulse peripheral to a point of constriction. A pneumatic pressure is used as in v. Basch's instrument. The practically important modification consists in substituting for the single pelotte to press upon the artery a pneumatic cuff which encircles the upper arm, and the cavity of which connects with a rubber bulb. Since the upper arm contains only a single bone, inflation of the cuff will interrupt all the arterial supply to the forearm in a perfectly equal way.

The cuff should be made of some firm mackintosh cloth, or, if of elastic rubber, it should be securely fastened to a lining of material which will not stretch. This is to prevent the loss of a part of the pressure from any eccentric inflation or distention of the wall. Fig. 60 represents the apparatus.

The patient's upper arm is inserted into the cuff (*a*). The latter is connected with a mercury manometer (*b*), and through it with a double rubber bulb (*c*). By pumping at *c* the cuff can be made to encircle the upper arm tightly, and the amount of pressure employed can be read in mm. of mercury upon the vertical tube of the manometer. This pressure is increased until the radial pulse disappears. The cuff, about 4 cm. wide, should be so adjusted as to fit the arm rather snugly without compressing it. The latter purpose is accomplished by a clamp, as is represented in Fig. 60. The instrument was tested experimentally both by Riva-Rocci and by Gumprecht.[2] The muscles should be well relaxed, because compression through thick muscles does not affect the result. The same objection applies to the principle involved in this method as was mentioned in discussing v. Basch's instrument (p. 135). The amount of pressure found depends not only upon the median arterial pressure, but also very decidedly upon the energy of the pulse wave, and with it upon the size of the pulse volume. For these reasons, and perhaps also partly on account of the resistance to compression which the muscles of the upper arm afford,

[1] Riva-Rocci, Un nuovo sfigmomanometro, Torino, 1896; Frascati e Comp. Tecnica della sfigmomanometria, *Gaz. Med., di Torino*, 1897, Nos. 9 and 10.

[2] *Zeits. f. klin. Med.*, 1900, vol. xxxix., parts 5 and 6.

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the figures which the writer has found in a great many attempts have always been very high. Normal individuals furnish a value of 150 to 160 mm. Hg., and the arteriosclerotic and nephritic as high as 230 to 250 mm., or even higher. Although all these values may be relatively too high, nevertheless the instrument possesses a distinct advantage in being purely objective as compared with v. Basch's (see p. 135). This may be realized by making several tests, one after the other, when it will be seen that they are almost invariably the same. This is especially apt to be the case if at each attempt the pressure is raised beyond the point at which the pulse cannot be felt, and then, when, as a result of the yielding of the rubber tubes and connections, the pressure falls slowly, the point is noted at which the pulse begins to appear again. In this way the attention is not distracted by the need of pumping, and the method is still more objective and quite exact. Of course, for a control it is advisable to note the point at which increasing pressure causes the pulse to vanish. The writer has frequently attempted to supplant the palpation of the pulse by the objective method of sphygmography, making short curves at different pressures until the needle makes but a straight line. It has advantages in demonstrating to a clinic. One caution should be added. Pronounced congestion, pain, and even cutaneous

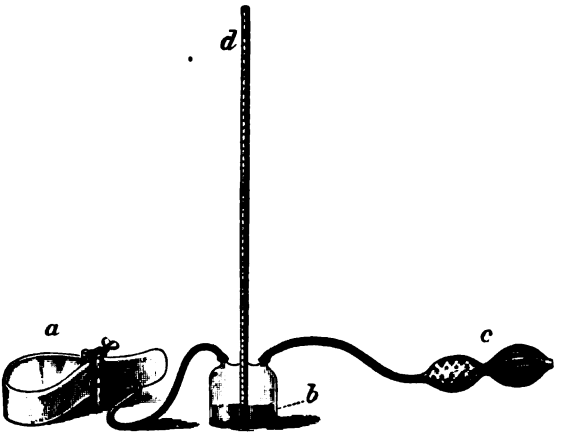

FIG. 60.—Riva-Rocci's sphygmomanometer.

hemorrhage may result from employing the instrument to measure very high pressure—*e. g.*, above 230 mm.

The transportable mercury manometer of Sahli, described upon p. 136, may also be employed in connection with the Riva-Rocci pneumatic arm-band, rendering this excellent instrument much handier and more practical.

Led by v. Recklinghausen,[1] a number of authors have pointed out that the cuff of Riva-Rocci's instrument is too narrow. They came to this conclusion from the fact that different pressure values are obtained when a wider cuff is employed. It is clear that if the cuff is too narrow the inflation produces a marked distortion, both of the cuff itself and of the surface of the arm, which endangers the transmission of the aërostatic pressure, since a certain fractional part of the manometric pressure is consumed in producing the aërostatic tension of the cuff and the tension of the tissues pressed upon. The cuff of the original Riva-Rocci instrument was 4½ cm. in width. These authors now demand a cuff 32 cm. wide. It is clear, however, that this not only makes the instrument more clumsy, but also renders the closure of the cuff more difficult and less secure. Additional errors also arise from the fact that such a broad cuff does not act upon an approximately cylindric surface, owing to

[1] v. Recklinghausen, *Arch. f. exper. Pathol. u. Pharm.*, 1901, vol. xlvi., parts 1 and 2; Hensen, *Deut. Arch. f. klin. Med.*, 1900, vol. lxvii.

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the irregular contour of the arm. Martin[1] has found that a cuff 10 cm. in width suffices for all cases. In the writer's opinion a great deal depends upon the manner in which the cuff is applied. If the cuff is emptied and applied accurately to the skin without the exercise of any pressure, cuffs only 5 to 6 cm. in width remain so flat during inflation that the transmission of the pressure is complete and the results are as accurate as they can well be from the nature of the procedure. We should banish the illusion that this procedure is of equal value with manometry upon the exposed and divided artery.

Gärtner's tonometer[2] depends upon a similar principle. It estimates the pressure which is required to interrupt the peripheral circulation. Gärtner makes use of the color of the tip of the finger instead of feeling the pulse to determine the condition of the peripheral circulation. His instrument consists of a small pneumatic compression ring, whose cavity is connected with a mercury or spring manometer and with a rubber bulb. The pneumatic ring is constructed of a metal hoop 1 cm. high and 2½ cm. in diameter. A rubber membrane lines this hoop, and with the latter surrounds an air space communicating with the manometer and bulb. The pneumatic ring is first slipped over the second phalanx of the little finger without pressure. The projecting end phalanx is then made bloodless by a rubber compressor like a glove finger, or by a ring cut out of a rubber tubing, rolled upon the finger as far as the pneumatic ring. This compression is kept up until the pressure in the pneumatic ring has been raised by means of the bulb sufficiently to keep the blood from returning to the finger; then the compressor is removed. The finger-tip now appears as pale as a corpse. By gradually releasing the pressure from the rubber bulb the finger becomes colored again. As a matter of fact, the finger assumes a cyanotic color, and so facilitates the differentiation. This is because the veins will still be compressed at the moment the digital arteries open under the arterial blood-pressure. This pressure at which the blood again streams into the finger can be regarded as equivalent to the blood-pressure in the digital arteries. In cases where the capillaries of the finger-tip are much contracted, it may happen that the finger-tip will not be properly colored after the release of the pressure on the arteries. In such cases Gärtner accomplished his purpose by relaxing the tonus of the fine vessels by producing an artificial congestion by a pressure of 20 to 40 mm., Hg., with the pneumatic ring for a half minute, and so paralyzing the vessels just before repeating the test. Gärtner assumes that the pressure in the small digital arteries differs but little from that in the radial. Of course, the measurement will be decidedly influenced by the height of the fingers, so that he recommends that the test be undertaken with the fingers at the height of the heart. Without question, Gärtner's instrument offers certain advantages over Riva-Rocci's. One of the most important consists in the avoidance of the unfortunate confusion between the static and dynamic conception of both Riva-Rocci's and v. Basch's instruments. Even here, however, although the pulse wave plays an insignificant part, it is not the mean blood-pressure which is measured, but a blood-pressure which closely approximates the maximum. Another advantage seems to the writer to be that it depends upon relations which are appreciated by the keenest of our senses—namely, the sense of sight.

Gärtner also considers that the resistance of the arterial wall, which especially in arteriosclerosis plays so important a part, influences the measurement less in the small finger arteries than in the large vessels. Another advantage is that the patient does not have to be undressed when Gärtner's method is employed. The normal pressure values Gärtner has estimated vary between 90 and 105 mm. There is only one objection to his method, and he does not deny it, nor has he been able to obviate it as yet. This is that the readings are decidedly influenced by the resistance of the tissues of the finger-tips. This resistance is very different in

[1] *Münch. Med. Woch.*, 1903, No. 24, p. 1021.

[2] G. Gärtner, Ueber einen neuen Blutdruckmesser (Tonometer), *Wien. Med. Presse*, 1899, No. 26. L. Schulmeister (Vienna) and F. Hugershoff (Leipzig) make the instruments.

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a laborer's hand and in a lady's hand. In the writer's opinion this source of error is so considerable that it discounts the value of the entire procedure, and the description of certain technical modifications of this method[1] will consequently be omitted, since they do not obviate this chief fault.

[Within the last few years the usefulness of blood-pressure determination has been quite generally recognized in America. Both here and in England new instruments have been brought forward, some of which represent a distinct advance over any of those previously mentioned, and merit separate description.

Hill and Barnard's sphygmometer[2] appeared a little after Riva-Rocci's, and uses the same method of circular compression. It does not, however, measure the pressure required to obliterate the pulse (systolic or maximum pressure), but, like Mosso's, the point of maximal pulsation (diastolic or minimal pressure). This is accomplished by the employment of a delicate spring tambour, graduated in mm., Hg., as the manometer (Fig. 61) shows the apparatus. The cuff, a hollow bag, 4½ cm.

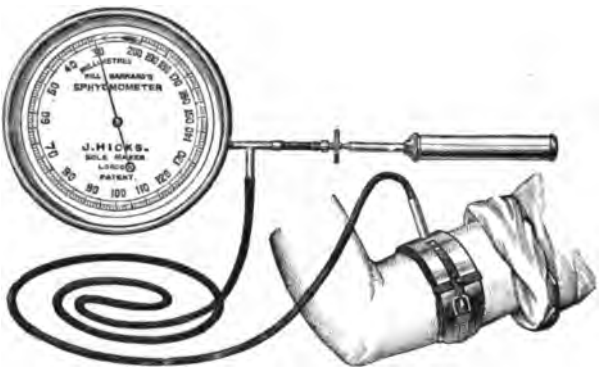

FIG. 61.—Hill and Barnard's sphygmometer.

wide, with an outer leather armlet, is buckled around the arm, and connected with the manometer by a screw-joint. The pressure is then raised by means of the hand-pump until the needle shows diminishing excursions on the dial. Then, by unscrewing a valve in the stem of the pump, the pressure is allowed to fall gradually, while the needle is closely watched. Its oscillations increase for a time to a maximum, then, after remaining the same for a short time, rapidly decrease. The last point at which they remain maximal represents the minimum arterial pressure, as already described. Hill thought that the midpoint of maximum oscillation was equivalent to the mean arterial tension, but this has been proved inaccurate. The instrument is well constructed, light, and compact. It has two great drawbacks. One is the narrowness of the cuff, for reasons which will follow. The other is, that so delicate

[1] See Martin, *Münch. med. Woch.*, 1903, No. 24, p. 1021.

[2] Hill, L., Barnard, H., *Brit. Med. Jour.*, 1897, vol. ii., p. 904. Made by J. Hicks, London; Agents for the United States, Oelschlager Bros., 42 East 23rd St., New York City.

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a manometer needs constant standardizing by comparison with a mercury one, and soon becomes inaccurate.

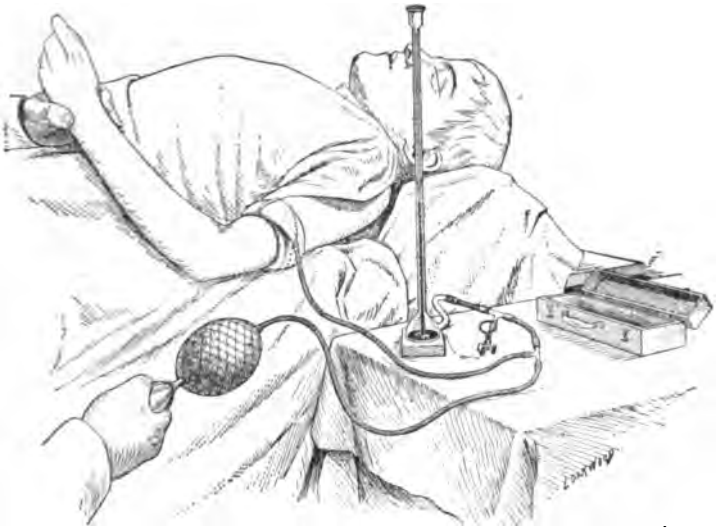

FIG. 62.—Riva-Rocci sphygmomanometer as modified by Cook.

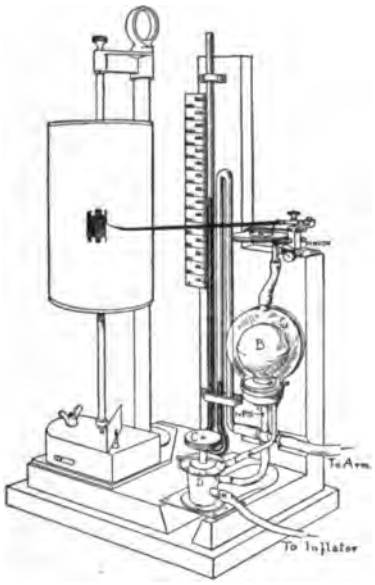

FIG. 63.—Erlanger's sphygmomanometer.

Hill and Barnard's so-called pocket sphygmometer is hardly more than a clinical toy. Oliver's dynamometer[1] cannot be recommended

[1] Oliver, George, *Jour. of Physiol.*, 1897-98, vol. xxii, p. 51.

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because it possesses all the faults of v. Basch's method of applying the pressure by a pad over the artery.

In America, Cook rendered a great service by so modifying the Riva-Rocci apparatus as to make it inexpensive and easily carried,[1] thus securing for it a much more extended application. His instrument so closely resembles its oirginal in the technic of its use that it needs no further description.

Two American instruments are now made with a 12 cm. cuff, Erlanger's and Janeway's. The former[2] is, undoubtedly the most accurate and valuable sphygmomanometer yet constructed. It gives readings both of systolic (maximum) and diastolic (minimum) pressure, and therefore makes possible the calculation of exact mean arterial pressure. Fig. 63 shows the apparatus in perspective.

Besides the manometer, compressing armlet, and inflator it contains a somewhat elaborate reading mechanism and kymographiondrum. This mechanism consists of the tambour, the interior of which is connected with the air chamber inside the glass bulb *G*. This air chamber has no other openings while the record is being made, but automatically

FIG. 64.—Janeway's sphygmomanometer.

connects with the outer air through the tube *E* and the stop-cock *C* when rapid changes in pressure are made, which might damage the tambour. The pulse waves are transmitted through the tube to "*P S*" and cause variations in volume of the rubber bulb *B*. These pulsations of *B* are, of course, reproduced by the air within *C*, and thus carried to the tambour, which inscribes them on the smoked cylinder. The purpose of the rubber bulb *B* is to shield the tambour from too sudden and great variations of pressure. The stop-cock is an important mechanism, but cannot be described intelligently without mechanical drawings. It is easily understood from the instrument itself. Unfortunately this apparatus is too bulky to be carried far, and is more complicated than desirable for strictly clinical work. For purposes of

[1] H. W. Cook, *Jour. Am. Med. Assoc.*, 1903, vol. xl., p. 1199.

[2] Erlanger, J., *Am. Jour. of Physiol.*, 1904, vol. x., Proceed. of Am. Physiol. Soc., p. 14.

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physiologic experiment on human beings, and whenever very accurate readings of both pressures are desired, it should be the choice.

Janeway's sphygmomanometer[1] is shown in Fig. 64. Its special feature is the portable U-tube manometer attached to a case, into which it folds for carrying. The whole when closed measures $10\frac{1}{4}$ x $\frac{5}{8}$ x $1\frac{3}{8}$ in., and with cuff and inflator weighs $2\frac{1}{2}$ pounds. The armlet is a hollow rubber bag, 12 x 18 cm., loosely covered and attached to an outer leather cuff, 15 x 33 cm., which fastens by two straps with friction buckles. For inflation a Politzer bag with valve is used. *E* is a stop-cock provided with a needle valve, by which the pressure can be reduced gradually. The method of getting the systolic pressure is practically the same as with Riva-Rocci's sphygmomanometer, the pressure being raised by squeezing the inflator until the radial pulse is lost, then gradually lowered until it returns. The reading at the moment of

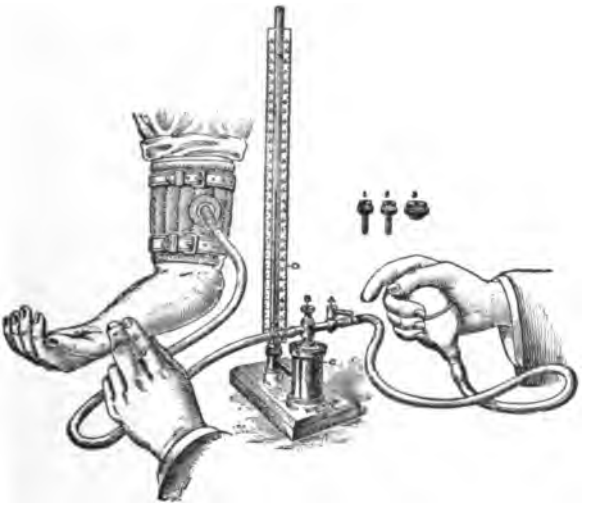

FIG. 65.—Stanton's sphygmomanometer.

return represents the systolic pressure. In addition, an approximate determination of the diastolic pressure can be made by noting the pressure at which the mercury shows the greatest pulsation (see sphygmomanometer). This is done by allowing the pressure to fall 5 to 10 mm. at a time after the return of the pulse has been detected, and observing at least 10 pulsations at each point. After a certain level (in normal pulses 25 to 40 mm. below the systolic pressure) the extent of the oscillations diminishes rapidly. The lowest point at which it remains maximal is the diastolic lateral pressure. The estimation of the two pressures is the only clinical method of computing mean arterial pressure, and has considerable diagnostic value in cases of aortic insuf-

[1] T. C. Janeway, *The Clinical Study of Blood-pressure*, New York, 1904, D. Appleton & Co., p. 89.

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ficiency and hypertension, where the elevation of systolic is much more marked than that of diastolic pressure.

The remaining American instrument, Stanton's,[1] is shown in Fig. 65. The manometer consists of a metal cistern (*C*) connected with a glass upright tube and scale (*D*), which can be unscrewed for carrying. The armlet is a hollow rubber bag, 3¼ in. (8 cm.) wide and 16 in. long, closed at both ends, and attached to an outer cuff of thick canvas, reinforced by tin strips. While it has not the width necessary for absolute accuracy, it nevertheless suffices for the vast majority of cases. A single rubber bulb is used for inflation. At *A* is a stop-cock, and at *B* a screw valve for the gradual lowering of pressure. With this sphygmomanometer systolic pressure may be measured and diastolic pressure approximately estimated, as already described with Janeway's apparatus. It is a portable and convenient clinical instrument.—T. C. J.]

VISIBLE PHENOMENA OF MOTION IN THE VESSELS.

CAPILLARY PULSE.

UNDER normal conditions the blood flows smoothly and without a pulse in the capillaries, because the resistance in the smallest arteries completely deprives the pulse wave of its energy at this point, or because, as v. Frey and Krehl believe, the pulse wave is completely reflected centripetally (see p. 117 *et seq.*). Under some conditions, however, the pulse is transmitted to the capillaries, and becomes evident to inspection in the form of a pulsating reddening and blanching of the parts in question. Whatever facilitates the entrance of a pulse wave into the capillary areas, or whatever renders the flow into the veins difficult, will, of course, favor the production of a capillary pulse. The larger the pulse wave and the more it approaches the type of "pulsus celer," the more the conditions for a capillary pulse are favored. A capillary pulse is sometimes observed over hyperemic, and especially over inflammatory, areas—*e. g.*, over felons. Very frequently the patient himself appreciates the increased pulsation in inflammatory parts as a throbbing pain. The capillary pulse due to a pulsus celer, especially in aortic insufficiency, is of far greater interest. This is a very common, although not a constant, sign in this valvular lesion. It is perhaps best appreciated by observing the alternate blushing and pallor at the finger-nail. Sometimes enough pressure upon the anterior part of the nail-bed to blanch the nail brings out the margin between red and white which oscillates with systole. The capillary pulse in aortic insufficiency may be very frequently appreciated at other places which are characterized by their redness—*e. g.*, ears, lips, cheeks.

[1] W. B. Stanton, *Univ. of Penna. Med. Bull.*, 1903, vol. xv., p. 466.

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A clean glass slide lightly pressed upon the extended lower lip will sometimes bring out the capillary pulse when it cannot be appreciated at the finger-nail. Another useful device is to rub a spot upon the forehead until it is hyperemic and then look for an alternation of redness and pallor.

Contrary to many statements, a capillary pulse is by no means pathognomonic of aortic insufficiency. Any condition which will produce a "pulsus celer" (exophthalmic goiter, fever, chlorosis) will be apt to show a capillary pulse. Even in health it may sometimes be observed. Yet although not absolutely pathognomonic of aortic insufficiency, it is so common in this lesion and so rare in other conditions that the sign really possesses considerable diagnostic significance. The sign becomes most distinct during the stage of compensation. The retinal vessels will also show a visible pulsation by the ophthalmoscope when a capillary pulse is visible elsewhere.

RESPIRATORY PHENOMENA OF MOTION IN THE VEINS.

The respiratory variations in the interior of the thorax, as is well known, influence the venous circulation very distinctly. Inspiration favors, expiration retards the flow of venous blood. This influence is

FIG. 66.—Venous engorgement during forced expiration—case of phthisis (New York City Hospital).

ordinarily not evident in the visible veins with superficial breathing, but forced breathing will produce an inspiratory diminution and expiratory increase in these veins, and if they are already distended by congestion the change will become still more evident. Both conditions are usually present in dyspnea.

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Intrathoracic variations in pressure become more distinct during coughing or other exertions of abdominal pressure. The intrathoracic pressure then becomes markedly positive. A coughing paroxysm or straining after a deep inspiration produces a prolonged distention and a subsequent sudden collapse of the veins, most plainly seen in the neck. When this periodic congestion is frequently repeated, especially in patients who suffer from chronic cough, a permanent dilatation of the veins, especially of the jugular, may result, so that with coughing or straining not only is cyanosis very marked, but the whole lower portion of the neck becomes swollen. The "bulbs" of the jugular vein may appear as large swellings, either just inside or outside of the insertion of the sternocleidomastoid (Fig. 66). The bulging of the supraclavic-

FIG. 67.—Enlargement of jugular vein in a case of leukemia with enlarged mediastinal glands (Dr. Joseph Collins, New York City Hospital).

ular fossæ during a cough, therefore, should not always be regarded as a distention of the lung apices (p. 98 *et seq.*).

In very rare cases the reverse condition is observed—*i. e.*, a distention of the veins during inspiration and a collapsing or a diminution during expiration; this always suggests that during inspiration some mechanical compression of the veins exists within the chest interior. This phenomenon has been described as a sign of fibrinous mediastinitis, like the pulsus paradoxus (see p. 126). It may, however, depend upon the effect of inspiratory pressure or traction upon the large veins leading to the heart, due to interference with the mobility of the thoracic contents (pericarditis, pleuritis, mediastinal tumors).

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DIFFERENT VARIETIES OF VENOUS PULSE.

DIFFERENTIATION OF VENOUS PULSATION FROM ARTERIAL PULSE.

When the distended veins become distinctly visible (in venous pulse the external jugular veins are chiefly concerned), it is generally a simple matter to distinguish between their pulsation and that of the neighboring arteries. The pulsation of some deeply seated vein (internal jugular) which cannot be directly observed is much more difficult to determine. But even then the venous pulse can be easily recognized, particularly by the large area of pulsation involved corresponding to the large size of the vein, also by the slow, undulating transmission of the beat, and by the very moderate amount of power to be felt in the pulsation, depending upon the slight amount of tension of the venous contents. If the pulsation is transmitted from an artery to a vein, compression of the vein will not affect the pulsation peripheral to the point of compression, and sometimes the resulting congestion will make the pulsation even more distinct. An accentuation of the pulsation from compression otherwise occurs only with the very rare penetrating pulse (see p. 152).

PHYSIOLOGIC VENOUS PULSE.

(Negative Venous Pulse; Systolic Venous Collapse; Venous Undulation; Negative Centrifugal Venous Pulse; The Presystolic Variety of the Negative Venous Pulse.)

The arterial pulse wave usually disappears in the capillaries—*i. e.*, it is reflected centripetally (see p. 144 *et seq.*), so that the blood no longer pulsates but flows uniformly in the venous radicals. Nevertheless under both physiologic and pathologic conditions peculiar pulsations synchronous with cardiac action are frequently observed in the greater veins lying near the chest (almost exclusively in the jugular veins).

In the following sections we shall describe several pathologic varieties of this pulsation. One type of venous pulse must, however, be considered absolutely physiologic, because it is constantly observed after exposing the vein in healthy animals, and because perfectly normal individuals sometimes show it. That this venous pulse is not observed in everybody is due to the fact that in some people the jugular veins are not visible or are seen only with considerable difficulty. Conversely, this physiologic venous pulse can naturally be seen with especial readiness if the veins have become more noticeably distended by congestion.

The *physiologic venous pulse* can be distinguished from the pathologic varieties to be considered later by the circumstance that compression of the vein by the finger obstructs the pulsation above the point of pressure, while below the compression the pulsation either disappears or diminishes, but never increases. This central obliteration or diminution of the pulse wave proves beyond a doubt that the pulse wave is not thrown

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back into the veins from the heart. The peripheral disappearance of the pulse wave proves that it cannot be a wave transplanted from an artery to the vein. The only remaining hypothesis is that the cardiac activity does not force the blood back into the veins, but that the continuous blood-current is rhythmically retarded and accelerated. This type of a venous pulse is generally called a negative venous pulse, because it depends upon the transmission of a negative trough or suction wave from the heart to the vein (see below). It is also sometimes termed an undulation on account of its appearance. Probably the reason that the undulation does not cease entirely centrally from the point of compression, as we should naturally expect from our supposition of its origin, is because it is almost impossible to compress the vein sufficiently to prevent the influx. The diminution may be rendered more distinct by compressing the subclavian as well as the jugular vein. The internal jugular should always be compressed, never the external jugular alone. The fact that the venous valves are intact does not in any way prevent the transmission of the physiologic venous pulse toward the periphery (*e. g.*,

FIG. 68.—Physiologic (negative) venous pulse (Riegel).

from the "bulbus" to the vena jugularis), because it is a negative wave motion which traverses the vein in the direction of the valve openings, according to the laws of wave motion.[1]

Fig. 68 represents a sphygmogram of the curves of the *carotid pulsation* taken simultaneously with the curves of the physiologic venous pulsation (Riegel). The latter is both visible and palpable in the external as well as in the internal jugular. Comparing the two, it is evident that the venous collapse corresponds to the rise of the carotid pulse, or, roughly expressed, the physiologic venous pulse is cardiodiastolic. Hence the name "systolic venous collapse."

If we neglect the peculiar wave (c') in the ascending limb of the venous curve, the normal venous pulse can be easily explained by assuming that the venous reflux depends largely upon the condition of the right auricular contraction. For auricular diastole (ventricular systole) seems to favor the reflux, and auricular systole (ventricular diastole) seems to check it. Estimating the time relations in Fig. 68 shows, however, that any such simple explanation is insufficient and

[1] *Cf.* Tigerstedt, *Lehrbuch der Physiologie des Kreislaufes*, 1893, p. 367.

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impossible. Besides, other influences synchronous with the heart beat affect the venous circulation—viz., first the state of contraction of the right ventricle or the suction effects, and second the effect of the so-called auxo- and meiocardia—*i. e.*, the intrathoracic variations in pressure produced by the systolic diminution and diastolic increase in the size of the heart. Although cardiac systole does exert a certain amount of suction and the diastole exerts pressure upon the interior of the thorax, it is impossible to explain the alternation of the venous with the arterial pulse without considering all the other factors which have any influence.

Before submitting his own explanation, the writer wishes to insert the following in connection with Fig. 68: The point *a*, where the carotid wave begins to rise, does not correspond (as is generally accepted) to the commencement of ventricular systole, but to the beginning of the expulsion time. The beginning of systole must therefore be located a little earlier. Point *a'* in the venous pulse curve corresponds to the same moment of time as the point *a* in the carotid curve. Beginning at this point, we can explain the venous pulse as follows: At the moment (*a*) the total volume of the heart is diminished, meiocardia occurs and an intrathoracic suction action is combined with it; at the same time the auricle dilates, and consequently the conditions for the flow of venous blood are the most favorable. A venous collapse expressed in line *a' b'* is therefore the natural effect and explains itself. The diastole of the ventricle begins a little beyond the point *b*, at a point not indicated upon the descending limb of the carotid curve. From this point it is difficult to explain the curve, because during ventricular diastole various contradictory influences affect the flow of venous blood into the veins—viz., the diastolic suction power of the right ventricle,[1] diastolic auxocardia, with an exactly opposite action, further the contraction of the ventricle, which backs up the blood in the veins at the end of diastole, and finally the part of ventricular systole (called the closure time) which immediately precedes the ascending limb of the carotid curve, which really does prevent the entrance of the blood, although it does not diminish the intrathoracic pressure.

All these factors, except the suction power of the ventricle, tend to obstruct the venous flow and to produce the congestion in the jugular veins expressed in the ascending limb *b' a'*, which fades only at the onset of the expulsion time. The secondary elevation at (*c'*) of the ascending limb is, however, not explained. Its position corresponds very satisfactorily with the supposition that it is due to the presystolic contraction of the right auricle producing a backing-up in the veins.[2]

We must, however, be well acquainted with its peculiarities to avoid confounding it with the two following varieties of venous pulse. It is evident from our explanation of its origin that to recognize the diastolic character of the venous pulse it is essential to contrast the height of the venous pulse apex with the apex of the carotid pulse (*i. e.*, in regard to

[1] Expressed better, the diastolic release of the obstacle to the blood's entrance.

[2] Compare the scheme of the time sequence of the cardiac phases, in Bernstein, *Lehrbuch der Physiologie*, 1894, p. 61, Fig. 13.

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the time) and not with the cardiac apex beat. This is because the apex beat of the heart coincides with the "closure time," and so still belongs to auxocardia, and more nearly corresponds in point of time to the apex of the venous pulse than does that of the cardiac pulse.

(See p. 152 for further assistance in distinguishing the physiologic from the pathologic venous pulses.)

It has been shown by Riegel that an accentuated negative venous pulse can occur under pathologic conditions from an increased contraction of the auricle. In these cases the presystolic serration, corresponding to the auricular contraction, is the larger, and may be so pronounced that the entire venous pulse has the character of a presystolic pulse. This variety is seen particularly in marked degrees of venous congestion and in cases of pericardial exudates.

Although it was formerly supposed that liver pulsation always belonged to the category of the so-called positive centrifugal venous pulse (see following paragraph), Volhard has shown that it is very frequently such an accentuated negative venous pulse as has just been described. To obtain his proof he employed the procedure of manometric comparison described on p. 152.

POSITIVE CENTRIFUGAL[1] (REGURGITATING) VENOUS PULSE; LIVER PULSATION.

A positive centrifugal venous pulse is only observed with tricuspid insufficiency. It arises in this valvular lesion because during systole the blood is forced back into the right auricle, and from there into the veins. Its elevation corresponds to cardiac systole, as will be readily recognized by a simultaneous sphygmographic representation of the jugular and carotid pulse.

A positive venous pulse curve differs considerably from a negative venous pulse curve. A few secondary elevations are to be seen in its ascending limb, probably because the factors which produce the physiologic venous pulse are felt here, too, and so interfere with the actual regurgitation; perhaps also because the positive venous pulse, although it is systolic, does really precede the onset of the carotid beat a little. Of course, the regurgitation of the blood through the insufficient tricuspid valve begins at the beginning of systole—*i. e.*, at the "closure time" (here with reference to the right ventricle), and not, like the arterial pulse, at the "expulsion time." Hence, it would naturally precede the arterial pulse a little.

If the valves at the upper end of the jugular bulb close properly,

[1] The term centrifugal venous pulse has only one meaning here. The positive centrifugal pulse corresponds to a positive (rise) wave of centrifugal course, and is contrasted with a positive centripetal pulse, which comprises a positive wave of centripetal course. At the same time the pathologic venous pulse of tricuspid insufficiency considered here as a centrifugal pulse should hardly be contrasted with the physiologic variety considered as a centripetal pulse, because the latter is also centrifugal—*i. e.*, it also proceeds from the heart, and passes through the trunk of the vein toward the periphery. The only difference between the positive centrifugal venous pulse discussed here and the physiologic or negative pulse consists in the fact that in the latter a negative wave (trough) is transmitted centrifugally.

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the positive pulse will be chiefly apparent in the bulb (bulbous pulse). There may, however, be a slight wave transmitted to the periphery, because the closure of the bulbous valves only prevents the actual regurgitation of blood and not its wave-like motion. The latter, a positive wave, in closing the bulb valves produces above them, from the backing up, a positive wave of exactly the same shape. Consequently, by carefully watching a bulbous pulse we can appreciate that the veins do pulsate above the bulb valves, but decidedly less than below. The difference is that as far as the valves a real backward motion or regurgitation of the blood can be plainly observed, but above the valves only the wave motion started by the reflux. Under such conditions a distinct systolic tone may sometimes be produced over the bulbous valves by the shock of the regurgitated blood (jugular valve sound, see Auscultation of the Veins). Very frequently, however, as the result of congestion, the venous valves become insufficient, so that the positive venous pulse may be seen just as distinctly in the upper part and in the small branches of the jugular vein as over the bulb itself.

Another difference between the positive centrifugal venous pulse and

FIG. 69.—Positive centrifugal (regurgitating) venous pulse (Riegel).

the negative physiologic venous pulse is that the rise of the former occurs approximately at the same time as that of the carotid pulse. Again, when the pulsating vein is compressed, the positive centrifugal venous pulse persists, and sometimes even becomes accentuated centrally, but disappears peripherally from the seat of pressure.

The appearance of the regurgitating venous pulse is not infrequently limited to the jugular vein; but in pronounced cases the veins of the extremities may also pulsate. A pulsation in the liver veins is especially important in the diagnosis of tricuspid insufficiency.

The *liver venous pulse* can be appreciated by palpating the liver, which is usually much enlarged in tricuspid insufficiency. The examiner should palpate as far as possible to the right of the median line, so as to avoid confusion with an epigastric pulsation or with an aortic pulsation, which is sometimes transmitted to the liver. (See later section upon Palpation and Inspection of the Cardiac Region.) To avoid such errors it is especially important to be convinced that the pulsation is really expansile—*i. e.*, that the volume of the liver increases intermittently. This can generally be best accomplished by firmly grasping the edge of the liver, or by employing bimanual palpation, one hand

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behind pressing the liver forward against the other hand in front. An *arterial liver pulse* sometimes occurs in aortic insufficiency as a result of the "pulsus celer." It can be distinguished from the *venous liver pulse* only by a careful consideration of all the other conditions.

The writer once demonstrated an *inflammatory liver pulse* over the left lobe of the liver as the result of a cholangitis following gall-stone colic. Duroziez's double murmur (see later) was heard over this lobe. The pulsation and the murmur both disappeared after several weeks, and the autopsy revealed a purulent cholangitis at the place in question and several miliary abscesses in the liver.

POSITIVE CENTRIPETAL OR PENETRATING VENOUS PULSE.

This rare type of venous pulse is produced when the arterial wave possesses sufficient force to penetrate the capillaries and transmit a pulsating motion to the venous radicals. The phenomenon depends upon conditions similar to those producing the capillary pulse, and has been found chiefly in aortic insufficiency. Quincke, however, believes that a capillary pulse does not always accompany a penetrating venous pulse; but, on the contrary, that we may be unable to appreciate the pulse wave in the capillaries because it is spread over too great an area, whereas the constriction that the venous radicals under favorable pressure conditions oppose to the course of the current may cause the pulse to again become visible. This type of venous pulse occurs especially with an arterial "pulsus celer." It does not appear in the jugular vein, but in the small veins of the extremities. Compression will obliterate the pulse in the central portion, but not in the peripheral part of the vein.

DIASTOLIC VENOUS COLLAPSE (Friedreich).

This very rare phenomenon was described by Friedreich in connection with systolicr etraction of the cardiac region from pericardial adhesions. (See later Palpation and Inspection of the Cardiac Region.) The diastolic recession of the chest-wall produces a diastolic suction within the thorax, and this is supposed to cause the veins to collapse. It is, so to speak, the reverse of the negative or physiologic venous pulse. In the latter the vein is distended during diastole; in the phenomenon described by Friedreich the distention occurs during systole. Therefore it may be easily confounded with the positive centrifugal (regurgitating) venous pulse, which is also systolic. Compressing the vein will, however, readily settle the question, for the diastolic venous collapse acts not like a positive centrifugal, but like a negative venous pulse—*i. e.*, it disappears, or is at least much diminished, centrally to the point of compression.

VOLHARD'S PROCEDURE FOR DETERMINING THE PHASES OF THE VENOUS PULSE; PRACTICAL DIFFICULTIES IN DISTINGUISHING THE DIFFERENT KINDS OF VENOUS PULSE; COMBINED VENOUS PULSE.

In view of the great difficulty in recognizing the phases of a venous pulse by the eye and hand, and the technical details necessary for its graphic representation, Volhard[1] has furnished a clever and simple procedure by means of which we can easily determine the time of a venous pulse in reference to the carotid pulsation. By means of small glass funnels, one of which is placed over the pulsating vein (or the liver) and the other upon the carotid, Volhard transmits the movements of the pulse to two water manometers which are filled with differently colored fluids and placed side by side. It can then easily be determined whether the fluids are displaced in the same or in opposite directions. The quickness of the movement is also an indication as to whether we have to do with a positive or with a negative pulsation (collapse) of the vein. On account of the influence of

[1] *Congr. f. inn. Med.*, 1902, p. 402.

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inertia the water must be at the same level in both manometers. The length of the column of air, however, is immaterial, since the air waves are transmitted with the same velocity as sound. In the author's experience the procedure sometimes gives very clear results, but it frequently fails owing to the difficulty of separating the venous pulse completely from the transmitted movement of the carotid.

Although the preceding distinctions apparently render the differentiation of the different kinds of venous pulse perfectly plain, we are nevertheless frequently confused in practice, especially because many venous pulses are combined phenomena. It has already been noted that the positive regurgitating venous pulse is frequently combined with the signs of the physiologic venous pulse because, naturally, with a tricuspid insufficiency the physiologic undulations of the veins would not disappear. The typical character of the positive regurgitating venous pulse must then be determined by means of the time relation of the pulse to the cardiac phases and by the results of the compression test. Even the physiologic venous pulse is frequently deformed because the carotid pulse imparts itself to the venous contents and antagonizes the venous pulse, both in its time phase and in the relation to the action to the compression test. In many of these cases it is absolutely impossible to determine definitely the nature of the venous pulse.

PERCUSSION.

PERCUSSION IN GENERAL; INSTRUMENTS.

THE examination of the human body by means of percussion (or striking) plays an especially important rôle in the modern diagnostic methods of internal medicine. The condition of the organ or organs underlying the spot percussed is determined or, at least, inferred from the sound produced. We accredit the discovery of this method to the Vienna physician Auenbrugger, for in the year 1761 a description of it appeared in his work, *Inventum Novum*. However, the method was not generally employed until after Corvisart, Napoleon I.'s body physician, translated Auenbrugger's work into the French (1808) and appended extensive observations of his own as commentaries. Manifold modifications, theoretic confirmations, and symptomatic improvements were then added to the method by a great number of authors, of whom we need only mention Piorry, the inventor of the pleximeter; Barry, the inventor of the percussion hammer; Wintrich, Skoda, and Traube. Hence, although the method is already more than a century old, it really only became the common property of physicians during the second half of the nineteenth century. To-day percussion and auscultation have become the corner-stone of diagnosis. The student must learn both at the very beginning of his clinical experience.

There are countless methods of percussion. Originally only *immediate percussion* was employed—*i. e.*, the striking of the surface of the body directly with the tips of the fingers. But where the body-covering is soft such a method will elicit only indistinct and impure sounds, and therefore to-day we almost exclusively use *mediate percussion*—*i. e.*, either a finger of the other hand or a specially constructed

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instrument—the "pleximeter"—is inserted between the striking finger and the surface of the body. The "plexor" is a small hammer furnished with a rubber tip. It may be substituted for the striking finger.

In *mediate percussion* we may use either: (1) Finger-finger percussion, (2) finger-pleximeter percussion, or (3) plexor-pleximeter percussion.

Individual taste or habit will generally determine a preference for one of these methods. There are certain differences and advantages in each of them, although we may be skilful or awkward with any one of them. The finger-finger percussion is the most difficult to learn; but in many cases it furnishes more accurate results than the pleximeter percussion, because the inherent note of the pleximeter is liable to confuse the percussion tone. Consciously or unconsciously, the sense of touch in the finger-finger percussion aids the sense of hearing (palpation percussion).[1] But the chief advantage is that the physician is independent of instruments, which may be readily forgotten, mislaid, or broken. The finger pleximeter, and especially the plexor-pleximeter percussion methods, are much easier to learn, and greater differences of sound may be more readily demonstrated and appreciated by a larger circle of listeners; nevertheless, a percussion note which is loud enough to be heard at considerable distance is generally faulty (see below). A physician should be familiar with all three methods; and he should be able to control his own results by applying first one and then the other, for despite the utmost care and great skill, the subjective character of the method often in difficult cases leaves a doubt in the mind of the examiner. A plexor and pleximeter are of no great advantage in the employment of the plexor-pleximeter method (p. 158), because we can substitute a bit of wood, a pencil, or the like for the flexor, and a coin or another bit of wood for the pleximeter.

In certain cases—*e. g.*, for determining slight pathologic dulness in the chest or abdomen or for differentiating the deep cardiac dulness—*mediate percussion* is to be recommended as a control. The examiner thus avoids too strong pressure of the pleximeter or of the finger of the left hand, a very common fault with beginners. *Mediate percussion* is generally made by striking with the tips of the four fingers and thumb of the right hand arranged like a pyramid.[2] In percussing the lung apices beneath the clavicles the bone serves as a pleximeter, and the middle finger of the right hand as the plexor.

Only continued practical experience can teach the technic of percussion, and but few rules are worth stating.

The percussion stroke should be made perpendicular to the surface of the part percussed. In finger percussion the nail of the percussing finger should be cut short, and the blow should be struck with the pulp of the last phalanx in such a manner that not only the direction of the stroke but also the axis of the last phalanx is perpendicular to the pleximeter and to the surface of the body part percussed. A perpendicular stroke is essential for producing a good, equal note. It is especially

[1] [The fine appreciation of variations in resistance to the pleximeter finger is of almost equal importance to the recognition of variations in resonance.—Ed.]

[2] [Most American clinicians use the middle finger of the right hand as the plexor and the middle or first finger of the left hand as the pleximeter.—Ed.]

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difficult for the beginner unless he is a piano player. Both in plexor and in finger percussion the stroke should be light, short, and elastic, produced by merely bending the wrist joint, at the same time avoiding any cramped position of the hand or fingers. Percussion should be *light* or *superficial* and even (see p. 163). The so-called *strong* or *deep percussion* should also be quite delicate, so that the note cannot be heard at any great distance.

The plexímeter—*i. e.*, the finger to be struck—should be placed with its palmar side upon and in close contact with the surface of the body and parallel to the border to be differentiated. Only slight pressure should be employed, because strong pressure even with light (superficial) percussion will cause a diffuse vibration of the body, and so simulate the effect of a stronger (deep) percussion.

Instead of the usual plexímeter, a bit of firm, gray erasing rubber, cut about 1 cm. wide, 1 cm. thick and 4 cm. long, will answer the purpose well if the physician is not skilled in finger percussion. Like the finger, such a plexímeter has practically no intrinsic curve; it can be easily bent to conform to the curves of the chest-wall, and it can be struck with the hard end of the hammer or with the finger.

In percussing a child's body very much less force must be used than for the adult. (See Deep and Superficial Dulness.)

QUALITY OF THE PERCUSSION.

RESONANT AND DULL TYMPANITIC AND NON-TYMPANITIC PERCUSSION NOTES.

Even a layman, percussing different regions of the body surface, is able to recognize that one part furnishes a resonant, another part a dull, note. An accurate differentiation between resonant and dull notes, or between different grades of resonance, is the groundwork of percussion. A typically resonant note can be obtained by percussing over the lung; a typical dull note, by percussing over great muscle masses—*e. g.*, the thigh. Experience has taught us that resonant notes are produced over organs containing air; dull notes over organs without air, quite irrespective of whether they are solid or filled with fluid. Therefore percussion, on the one hand, defines the boundaries of the different organs; and, on the other hand, by changes in the resonance of the note, determines any increase or decrease of air contained in the organ. The less air, other conditions remaining the same, the duller the note. The resonance of the note also varies decidedly with the deep diameter of the air-containing organ—*i. e.*, the diameter in the direction of the percussion stroke. The thicker the layer of air the more resonant the note. The influence of the tension of the wall surrounding the air cavity upon the resonance of the note will be mentioned later.

We apply the term loud, clear or resonant as contrasted with faint, dulled, dull or flat to the percussion notes of the ordinary living body. When a resonant note is modified by a dulled note, we speak of the presence of *dulness;* a note which is neither very resonant nor very

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dull is called *relatively dulled;* an absolutely dull note, on the contrary, *absolutely dulled* or *flat.* The terms *absolute* and *relative dulness* have a corresponding signification.

In defining the quality of a percussion note many modern authors employ the terms short and long as synonymous with dull and resonant. Theoretically this is not quite correct, because the latter expressions characterize the intensity of the note; whereas short and long refer, on the contrary, to its duration, and the latter must depend upon the size of the vibrating mass. The length of a musical instrument's note is of very essential importance to the tone and value of the instrument. A musical ear can, in fact, easily differentiate this peculiarity of duration in a percussion note. However, in the relations of percussion which concern us a resonant note is almost always long and a dull note short. The exceptions to this rule have no special diagnostic importance, so that the expressions short and long may as well be eliminated.

A consecutive series of note qualities range in countless gradations between the two limits resonant and dull, the differences depending upon the amplitude of the note vibrations. There are, however, other very important differences of quality which we must heed and which depend upon variation in the form and number of vibrations. The most important of the latter is the distinction between *tympanitic* and *non-tympanitic* resonant notes. This distinction is illustrated by comparing in a healthy person the note obtained over the lung with that obtained over the air-containing abdominal viscera. The pulmonary note is resonant, but not tympanitic. The note over the stomach and intestines is resonant, but also tympanitic. Both tympanitic and non-tympanitic notes are really noises in the purely physical sense; but in tympanitic notes the vibrations are sufficiently periodic (*i. e.*, tuneful) for the ear to be able to compare their number with the number of vibrations of other sounds; in short, to be able to recognize a certain pitch. This theoretic differentiation between tympanitic and non-tympanitic notes will not make the matter much clearer to a beginner; but in a practical demonstration a musical ear will very readily appreciate the distinction between the resonant but non-tympanitic lung note and the true tympanitic note of the bowels. No sharply defined boundary really exists between tympanitic and non-tympanitic notes.

Various explanations have been adduced to show how at one time an air-containing organ furnishes a resonant non-tympanitic note, and at another time a resonant tympanitic note; but most of these explanations have no trustworthy physical basis and are not tenable. Physicists have thus far busied themselves very little with this acoustic question.

A discussion of the so-called theories of the tympanitic and non-tympanitic notes may therefore well be omitted, and merely the facts mentioned as to when the non-tympanitic note of an air-containing organ is transformed into a tympanitic note, and *vice versâ.*

Experience teaches us that a non-tympanitic note will be transformed into a tympanitic note when the tension of the air within the organ diminishes—*i. e.*, when its wall is relaxed; and the converse is also true, that with an increase in tension a tympanitic will be transformed into a non-tympanitic note. If we inflate a pig's bladder while at the same

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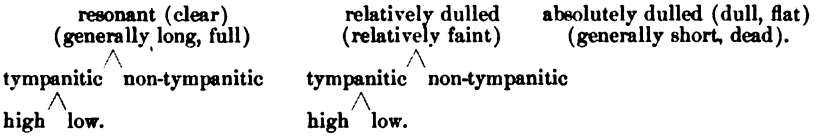
time we percuss over the bladder, we shall notice that up to a certain point of distention the note is tympanitic, but with further inflation non-tympanitic. During the transformation of a non-tympanitic into a tympanitic note its intensity or sonority increases; in other words, the note first becomes hyperresonant before it becomes tympanitic.

The essential characteristic of a tympanitic note, as has been said, is a certain definite pitch, so that naturally we can distinguish a low tympanitic from a high tympanitic note, and gradations between the two. Different tone heights (pitch) cannot be distinguished easily in non-tympanitic notes. The pitch of a percussion note depends upon various factors, above all upon the tension of the wall enclosing the air space and upon the size of the latter.

It is self-evident that more than one of the qualities discussed may characterize the same percussion note. Thus, *e. g.*, we may speak of a relatively dulled high tympanitic note; or of a resonant low tympanitic note.

The following scheme of the tone qualities described above may be useful:



There are two other specific tone qualities which must be mentioned —viz., *metallic resonance* and the "*cracked-pot*" *sound* or *cracked-pot resonance.*

METALLIC RESONANCE.

Metallic resonance means a peculiar quality of the percussion note, which is best characterized by its name. It can be imitated by percussing one's own distended cheeks with a plexor and pleximeter.

Its metallic character depends upon an individual metallic overtone of a definite pitch, which can be appreciated sometimes during the entire duration; sometimes, however, only at the end of the sound. In the latter case we speak of it as a *metallic after-resonance.*

We are especially indebted to Wintrich for experiments upon metallic resonance. He showed that this sound arises only when large air spaces are percussed. These may be closed or open; but if open the orifice must be relatively narrow—*i. e.*, in proportion to the cross-section of the cavity. The inner surface of the cavity-wall must be comparatively smooth; because, according to Wintrich, metallic resonance depends essentially upon the reflection of the air waves which percussion sets into vibration within the cavity. This produces the high inharmonious overtones responsible for the metallic character of the sound. The thinner the walls the easier the metallic character can be appreciated.

If the walls of the cavity are yielding (flexible), metallic resonance will not result unless they are under a certain moderate tension. Again,

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a cavity must be of a certain size to produce metallic resonance; according to Wintrich, its greatest diameter must be at least 6 cm. Small cavities very rarely furnish metallic resonance.

The metallic resonance noted in percussing the normal human body is in most cases so faint that it can be appreciated only when the ear is very near or is connected with the percussed area by means of a stethoscope (auscultatory percussion). The easiest way to appreciate metallic resonance is by means of the "stick-plexímeter" method. In this method we percuss with the handle of the plexor upon a bit of stick, a coin, or some other firm object placed upon the body, and at the same time auscult with the stethoscope in the immediate neighborhood. By the reverberation of its high overtones the resulting shrill noise seems particularly to favor the production of a metallic resonance. Metallic resonance may accompany non-tympanitic as well as tympanitic notes; but in the former case it can be appreciated only by the aid of the stick-plexímeter method of percussion.

Metallic resonance can be elicited sometimes over physiologic cavities—*e. g.*, the stomach and intestines; sometimes over pathologic collections of air—*e. g.*, lung cavities, pneumothorax, pneumopericardium.

If the metallic resonating cavity contains fluid as well as air, change of the patient's position may alter the pitch of the metallic resonance. This is because its pitch depends partly upon the greatest diameter of the air cavity, which would be altered by the shifting of the relative positions of the fluid and the air (see p. 213).

CRACKED-POT RESONANCE.

(Bruit de Pot Fele; Noise of the Spinning-top, of the Chink of Coins.)

This peculiar clanging or rattling percussion noise can be simulated by filling the hand with coins, shutting it tight enough to allow only a slight space for the coins to move, and then shaking the hand; or, in another way, by clasping the hands together firmly, leaving a slight air space between, and then striking the back of one hand against the knee. The force of the blow will expel some air through the narrow chink between the hands. Another method is to strike a hollow rubber ball with a narrow opening vigorously enough to expel some air with each stroke. Such experiments, as well as the conditions in the chest which are responsible for the phenomenon, make it probable that the cracked-pot sound observed in man is a stenotic murmur, made by the percussion blows expelling air quickly through a narrow slit-like opening. (See p. 213 for its diagnostic significance.)

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TOPOGRAPHIC PERCUSSION.

PERCUSSION CHARTS; SUPERFICIAL AND DEEP DULNESS OF ORGANS; LIGHT AND STRONG PERCUSSION; SITUATION OF THE ORGANS; ORIENTATION POINTS AND LINES.

Topographic percussion means the determination of the boundaries of the organs of the body by means of percussion. By this method we attempt to project the boundaries of the organs upon the body surface; and to represent these relations conveniently in the history sheets the

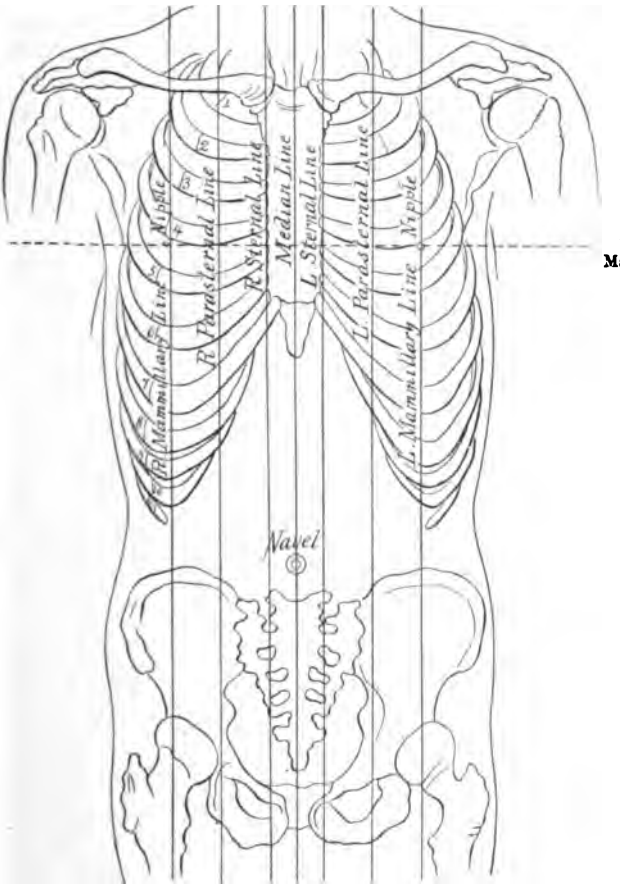

FIG. 70.—Chart of anterior body-half.

borders are sketched upon a chart of the human body, with a skeleton drawn inside. Figs. 70, 71, and 72 show such percussion charts. Perhaps they would be still more convenient if they were double the size. Since every man's skeleton is not of the same shape, it is advisable to represent by some mark (*e. g.*, a cross) the boundary points which are

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quite normal in their relation to the skeleton (see Fig. 79). Such a chart is rarely absolutely accurate.

The possibility of employing topographic bounding of organs which are situated in part one over the other depends upon the fact that some are filled with air and some are solid. A solid organ gives a dull, a hollow organ a resonant, note. In rare cases the qualitative variations of the resonant note can be utilized for defining the boundaries. For example, we can differentiate the resonant but non-tympanitic note of the lung, or the low tympanitic note of the stomach, from the high tympanitic note of the intestine. But naturally the differences are often very slight, and the qualities of the resonant notes merge into each other without sharp boundaries, so that the distinction is by no means easy.

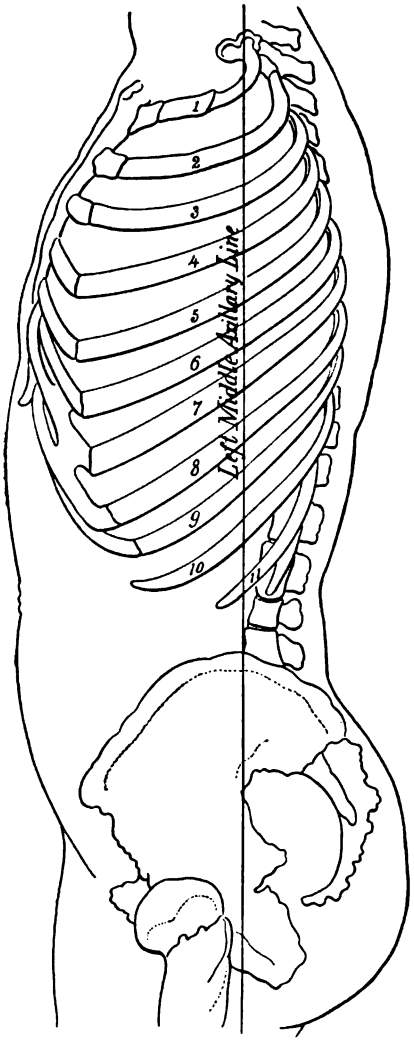

FIG. 71.—Chart of left lateral body-half.

The first essential for utilizing topographic percussion is to localize the percussion stroke. Where the boundaries to be defined are superficial—that is, where they lie directly under the body-wall—the lightest possible percussion will evidently succeed best. As soon as we percuss more vigorously, the vibrations will be strongly transmitted over the boundaries, producing a mixed note. *Therefore, it is a good general rule that to appreciate superficial boundaries we should percuss as lightly as possible.* In other words, the percussion should be gentle enough not to produce any note at all over solid tissues. Then the same strength of percussion will produce a very plain, resonant note just as soon as we encroach upon the edge of an organ containing air—*e. g.*, in differentiating the lower lung boundary from the liver.

This light percussion, valuable in estimating superficial boundaries, is called *superficial percussion;* and the dulness mapped out by the method is called *superficial dulness.* It is the easiest for the beginner

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to differentiate. Its position and extent generally correspond quite accurately to the position of the part of the organ which is next to the body-wall.

Special attention should be directed to another point in determining the superficial boundaries. The skilled observer instinctively heeds it; but the beginner, despite his best endeavor to percuss lightly, disregards it, and so fails in a correct differentiation. It is not only necessary to percuss

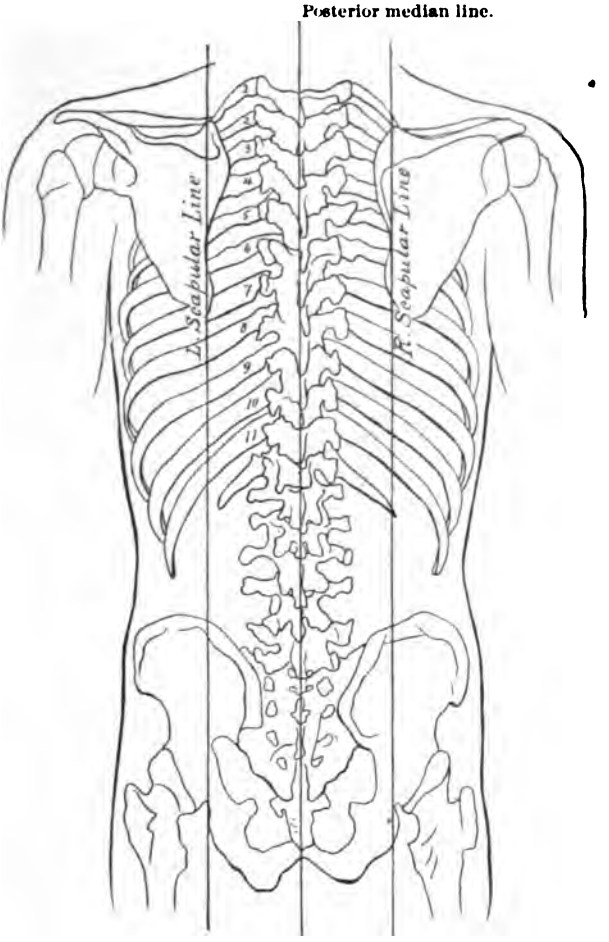

FIG. 72.—Chart of posterior body-half.

lightly, but the plexímeter, or the finger of the left hand serving this purpose, should be brought into very light contact with the surface of the body. The mere weight of the finger is sufficient. Firm pressure of the plexímeter (or finger) will simulate the effect of strong percussion, because the vibrations will penetrate much more intensely further into the depths and to the sides, and so prevent a linear localization of the boundary between two superficial organs, the one containing air and the other solid.

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In many cases the boundaries of organs which we wish to differentiate lie in the deeper parts—*e. g.*, most of the heart is covered by the lungs, and yet it is very desirable to determine its size by percussion. Naturally this is a much more difficult differentiation than that of superficial boundaries. Light percussion will be useless. On the contrary, the blow must penetrate the tissues to the depths of the organ in question, in order to obtain tone differences sufficient to furnish, at least approximately, conclusions upon the position of the deep-lying boundaries. The intensity of the note will also depend upon the thickness of the layer of air that we percuss. The more air-containing tissue that is set in vibration, so much louder (more resonant) the note.[1] If *a b* (Fig. 73) represents a cross-section through the anterior wall of the thorax, and *c d* one through the heart, then by vigorous percussion in the direction of the arrows *e* and *f* the elliptic shaded areas will be set in vibration. Weil has named such an area "the acoustic sphere of action." This acoustic sphere of action at *e* will be entirely within lung tissue, but at *f* partly in the solid heart and partly in lung tissue. Hence, a smaller volume of air-containing lung will be set in vibration by percussing at

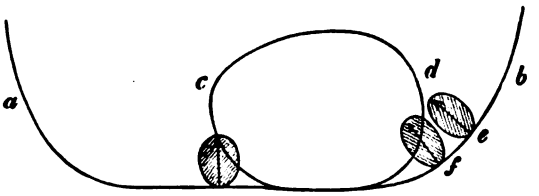

FIG. 73.—The acoustic spheres of action of the blow in deep percussion—origin of the deep dulness.

f than at *e*, so that the note over *f* will sound somewhat fainter, or, as we say, relatively dulled.

It should be remembered that repeated forcible percussion in certain cases increases the resonance. The probable explanation of the phenomenon is that the continued forcible blows excite a "lung reflex"—in other words, a localized temporary emphysema—in response to the irritation of the blows. The position of this relative dulness will, under some circumstances, enable us to mark out the organs which are situated more deeply. Such percussion, necessarily stronger than superficial percussion, is called *strong* or *deep percussion*, and the corresponding dulness, *deep dulness*.

The percussion stroke in superficial percussion is light and should include as small a sphere of action as possible; but the strength of stroke in deep percussion should be so adjusted that its sphere of action will reach to the deep-lying organs, as is shown in Fig. 73. It is very difficult for a beginner to arrive at the correct strength of percussion. It requires long practice. A helpful suggestion is that the strength of

[1] According to the law that the intensity of the note equals the mass multiplied by the square of the velocity. In consequence of the inertia of the vibrated mass, the tone will also be fuller; that is, the sound will last longer (see p. 155).

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percussion should be so adjusted as to produce the most intense dulness and to make the differences between the notes as plain as possible.

The beginner will err again by percussing too strongly, just as in superficial percussion (*p.* 155). *To bring out deep dulness, one should not percuss the patient with all possible strength and shake the greater part of the thorax, as negative or untrustworthy results are obtained by such a method. Ordinarily the stroke should be only a little more vigorous than is necessary to map out the superficial dulness. The pressure used in applying the plexímeter* (*or the finger of the left hand*) *to the body-wall should be only a little stronger than for superficial dulness.*

From what has been said above, it is clear how impossible it is to demonstrate the finer results of percussion to listeners at a distance. A percussion strong enough for this purpose furnishes most incorrect results, and is admissible only for demonstrating quite coarse differences.

Weil's explanation is now apparently generally accepted—viz., that the *deep dulness* of organs depends only upon the volume of the air-containing part which is set in vibration. It was formerly believed that the solid organs themselves not only produced a slight tone, but were also capable of partially dulling the loud resonant tone of the neighboring air-containing organs. According to this hypothesis the deep dulness depended upon the dulling influence of the solid organs in the neighborhood of those filled with air. Weil has experimentally proved that such dulling influence does not exist.

The necessary strength of the percussion stroke naturally depends not only upon the nature and the position of the surrounding organs, but upon the thickness of the body-wall covering. For example, the inferior lung boundary can ordinarily be easily distinguished from the liver by very light percussion, but not in very fat or edematous patients, because then the acoustic sphere of action of the percussion does not penetrate beyond the thick layer of the thoracic wall. In such cases only very vigorous percussion can bring out a clear, plain lung tone, and at best the superficial as well as the deeper boundaries are very difficult and sometimes impossible to map out.

If we only percuss lightly enough and with slight plexímeter pressure *superficial dulness* is frequently quite intense. For this reason it is often called *absolute dulness*. On the other hand, deep dulness is never complete or absolute, and hence is called *relative dulness*.

The designations *absolute* and *relative dulness* should be avoided in topographic percussion, because in mapping out organs it is much more important to know whether a dulness is obtained by deep or by superficial percussion than whether it is more or less intense. Despite light percussion, superficial dulness will not be absolute if the air-containing surroundings are set in vibration.

It is easier to teach practically the other essentials to the technic of topographic percussion than to describe them. In mapping out an organ the beginner should compare the note in two places, one of which plainly lies outside and the other inside the edge of the organ to be defined. He should then percuss between these two places, gradually

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advancing the pleximeter parallel to the edge to be determined until one point of the boundary is determined and marked upon the skin with a pencil. In a similar way other points of the boundary are marked, and finally all are joined by a line which will correspond pretty accurately to the edge of the organ. Such a line can be represented schematically (as described above) upon a chart.

For rapid and comprehensive registering of the results of percussion we use in the Bern Clinic different colored pencils. We represent the superficial dulness obtained by light percussion by blue; the deep dulness obtained by strong percussion by red. The intensity of the dulness is expressed by varying the intensity of the color. A dulness appreciable both to deep and superficial percussion is represented by a mixed red and blue color. Even color tones and evenly mixed colors are easily made by using very soft pastel pencils and a leather stump. In order to distinguish the boundaries of organs which are palpable from those which we obtain by percussion, the former are denoted by a continuous black line. This method of representation is followed throughout this book.

A comprehension of the topographic results of percussion naturally presupposes an exact knowledge of normal visceral topography. Many anatomic facts relative to the position of organs do not, of course, apply accurately to such relations in the living body, because the position of the organs is markedly influenced by the breathing, and because even the median vital position of the thorax and of the diaphragm is quite different from that in the cadaver. This is especially noticeable in the position of the lung edges. In the first place the lung boundaries in the cadaver assume an entirely individual equilibrium, on account of the elastic relations of the thorax and of the lungs, the rigidity of the respiratory muscles, etc. This is, or at least may be, essentially different from the median position in the living. The cadaveric position of the lung edges is generally that of exaggerated expiration. Frozen sections, inserting needles through the lung edges before opening the thorax, or cutting windows in the thoracic wall without disturbing the costal pleura, furnish correct results. All other anatomic testimony is, in the nature of things, worthless, because as soon as the thorax is opened the lungs are retracted, tending to assume a position of equilibrium. Topographic percussion is therefore a valuable aid to anatomists for determining the anatomic position of organ boundaries during life. The plates illustrating the position of the viscera in this book are taken from the classic plates of Luschka and the models of Ferber.[1] They have been drawn especially for clinical purposes to show the median position of the movable organs, and with particular attention to the sources of error mentioned above. The reader is recommended to study Ferber's models.

Symington's[2] cross-sections or Dwight's *Frozen Sections of a Child* illustrate the topographic relations in the child.

[1] *Situsphantom der Organe der Brust und oberen Bauchgegend*, Bonn, Max Cohen & Son, 1877.

[2] *Topographic Anatomy of the Child*, Edinburgh, 1887.

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Figs. 74–76 reproduce outlines from Luschka's plates. To orient the positions of the borders to the skeletal points, the ribs and the vertebral spines are counted. Here it is well to remember: The second rib is usually marked upon the sternum by the angle of Ludwig; it is ordinarily the highest rib which we can plainly grasp between the fingers from in front. Generally the first rib is almost hidden beneath the clav-

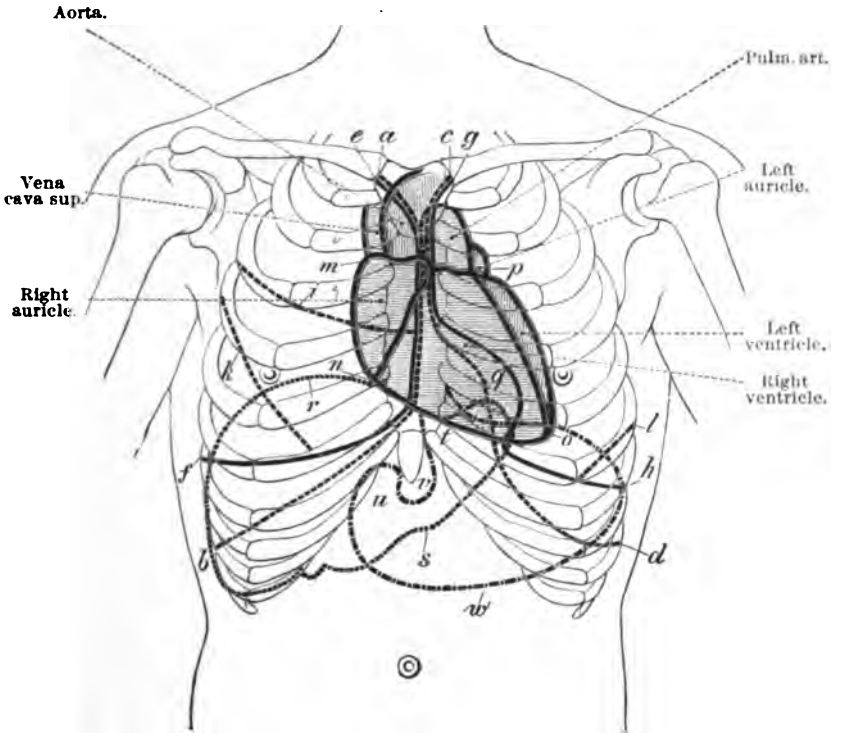

FIG. 74.—Position of the thoracic and upper abdominal viscera from in front: *a b*, Boundary of right pleural cavity; *c d*, boundary of left pleural cavity; *e f*, edge of right lung; *g h*, edge of left lung; *i*, upper incisura lobularis (right lung); *k*, lower incisura lobularis (right lung); *l*, left incisura lobularis; *m n* right, *n o* lower, *p o* left border of heart; *q*, mediastinal sinus situated between the pleural boundaries and the incisura cardiaca of the anterior edge of the left lung; *r*, highest point of the liver, overlapped by lung; *s*, lower edge of liver; *t* pars cardiaca, *u* pars pylorica, *v* small curvature, *w* large curvature, of the stomach (modified from Luschka-Weil).

icle. The lowest of the ribs which is directly attached to the sternum (true ribs) is the seventh. The first of the "floating ribs"—*i. e.*, those with their tips freely suspended—is the eleventh. In counting the vertebral spines we generally begin with the seventh cervical. Its prominence when the head is bent forward (vertebra prominens) makes it easy to recognize. Where three vertebral spines are quite prominent in this region, the

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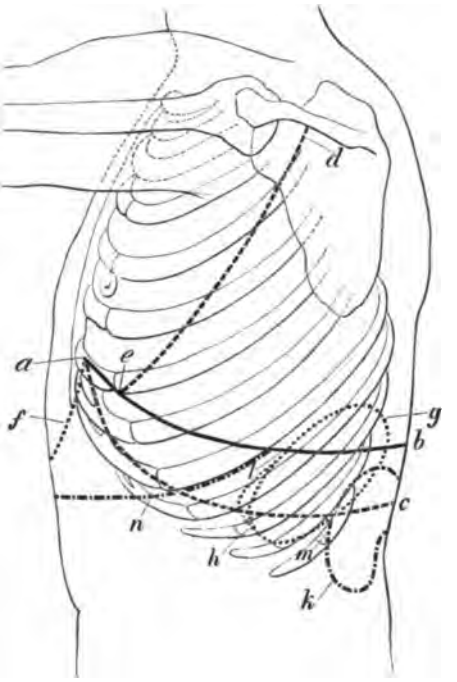
seventh is usually in the middle. Where the seventh cannot be positively determined, the vertebra should be counted from below—*i. e.*, from the fifth lumbar vertebra upward. The lower angle of the scapula, with the arms hanging, ordinarily corresponds to the seventh rib and the seventh dorsal vertebra. The base of the xiphoid cartilage can be



Pulmonary border.
Pleural boundary and incisuræ interlobulares.
Stomach and kidney.
Liver and spleen.

FIG. 75.—Position of the thoracic and upper abdominal viscera from the left side: *a b*, Lower edge of the left lung; *a c*, lower boundary of the pleural cavity; *d e*, incisura interlobularis; *f*, edge of the left lobe of the liver; *g* posterior, *h* anterior end of the spleen in its oval form, in the rhomboid form it pushes itself in between the anterior (*g l*) and the posterior (*g h*) edge of the piece (*l h*); *k*, convex edge of the left kidney; *l*, splenic lung angle; *m*, splenic kidney angle; *n*, the part of the greater curvature of a moderately distended stomach lying against the wall (from Luschka-Weil).

utilized in topography, but its tip varies considerably in length and position, and is therefore unreliable.

Besides the skeletal parts we make use of so-called orientation lines. These are vertical lines which intersect the ribs at certain angles. They are the following (see Figs. 70, 71, and 72):

1. The anterior and the posterior median line.

2. The right and the left sternal line—drawn vertically through the edges of the sternum.

3. The right and the left parasternal line—drawn halfway between the sternal border and the nipple.

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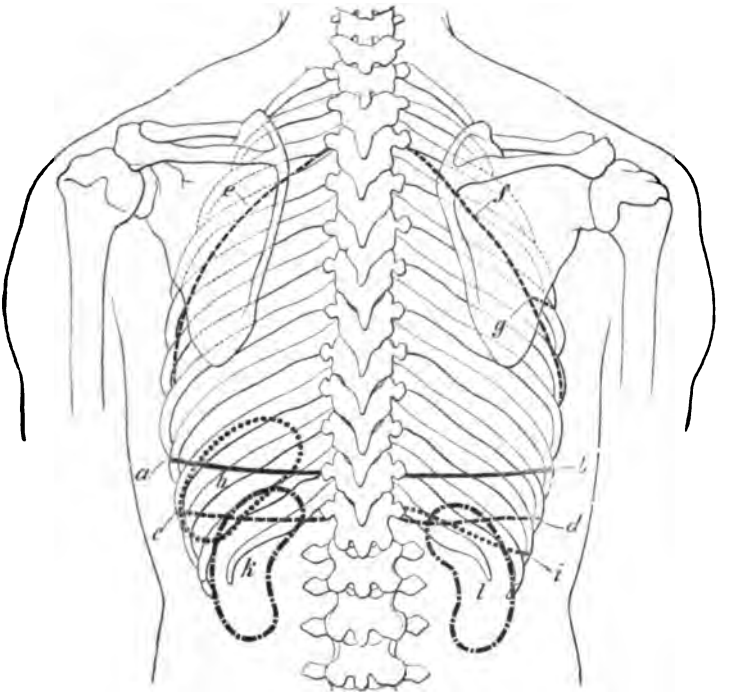
4. The right and the left mammillary line (or nipple lines)—drawn perpendicularly through the nipple.

5. The middle, the anterior, and the posterior axillary line—drawn through the middle, the anterior, and the posterior edge of the axilla.

6. The right and the left scapular line—drawn perpendicularly through the inferior angle of the scapula, with the arm hanging down.

The position of the mammillary line is inconstant both in men and in women. The so-called *midclavicular line*, which is dropped perpendicularly from the middle of the clavicle, is therefore a more accurate landmark. A horizontal mammillary line is sometimes employed; that is, a horizontal line on the surface of the thorax, drawn through the nipples. Its position is, of course, influenced by the height of the nipples. In men these are usually found to be between the fourth and fifth or upon one of these ribs (rarely between the fifth and the sixth ribs), about 10 cm. distant from the lower thoracic edge, and about 16 cm. from the lower edge of the clavicle.



Lung borders.
Pleural boundaries and incisuræ interlobulares.
Kidney.
Liver and spleen.

FIG. 76.—Position of the viscera from behind: *a b*, Lower lung border; *c d*, lower pleural boundary; *e* and *f*, incisuræ interlobulares; at (*g*) on the right side it is divided into the sulcus interlobularis dextra superior and inferior; *h*, spleen; *i*, lower liver edge; *k*, left kidney; *l*, right kidney (from Luschka-Weil).

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The terms used in topographic anatomy are usually serviceable in topographic percussion—such as supra- and infraclavicular grooves, supra- and infraspinatous fossæ, interscapular space, epigastrium, hypochondrium, mesogastrium, hypogastrium, etc. (see Figs. 77 and 78).

TOPOGRAPHIC PERCUSSION OF THE LUNGS.

NORMAL LUNG BORDERS.

The boundaries of the lungs move normally with respiration; hence we must distinguish, on the one hand, an *expiratory*, and, on the other, an *inspiratory* position. This distinction is especially important for

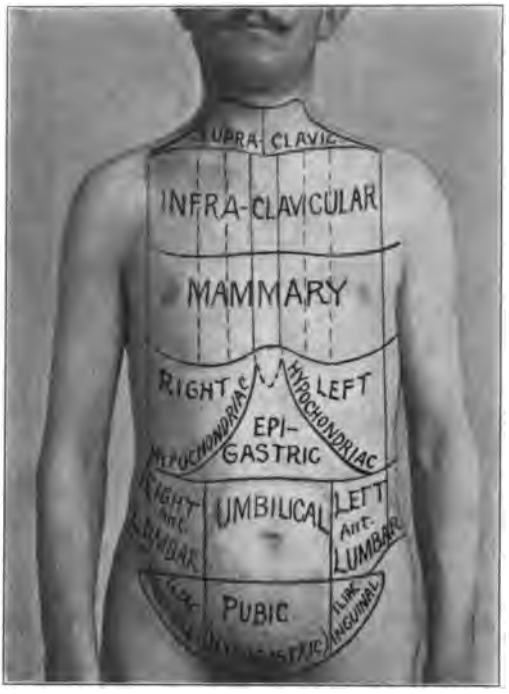

FIG. 77.—Topographic areas of trunk—anterior view.

determining the mobility of the lung edges. In general, the median position of the lung borders when the patient breathes superficially is sufficient for most purposes. The excursions of the lungs are then hardly greater than the limits of error which are inherent in percussion. The boundaries designated as normal correspond to such a median position of the lung edges.

We usually determine the inferior boundaries of the patient's right lung (the lung-liver boundary) anteriorly while he is lying down; posteriorly, while he is sitting or standing. Such a boundary line intersects the parasternal and midclavicular lines at the upper edge of the sixth rib; the axillary lines, at the eighth to ninth ribs; the scapular

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FIG. 78.—Topographic areas of trunk—posterior view.

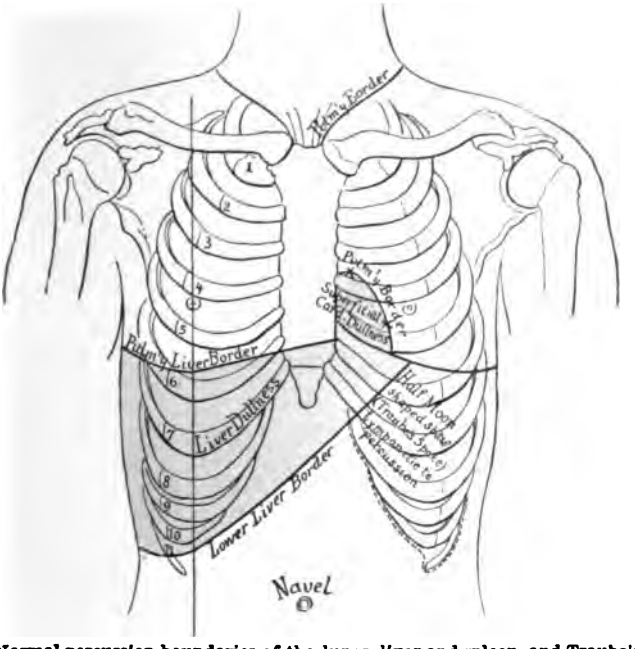

FIG. 79.—Normal percussion boundaries of the lungs, liver and spleen, and Traube's space—anterior view.

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lines, at the tenth rib; the posterior median line, at the eleventh vertebral spine. The border, therefore, runs very nearly horizontal (see Figs. 79 and 80).

The precordial edge of the left lung forms a segment, within which the heart lies directly against the thoracic wall. This segment corresponds to the so-called *superficial cardiac dulness* (Figs. 79 and 85). The border of the lung bounding this area lies above at the left edge of the sternum, upon the fourth rib, and runs from there horizontally to the left;

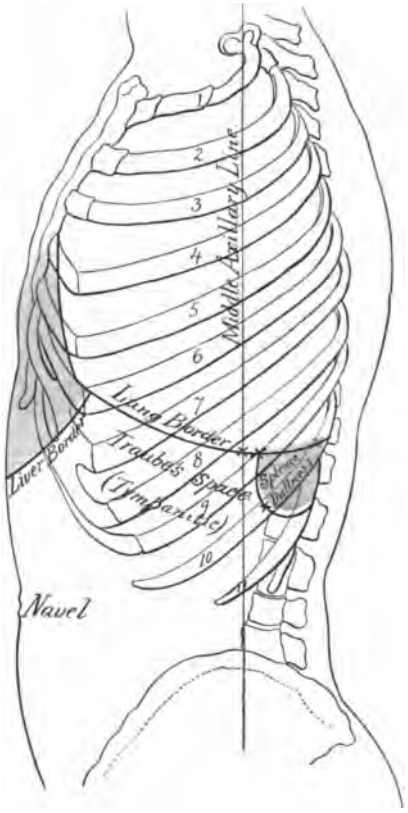

FIG. 80.—Percussion boundaries of the lung, liver and spleen, and Traube's space—from the left side.

at the parasternal line it curves downward to the level of the sixth rib. and then takes the same course as the inferior border of the right lung. For practical purposes we may assume that the inferior lung edge, with the exception of this segment over the heart, follows practically a horizontal course upon both sides. The edge of the left lung can be easily and accurately differentiated by the superficial cardiac dulness; but farther to the left the loud, resonating stomach is apt to confuse the percussion. From the axillary line backward the defining of the edge of the lung becomes easier again, because the spleen, the powerful

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muscular masses of the quadratus lumborum, and the lumbar limb of the diaphragm lie below the lung.

The anterior lung borders run almost vertically beneath the sternum (Fig. 74). It is impossible to percuss them, because only a small space exists between them, and because an exact localization of the percussion stroke upon the sternum is very difficult. The bone vibrates to percussion more or less as a whole, like a great pleximeter, and transmits the vibration widely over the surface. The superficial cardiac dulness can be percussed accurately only when a considerable part of that bone overlies or is bounded by a dull-sounding tissue (see Figs. 88, 91). For this reason the right border of the superficial cardiac dulness ordi-

FIG 81.—Apical percussion: Lines above clavicle at normal height.

narily corresponds to the left edge of the sternum, and hence has little diagnostic importance.

The apices of the lungs form slightly voluminous cones covered by very thick layers of muscle, which render the upper pulmonary boundaries much more difficult to determine as linear projections than the lower. Moreover, the trachea lies in such close proximity to the lung apices that percussion is very liable to set this in vibration. Hence the difficulty of properly percussing the apices. Figs. 79 and 81 represent the boundary lines of the lung apices in individuals who are neither too muscular nor too fat. The highest point of the upper lung border lies from 3 to 5 cm. above the clavicle.

[In a recent paper[1] Dr. R. W. Philip, Physician to the Victoria Hospital for Consumption, has called attention to the significance of

[1] *Practitioner*, London, vol. xvii., 1903.

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the supraclavicular triangle in relation to early apical changes. He believes, from a large number of observations, that the mean of lung resonance above the clavicle is at least 1½ in. (4 cm.); that frequently it is over 2 in. (5¼ cm.).

Assuming that lung resonance may be determined in the healthy subject over an area of three fingers' breadth above the clavicle, Dr. Philip believes that apical changes are often overlooked by limiting percussion to a single finger's breadth above the clavicle, a method

FIG. 82.—Apical percussion: Line above left clavicle 1½ cm. below normal; left apex dull (New York City Hospital).

which obtains with many examiners. The editors' experience corresponds with Dr. Philip's.

The accompanying photographs (Figs. 81–83) illustrate a normal extent of pulmonary resonance above both clavicles; a decided limitation of the resonance above the left clavicle in a case of pulmonary tuberculosis at the left apex; and the method of percussing the apices from behind. We have intentionally omitted any reference to so-called "tidal percussion of the apices." Its value has not been settled beyond dispute.—ED.]

The lung borders vary somewhat according to the age of the patient. For example, in old people the lung-liver boundary is situated somewhat lower (about one intercostal space). The superficial cardiac dulness is often somewhat diminished and situated about one intercostal space lower than in young adults. This change depends upon the diminished elasticity of the senile lung. Many authors denote this change as senile emphysema, provided nothing else abnormal is detected. The writer doubts if such nomenclature is correct, and has been unable

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to determine a higher level of the pulmonary edge in children than in perfectly healthy adults.[1]

ACTIVE AND PASSIVE MOBILITY OF THE LUNG BORDERS UNDER NORMAL AND UNDER PATHOLOGIC CONDITIONS.

Vigorous respiration will depress the lung borders several centimeters during inspiration and elevate them the same distance during expiration (*active mobility*). Percussion will very plainly demonstrate this. In the axillary line the extreme positions of the lung border may reach 4 cm.

FIG. 83.—Method of apical percussion.

above and below the mean, so that the total excursion may be as much as 8 cm. Litten's diaphragm phenomenon (p. 78 *et seq.*) is the visible expression of such excursions. Deep inspiration may almost or entirely obliterate the superficial cardiac dulness.

Change in a patient's position will demonstrate a *passive mobility* of the lungs. Changing from the dorsal decubitus to the erect posture may elevate the lung-liver boundary (or very rarely depress it). In some cases no change is noted. This variable result probably depends upon the preponderance of one of two opposing factors which influence the position of the diaphragm: 1, the weight of the liver; and 2, the increased abdominal pressure due to the contraction of the abdominal muscles in the upright posture. If the abdominal walls are tense enough to contract vigorously in sitting and standing, a slightly higher position of the inferior lung boundary seems to be the rule, because the intra-

[1] Sahli, *Die topographische Percussion im Kindesalter*, Bern, Dalp'sche Buchhandlung (now Schmid, Franche and Cie.), 1881.

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abdominal pressure will be increased. Whereas if the abdominal walls are relaxed—*e. g.*, with the characteristic pendulous abdomen—the opposite effect will be observed, because the weight of the liver will depress the diaphragm. Changing from the dorsal decubitus to a lateral posture will depress the pulmonary border of the uppermost lung about 3 or 4 cm. at the axillary lines. A deep inspiration while this position is retained may bring the border about 9 cm. lower than in the dorsal decubitus with median respiratory position; and with a full expiration the lung border may under some circumstances make an excursion of as much as 13 cm. By means of all these different types and degrees of lung mobility, it is generally possible to demonstrate the clinically important sign of *diminished or absent lung mobility*. The mobility of the lung border is diminished in (1) *pulmonary emphysema* and in (2) *partial consolidations of the lung*. Although the percussion note may not be noticeably dulled if these consolidations are scattered, such a condition may be suspected from the immobility of the border. (3) *Firm pleuritic adhesions* between pulmonary and costal pleura also prevent mobility.

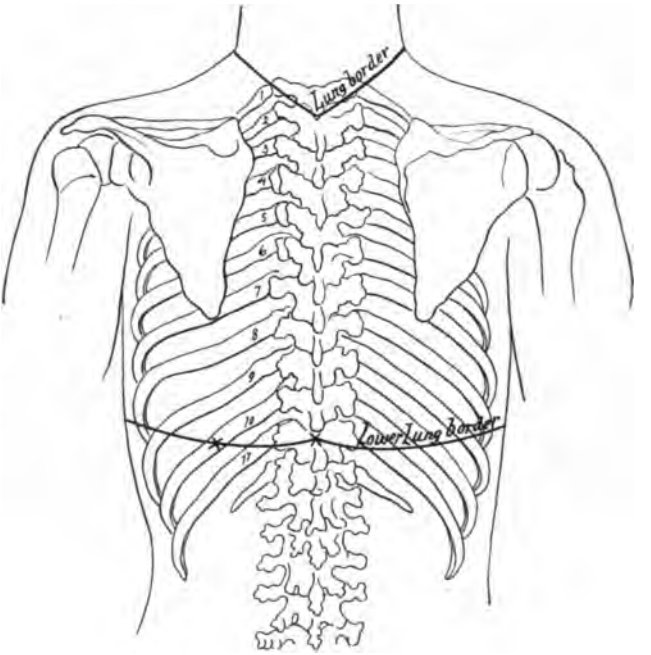

FIG. 84.—Normal percussion boundaries—from behind.

Some examiners attempt to demonstrate pleuritic adhesions of the lung edge by percussing below the border determined during quiet breathing while the patient breathes deeply. If the loudness of the note is much increased with inspiration, they then claim that the lung edges are freely mobile. This method of examination, according to the author's experience, often causes error. Even if the lung is quite adhe-

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rent, the intensity of the note beneath the lung border is almost certain to increase during inspiration. Such an increase does not necessarily prove a descent of the boundary, but merely suggests a thickening or an inflation of the lung edge. In other words, a greater accumulation of air at or near the pulmonary edge influences the note below it, because even with the lightest percussion it is not possible to absolutely localize the percussion stroke.

A much better method for demonstrating mobility of the pulmonary border is to determine the boundary in the position of extreme inspiration while the patient holds his breath, mark it on the chest, and then do the same during extreme expiration.

ABNORMAL POSITION OF THE LUNG BOUNDARIES.

Under pathologic conditions the lung boundaries may be extended as well as contracted.

Extension of the lung boundaries occurs in *emphysema*, where the lung-liver boundary may reach down to the eighth rib in the right midclavicular line, to the ninth or tenth rib in the axillary line, and to the twelfth vertebra behind in the posterior median line; in fact, quite to the inferior limit of the thorax. The emphysematous increase may sometimes be plainly demonstrated even at the lung apex, and over the superficial cardiac dulness, which may be either entirely or almost obliterated. Both the active and the passive mobility of the borders in emphysema are diminished on account of the permanent inspiratory position of the diaphragm and a certain fixity of the lung so characteristic of the disease. Emphysema ordinarily is developed upon both sides, and, as a rule, quite uniformly; but a partial emphysema does occur (perhaps incorrectly called *vicarious emphysema*), in which the changes are localized at the lung borders. Even in the common type of pulmonary emphysema the pulmonary distention is not always uniform. Frequently percussion shows that the emphysema is limited to the region over the heart, whereas the inferior lung border is not any lower than normal. This is often seen in fat or dropsical individuals, apparently because the increased abdominal contents crowd the diaphragm upward.

In a similar manner the pulmonary boundaries are also extended in attacks of *bronchial asthma* and in *obstructive bronchitis* because there is a greater resistance to the emptying than to the filling of the lungs. For analogous reasons, when a bronchus is narrowed the affected pulmonary lobe is dilated.

Certain *cardiac affections*, particularly mitral lesions, lead to pulmonary dilatation, the lungs being permanently engorged with blood and in the condition of so-called cardiac lung rigidity; brown induration is usually present as well. The lungs in these cases resemble those of emphysema, since they are dilated, their elasticity is partly lost, and they make but slight excursions.

The lung borders in *enteroptosis* (p. 192) are nearly always depressed.

Retraction of the lung border results from the crowding of the lung

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edges by the neighboring parts. 1. The diaphragm will be pushed upward by all conditions which increase the intra-abdominal pressure—*e. g., meteorism, ascites, abdominal tumors* (especially if situated at the convexity of the liver). Negative intrathoracic pressure will therefore be diminished; so the lungs must be retracted, not only upward, but concentrically in all directions, from in front backward and toward the hilus, even enough to expose the heart to a considerable extent. 2. An enlarged heart (or a pericardium filled with fluid) can also crowd the lungs aside enough to increase the superficial cardiac dulness. (See Heart Percussion.) If such a crowding is very marked, the resulting diminution of negative intrathoracic pressure will elevate the inferior lung borders. 3. All processes associated with a pulmonary shrinking may occasion a retraction of the lung boundaries—*e. g.*, the *chronic forms of tuberculosis*, which produce a connective-tissue retraction of the lungs; and cases of *pleurisy* in which, after the absorption of the exudate, the expansion of the compressed portion of the lung is prevented by the formation of a firm connective-tissue coating. The shrinking usually proceeds concentrically in such conditions, so that the lungs are often retracted on all sides; that is, as much over the heart as at the inferior borders, and sometimes even at the upper borders, toward the hilus. Chronic tuberculosis frequently leads to a retraction of the pulmonary apex. Therefore the demonstration of a unilateral low position of the superior lung border is of special importance for the early diagnosis of apical tuberculosis. (See Editor's Note, p. 172, and Fig. 81.)

We must remember that the normal positions mentioned above are only averages, and that abnormally long or short chests would naturally modify the position of the lung borders in relation to the ribs, while the condition could in no way be considered pathologic. Errors in this respect, especially in relation to the diagnosis of emphysema, frequently occur in practice. They cannot be avoided by definite rules, but only by practical experience and by the development of a geometric vision.

TOPOGRAPHIC PERCUSSION OF THE HEART.

NORMAL SUPERFICIAL AND NORMAL DEEP CARDIAC DULNESS.

Superficial cardiac dulness is the dulled area which corresponds to the segment of the left lung about the heart (Figs. 79 and 85). Its extent really tells more about the position of the lung edge than about the size of the heart. Nevertheless, if the heart is enlarged or if the pericardium becomes distended with fluid, the edges of the lung will be pushed back and the superficial cardiac dulness increased. Certain cautions are necessary to prevent mistakes in estimating the size of the heart or pericardium from the extent of this superficial cardiac dulness. For example, despite an enlarged heart, the superficial cardiac dulness is not necessarily increased in emphysema, even when the lung edges in the neighborhood of the heart are fixed by pleuritic adhesions. The deep dulness is more important in estimating the size of the heart and the contour of the pericardium. The deep cardiac dulness will never

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be very intense, but is always modified (a so-called relative dulness). The beginner often finds it very difficult to determine. The superficial cardiac dulness, on the contrary, is frequently absolute, and therefore easier for the beginner's ear to appreciate. Both varieties of dulness should therefore be mapped out upon the chest. The superficial often confirms the results of the deep percussion.

The form and size of the superficial dulness, also called the small cardiac dulness, have already been described in the section on Topographic Percussion of the Lungs (see Fig. 79, p. 169 *et seq.*). Fig. 85 represents the relations of the superficial and the deep or great cardiac dulness in the average healthy adult. The boundaries of the latter run from

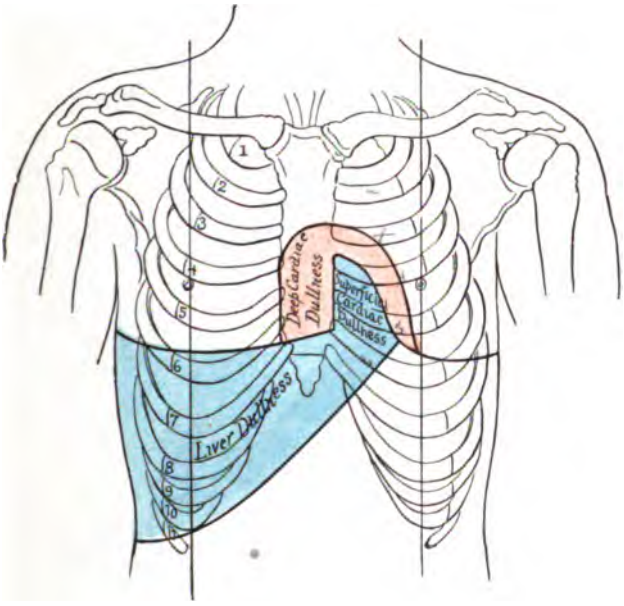

FIG. 85.—Superficial and deep cardiac dulness under normal conditions.

the upper edge of the third left rib nearly parallel to the border of the superficial cardiac dulness, bend toward the left in a bow-shape, with the convexity outward, become perpendicular slightly inside the mid-clavicular line, and end near this point at the apex beat. The heart is bounded below by the liver, so that the deep dulness, like the superficial, merges into the hepatic dulness and cannot be differentiated from it. Where the liver is covered by intestines filled with air, or where it is pushed upward to the right, superficial percussion will generally evoke a loud tympanitic tone just below the heart. Most authors limit the right boundary of the deep dulness at the left sternal border; nevertheless, according to the writer's experience, the majority of healthy adults show a slight dulness up to the right sternal border (Fig. 85). But in many cases the whole extent of the sternum furnishes such a

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loud tone that the deep cardiac dulness is really limited by the left sternal border. (See Topographic Percussion of the Lungs.) These individual peculiarities depend upon the vibration of the sternum, upon the thickness of the layer of lung covering the heart, etc. In determining the upper borders of the deep dulness, the fact that the sternum transmits the blow so deeply necessitates a light percussion.

Both the superficial and the deep dulness are smaller in the aged than in younger adults, because the lungs of old people cover the heart more extensively. In children both varieties of dulness are, on the contrary, larger, because the amount of lung tissue which covers the heart is thinner, and so the sphere of acoustic action of the percussion blow reaches the solid organ earlier. This distinction is shown by comparing Figs. 73 and 86.

Fig. 73 pictures the relation of the acoustic sphere of action of the percussion blow in adults; Fig. 86, in children. In the latter it is plain that the deep dulness will sometimes be larger than the organ itself; whereas in adults, and even more noticeably in older people, percussion can only include a portion of the entire size of the heart. Long

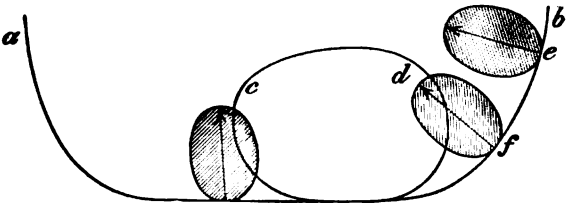

FIG. 86.—Relations of the size of the acoustic sphere of the action of the percussion blow in children (see Fig. 73).

practice will prevent mistakes, but even then we must employ the other examination methods, such as palpation of the apex beat.

Variations of the thoracic dimensions in different individuals make the conclusions to be drawn from deep cardiac percussion still more doubtful. The heart boundaries are ordinarily mapped out in accordance with the orienting lines of the body—*e. g.*, the position of the left border of the heart in reference to the mammillary line. This sometimes leads to erroneous conclusions. Although, as a rule, the left border of the deep cardiac dulness lies somewhat inside the mammillary line, it is self-evident that if the mammillary line is pushed inward even a normal heart would reach outside. Even the midclavicular line is not absolutely constant, because the length of the clavicle varies, and with it the breadth of the thorax. If the sternum is broad, a dulness beyond the right sternal edge would be more significant than if the sternum is narrow. Hence the importance of the absolute size of the cardiac dulness. At the Bern Clinic we always measure its horizontal diameter in the third and fourth intercostal spaces, and the distance of the left and right margins of dulness from the anterior median line. Riess[1] has

[1] *Zeit. f. klin. Med.*, 1888, vol. xiv., p. 12.

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estimated the normal figures from averages upon a large number of measurements in medium-sized healthy adults, as follows:

Distance of the right border of the deep cardiac dulness from the anterior median line: Third intercostal space 2¾ cm.; fourth intercostal space 3¾ cm.

Distance of the left border of the deep cardiac dulness from the median line: Third intercostal space 4¾ cm.; fourth intercostal space 7½ cm.

Total width of the deep cardiac dulness: Third intercostal space 7½ cm.; fourth intercostal space 11¼ cm.

These figures seem to the writer rather too small.

Cardiac percussion can sometimes be simplified by directing the patient to bend forward or to take a deep inspiration.

Before an attempt to estimate the deep cardiac dulness in women with prominent breasts, the patient or a nurse must first push the left mamma upward and to the left.

ACTIVE AND PASSIVE MOBILITY OF THE SUPERFICIAL AND DEEP CARDIAC DULNESS.

The heart borders, like those of the lungs, change their position actively with respiration and passively with change of the patient's position. Active mobility concerns only the respiratory overlapping of the heart by the lung edges, whereas passive mobility influences the position of the heart as well as of the lung borders.

The boundaries described above are estimated during quiet breathing in the dorsal decubitus. Deep inspiration diminishes the size of both superficial and deep cardiac dulness more or less decidedly. Forced expiration produces the opposite effect, and in rare cases may bring the superficial dulness to the right of the right sternal edge, the right edge of the lung retreating so far. Therefore percussion during forced expiration (without exerting abdominal pressure) will sometimes reveal the true size of a heart which is extensively covered by the lungs.

In the left lateral posture the heart falls to the left and displaces the anterior edge of the left lung. The anterior edge of the right lung is only exceptionally found beyond the left sternal edge, because its excursion is normally limited by the line of the pleura beneath the sternum (see Fig. 74). Therefore in left lateral positions both the superficial and the deep cardiac dulness are increased to the left.

In right lateral positions the opposite displacement occurs. Both the deep and the superficial dulness will reach beyond the right of the sternum, while the superficial dulness to the left of the sternum may entirely disappear.

Changing from the recumbent to the sitting posture does not produce any constant change in the form or size of the cardiac dulness. Both the superficial and the deep dulness may seem somewhat more intense in the upright posture. If the patient bends forward both are increased, because the heart pushes the lungs aside, approximating itself more completely to the anterior thoracic wall. Where emphysema or thick

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thoracic walls obscure the percussion in the recumbent posture, sitting up and bending forward may be helpful. To prevent mistakes the patient must be careful not to bend sidewise or twist his back, for we must remember that in such a position a normal heart will furnish a broader and more intense dulness.

The absolute measures of the mobility of the superficial and the deep cardiac dulness vary so much with the individual that the figures would be superfluous.

PATHOLOGIC CHANGES IN THE SUPERFICIAL AND DEEP CARDIAC DULNESS.

Diminution of the Superficial and Deep Cardiac Dulness.

In *advanced emphysema*, in *left-sided pneumothorax*, in *pneumocardia*, and in *precordial emphysema* both the superficial and the deep cardiac dulness may be either diminished or entirely disappear. The cardiac atrophy which is sometimes observed at the autopsy table is too slight to be recognized by percussion. *Emphysema* also causes an especially low position of the cardiac dulness, on account of the deep position of the diaphragm. Frequently no deep cardiac dulness can be made out, and the superficial, if present, may appear only at the fifth or even the sixth rib. In *left-sided pneumothorax* we should expect that at least a part of the superficial cardiac dulness would persist, because the normal division line of the pleuræ, which would divide the superficial cardiac dulness in half, shifts (Fig. 74). As a matter of fact, however, left pneumothorax almost always dislocates the heart, the division line, and with it the mediastinum, so far to the right that the superficial cardiac dulness to the left of the sternum may entirely disappear. In *right-sided pneumothorax* (Fig. 99) the air resonance may overlap the left edge of the sternum, in consequence of the mediastinum being pushed to the left, so that the superficial cardiac dulness will appear to be narrowed from the right. The demonstration of cardiac dislocation (p. 188) will suggest and explain this condition. Percussion elicits an abnormally resonant metallic note (often tympanitic) over the area of cardiac dulness in *pneumopericardium* and *precordial emphysema*. The pericardial sac in the former ordinarily contains fluid as well as air; this becomes evident when the patient sits up, for the lower portion of the abnormally resonant area becomes dull, the fluid following the laws of gravity. (The auscultatory signs will be mentioned later.)

With marked gaseous distention of the intestine or of the stomach, even careful percussion may elicit a tympanitic note over the area of superficial cardiac dulness, because the vibrations are transmitted to the abdominal contents. But very gentle percussion, in which the pleximeter is applied only by its own weight, and perhaps best with the patient bending forward, will probably enable us to demonstrate the superficial cardiac dulness, and so to differentiate tympanites from the overlapping of the heart by air-containing tissue. A deep cardiac dulness in such cases is difficult, if not impossible, to obtain.

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Enlargement of the Cardiac Dulness from Abnormalities of the Lung Borders.—Both the superficial and the deep cardiac dulness will be increased if the heart pushes back the anterior lung boundary, or if some anatomic process in the latter (infiltration or atelectasis) adds a dull tone of its own to the cardiac dulness. The increase of cardiac dulness does not then depend upon any alteration of the heart's size. Such conditions can be properly interpreted only by carefully considering the entire clinical picture, and by making use of other methods of examination. The most frequent examples are *pulmonary contraction* and the *concentric retraction of the lung*, or an upward dislocation of the diaphragm from marked ascites, meteorism, and the like (see pp. 175 and 176).

Increase of the Cardiac Dulness from Actual Increase of the Size of the Heart or of the Pericardial Contents.—Here the superficial and the deep cardiac dulness are generally increased

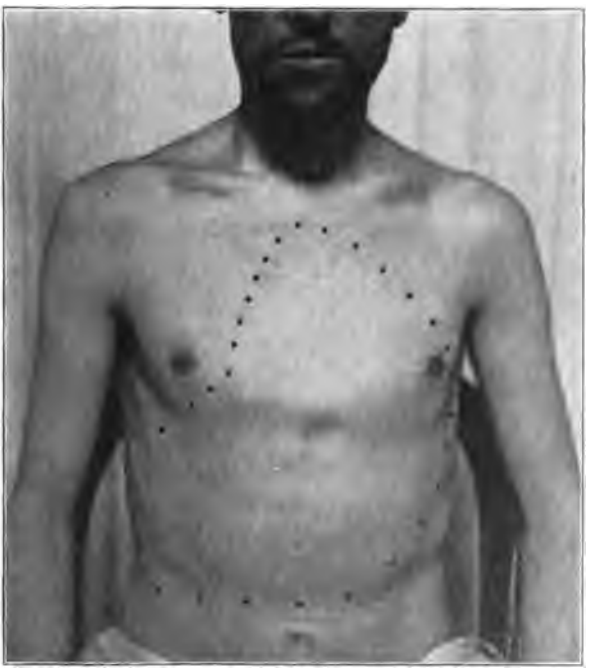

FIG. 87.—Precordial bulging: Enlarged heart and liver; double mitral lesion; percussion boundaries outlined by dotted lines. This picture does not bring out the marked precordial bulging which was very evident in looking at the patient. The width of the cardiac dulness at the nipple line was 21 cm. (New York City Hospital).

correspondingly unless an emphysema or adhesions of the pulmonary border complicate the picture (p. 176). In the event of such a complication the superficial cardiac dulness may be much less enlarged than the deep; both may remain small; or even neither may be increased,

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on account of the marked covering of the heart. In this way even considerable cardiac enlargement might escape clinical demonstration.

The superficial cardiac dulness is very important in demonstrating cardiac enlargement, because the beginner finds it much easier to estimate than the deep dulness; because there is less chance for a subjective variation; and because in decided cardiac enlargement or increase of pericardial contents on account of the considerable lung retraction the entire cardiac dulness frequently becomes superficial.

Actual Enlargement of the Heart.—The heart is pathologically enlarged both by hypertrophy of its walls and by dilatation of its cavities. The extent of the former enlargement must, of course, be limited. The latter will be much greater and more easily recognized. If, while the chambers of a heart retain their normal size, its walls increase about 0.5 cm. in thickness from simple hypertrophy, this would produce a difference of only 1 cm. in the width of the heart. Now, as a matter of fact, we cannot percuss accurately enough to appreciate 1 or 2 cm. increase in cardiac size, nor do we practically ever see post mortem as much hypertrophy as 0.5 cm. unless complicated with dilatation. Evidently, then, any enlargement of cardiac dulness which can be demonstrated by percussion must depend upon the dilatation of the cavities, whether there is any coexisting hypertrophy or not.

An appreciable enlargement of cardiac dulness due to a pure hypertrophy of the heart—*i. e.*, one unassociated with dilatation—is, however, sometimes observed in very rare cases, particularly in chronic nephritis. In such cases a diagnosis of pure hypertrophy must be supported by other examination methods (increased force of apex beat, high-tension pulse, accentuated second tone (see below)).

The cardiac dulness may be increased in all directions or only in one direction. We naturally suppose a dislocation of the left border of cardiac dulness to the left to depend upon a dilatation of the left ventricle; a dislocation of the right border to the right, upon a dilatation of the right ventricle; an increase upward, upon a dilatation of the auricles or of the great vessels. But the autopsy table has shown so many exceptions that these conclusions are by no means sure—*e. g.*, marked dilatation of the left ventricle may increase the cardiac dulness upward without there being any dilatation of the auricles or of the great vessels; because, if the ventricle is dilated, the oblique position of the heart pushes the dulness upward. The clinical findings might very often induce us to assume a dilatation of the left ventricle when the dulness, as a matter of fact, depends only upon a dilatation of the right ventricle, or *vice versâ;* or, again, to assume that only one ventricle is enlarged, whereas the autopsy shows that both share equally in the enlargement. The reason of this is that any dilatation of one chamber of the heart must secondarily dislocate the entire heart. Thus, dilatation of the right ventricle increases the dulness not only to the right, but often to the left, pushing the left ventricle in that direction. The position of the mediastinum is a factor in the difference of pressure at either side—*i. e.*, in the two pleural cavities—so that the actual position of a heart with dilated cavities is a complicated result of its own enlargement and of the

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difference of pressure at the two sides of the mediastinum, which difference is equalized by a dislocation of the mediastinum together with the heart. To make the matter still more complicated, the resistance to cardiac dislocation is by no means equal in all cases. Great differences are caused by the varying resistance of the mediastinum, by the varying amount of depression of the convexity of the diaphragm, and by individual variations of the prolongations of the pericardium upon the great vessel trunks. The existence of such differences is plainly proved by the different dislocations of the heart from causes acting outside of it (pleural exudates).

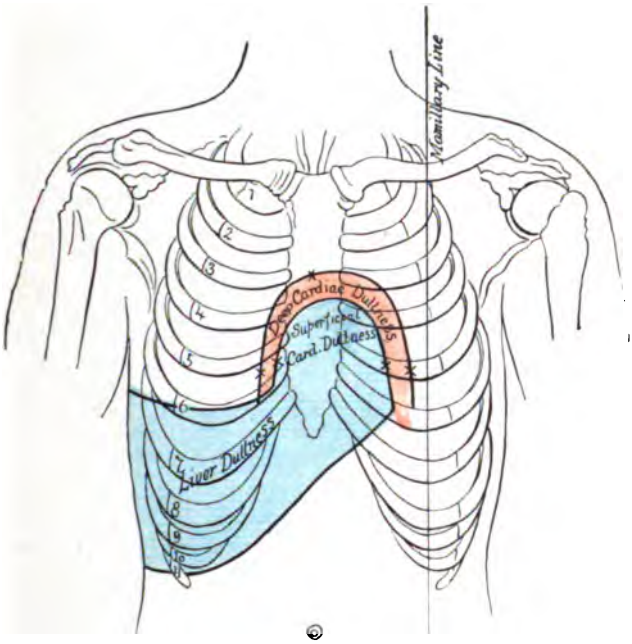

FIG. 88.—Cardiac dulness with dilatation of the right ventricle: Dulness is especially increased to the right.

It is generally more difficult to demonstrate enlargements of the right heart by percussion than those of the left, because, on account of the notch in the left lung, the left heart is more accessible to percussion than the right, which is covered more completely by lung and by the sternum. Besides, the right ventricle (Fig. 74) rests upon the convexity of the diaphragm, so that if that chamber dilates, the heart will have the tendency to find the necessary space in the left thoracic cavity, because the arch of the diaphragm would present too great a hindrance to its enlarging to the right. Hence a moderate dilatation of the right ventricle often produces only a dislocation of the left cardiac boundary; while it requires a very considerable dilatation of the right ventricle to increase the dulness to the right of the sternum. The most familiar example of this is the displacement of the apex beat to the left

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in pure mitral stenosis, where the left ventricle is certainly not dilated. Sometimes, however, the enlargement of the right ventricle (Fig. 88) can be definitely demonstrated to the right.

So many exceptions make it clear that a diagnosis of the enlargement of a certain cavity cannot be determined from the direction of the increase of cardiac dulness without the general clinical picture and without other methods of examination. The position of the apex beat, as we shall see below, is perhaps as helpful as anything. Figs. 88 and 89 show typical examples of the position of cardiac dulness in dilatation of the right and of the left ventricle.

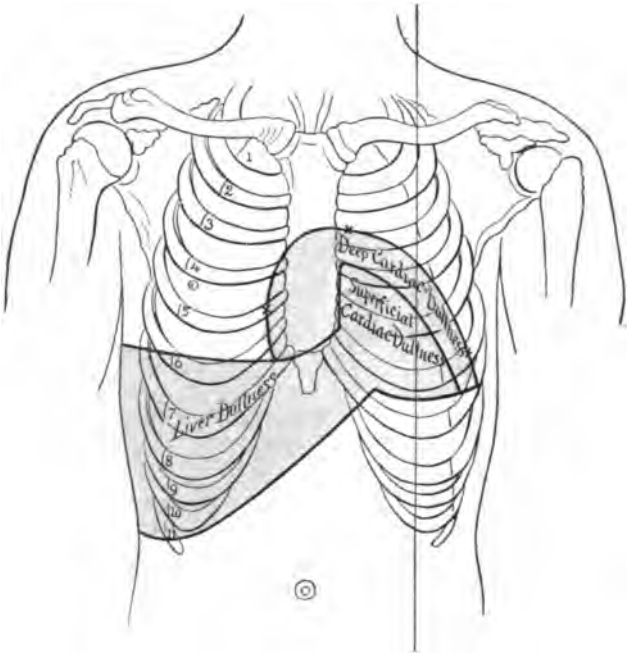

FIG. 89.—Cardiac dulness with dilatation of the left ventricle.

Enlargement of the cardiac dulness upward may be due to dilatation of the ventricles, of the auricles, or, of the great vessels. Percussion will frequently differentiate the latter two from the former; if the increase of the dulness upward is very pronounced (*i. e.*, in proportion to the lateral enlargement), or if it assumes the form of a projection from or an appendage to the ordinary form of cardiac dulness, it is to be referred to dilatation of the auricles or great vessels.

Fig. 90 presents such an example, and illustrates the importance of determining by cardiac percussion the relation of the superficial to the deep dulness in enlargement of the heart. Figs. 88 and 89 show an enlargement outward, with the borders of superficial and deep dulness practically parallel to each other, so that, as under normal conditions, the superficial dulness is entirely surrounded (except below) by a strip

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of relative dulness. In Fig. 90 the borders of the superficial and of the deep dulness merge above and near the apex, so that there is only a superficial dulness. This illustrates a reasonably frequent occurrence. It results from the enlarged heart crowding back the lung edges, so that the heart lies directly against the thorax, and naturally furnishes only a superficial dulness. In Fig. 90 the left auricle has pushed the border of the lung so far aside that this chamber and the pulmonary artery both lie close against the chest-wall. The less markedly dilated left ventricle is still partly covered by the lung ; hence, outside of the superficial dulness, a slight relative or deep dulness persists. In Fig. 90 the intensity and the superficial character of the ex-

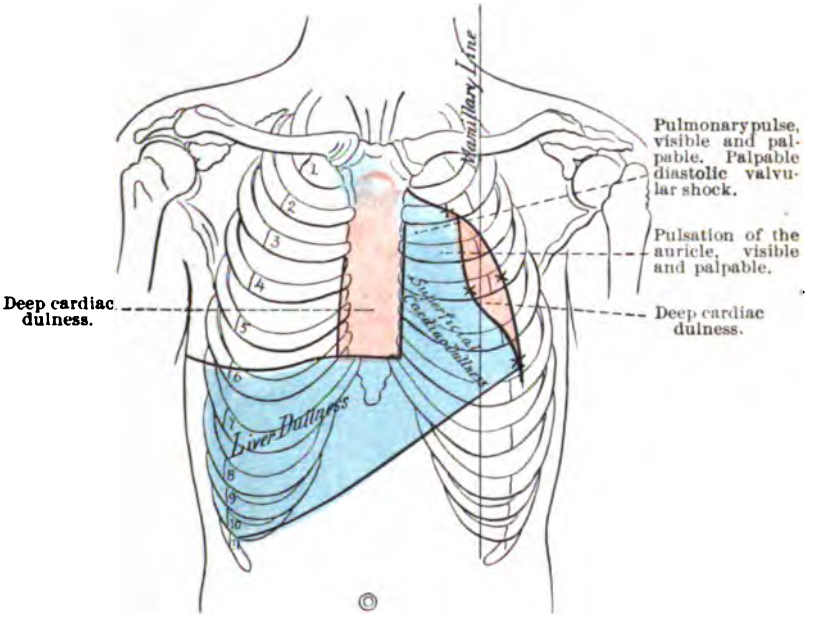

FIG. 90.—Dilatation of the left auricle and left ventricle, with an exposure of the pulmonary artery, in a case of mitral insufficiency.

tensive dulness above, contrasted with the amount of the dulness to the left, the characteristic appendage to the normal superficial cardiac dulness upward, and the results of palpation mentioned in the figure, should suggest the diagnosis of a dilatation of the left auricle or of the pulmonary artery.

Fig. 91 represents a marked dilatation of the right auricle and right ventricle in tricuspid insufficiency. Here again the lungs are pushed so far aside that the dulness is superficial, and the left edge and the apex of the heart lie perfectly free. Above and to the left a small zone of relative dulness remains.

It can be seen in this figure, just as in Fig. 88, that the superficial dulness includes all the lower part of the sternum. Hence the dulness

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can be well defined above, although normally it is difficult to determine by percussion the solid from the air-containing parts (see p. 171) beneath the sternum.

From the bulging of the cardiac dulness upward, as in Figs. 90 and 91, percussion cannot always decide whether the auricles or the great arteries are responsible for the enlargement. The other relations, especially the kind of pulsation over the area, will then assist in settling the condition. (See Palpation and Inspection of the Heart Region.) The percussion results in Fig. 92 are, however, sufficiently characteristic to justify the diagnosis of a dilatation of the aorta.

Fluid Effusion in the Pericardium.—When fluid accumulates in the pericardium, whether as a result of general dropsy or of inflam-

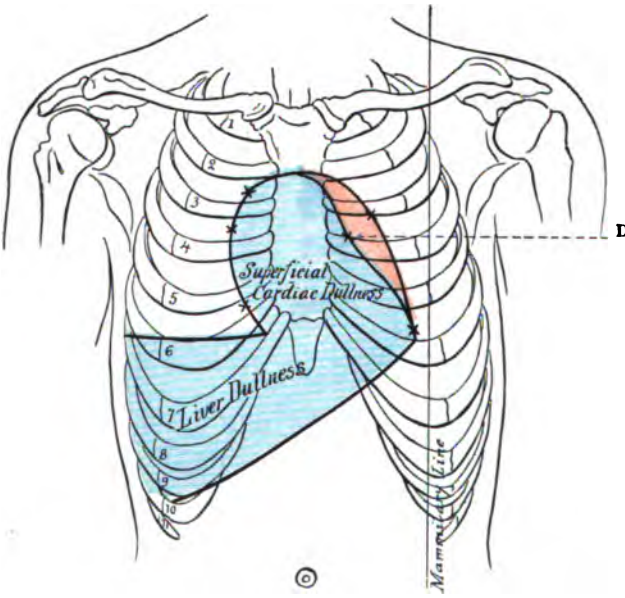

FIG. 91.—Dilatation of the right auricle and right ventricle in a case of tricuspid insufficiency.

mation of the serous membrane, its cavity becomes more and more distended and the edges of the lung are pushed aside, just as by the enlarged heart. If the pericardium lies against the thorax-wall, percussion elicits a superficial (absolute) dulness; but if it is still covered by the lungs the dulness will only be relative, and the superficial and deep dulness will run concentrically. If the distention of the pericardium is very marked, the lungs will be pushed aside and the entire dulness will be superficial, just as in marked cardiac enlargement (see p. 184). The form and the position of the cardiac dulness enlarged by pericardial effusions are, from the anatomic conditions, generally very characteristic (Fig. 88). The specific gravity of the effusion is always lower than that of the heart itself; therefore, with large effusions—*i. e.*, if the lateral portions of the pericardium are filled—the fluid will

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occupy the upper portion of the cavity, and the heart itself the lower. Even relatively slight effusions will increase the cardiac dulness in the dorsal decubitus, because the fluid is then collected against the anterior thoracic wall. If the patient is slightly raised from the recumbent posture, the fluid first collects at the superior bulging of the pericardium, in the neighborhood of the great vascular trunks, beneath the upper end of the sternum, and there pushes the lungs aside. Therefore, quite early in pericardial effusions, the cardiac dulness reaches high up under the sternum or in its neighborhood and assumes a characteristic triangular shape, with a blunt apex above and a broad base below. This triangular form is nothing more than the expression of the shape of the portion of the dilated pericardium which lies against the anterior

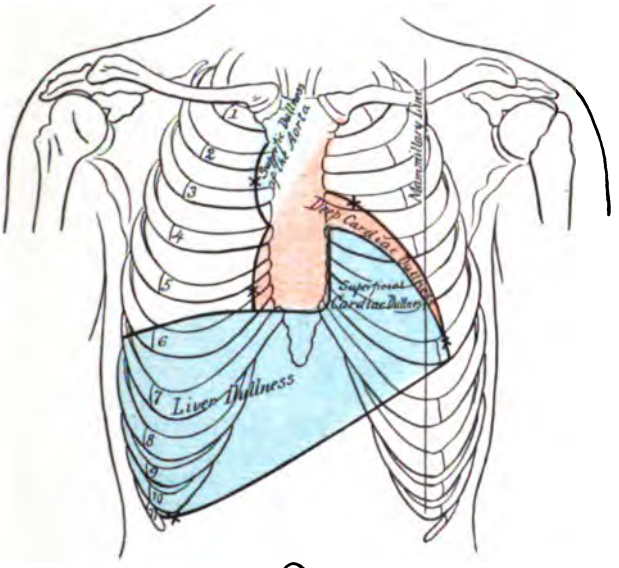

FIG. 92.—Cardiac percussion results in a case of diffuse dilatation of the aorta from aortic insufficiency; dilatation of the left ventricle.

thoracic wall, and since the pericardium is wider at its diaphragmatic portion than at the great vessels, the lungs are pushed farther aside at the lower part than at the upper part. Pericardial distention shows especially plainly at the so-called cardiohepatic angle (Fig. 74); for the lung is very early pushed aside to the right of the sternum, and so changes this angle, normally about 90°, to a much more obtuse angle.[1] An erect posture makes the dulness broader and lie somewhat lower than in the dorsal decubitus (if the effusion is larger), because the fluid, following gravity, flows more forward. Adhesions of the pericardium may prevent the application of any rules for determining the shape of the pericardial dulness.

[1] [This sign has been emphasized in the diagnosis of pericardial effusions in children, by Rotch, of Boston.—ED.]

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The same depression of the upper borders of dulness during the sitting posture has been observed in actual enlargement of the heart from valvular lesions. Therefore we cannot always diagnose a pericardial effusion from the alteration in dulness noted above in the change of posture from lying to sitting. The action of gravity upon the enlarged heart may depress the diaphragm and so cause a lower position, or it may be that more venous blood is retained in the lower half of the body in the erect posture, so that the auricles are not as completely filled. But if the erect posture produces a broadening of the lower part of the dulness, it is certainly more suggestive of a pericardial effusion than if it produces a mere depression of its upper border.

DISLOCATION OF THE HEART DULNESS IN TOTO.

The position of the movable organs of the thoracic and abdominal cavities is the result of the muscular and elastic forces pushing or pulling them on all sides and of the given limitations to their power of movement. The position of the heart is essentially due to the position of equilibrium in which the mediastinum is held between the two pleural cavities and to the position of the diaphragm. Any change of the diaphragmatic position or any disturbance of the equality of pressure between the two pleural cavities will produce a dislocation of the heart.

If a dislocation of the diaphragm develops slowly, the dislocation of the heart will be especially marked, because a gradual stretching overcomes the resistance which makes a dislocation of that part of the diaphragm upon which the heart rests so difficult (fixation of the central tendon by the mediastinum, esophagus, aorta). *Meteorism*, *ascites*, and *voluminous abdominal tumors* push the heart *upward; emphysema* and *collections of fluid* or *air* in the pleural cavities, *downward*.

If the negative pressure in one pleural cavity becomes less markedly negative, or, still more, if it becomes positive, the heart will be pushed toward the side where the absolute pressure is less—*e. g.*, large collections of air or fluid in one pleural cavity dislocate the heart to the opposite side. Thoracentesis generally proves that an effusion which dislocates the heart to any extent is under positive pressure; nevertheless, an effusion under negative pressure would exert a similar dislocating action. For such an action does not depend upon the absolute height of the pressure, but upon the difference between the pressures affecting the two sides of the mediastinum. The heart is merely pushed to one side until this difference in pressure is equalized. The practical importance of this fact, that even a fluid effusion under negative pressure may dislocate the heart, is the warning it gives us always to avoid the risk of introducing air into a chest by aspiration, whether the heart is dislocated or not.

Dislocation of the heart may occur not only when the negative pressure on one side becomes less negative or becomes positive, but also when it becomes still more markedly negative. Here the action is more that of suction than of pressure, and is observed in pleural effusions which have led to a retraction of the lungs. As the exudation becomes absorbed, the heart is drawn over *toward the affected side* to fill the empty space. In fresh pleurisy the heart is pushed *toward the healthy side*. In retraction of the lung from other causes than pleurisy—*e. g.*, from inter-

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stitial pneumonia or tuberculosis—the heart is likewise dislocated to the affected side. It may remain there permanently from continued tugging, or it may gradually return to its normal position from a gradual distention of the lungs.

There is no law to determine how deformities of the thorax will dislocate the cardiac dulness.

The cardiac dulness in *situs inversus* and in *dextrocardia* lies upon the right side, forming a sort of reflected mirror picture, symmetric to its normal form and position.

By starting from the normal relations we can easily understand the form and position of a dislocated cardiac dulness. Slight dislocations (from pleural effusions, etc.) only produce a lateral displacement, while marked dislocations occasion both a lateral and a pendulum movement. On account of the normal oblique position of the heart, such a pendulum movement probably occurs more readily in dislocations to the left; whereas, on account of the resistance of the central tendon of the diaphragm, a similar pendulum dislocation to the right requires much more force. The results of pathologic findings and the experiments of Ferber[1] finally settled the much disputed pendulum movement of the dislocated heart.

In the ordinary course of a dislocated heart from a pleuritic exudation we can percuss only the borders of the latter that lie opposite the exudation, because the dulness of the exudation merges with the cardiac dulness. If the heart is dislocated to the left, the left edge of the heart will assume the same position as in left-sided cardiac dilatation. If a left-sided exudation dislocates the heart considerably to the right, the right cardiac boundary and even the apex beat (exactly as in dextrocardia) may be found in the neighborhood of the right mammillary line or even still farther outside. Both superficial and deep cardiac dulness will be found to the right of the sternum; the former will merge into the liver dulness, and the latter will run around the superficial dulness, in the shape of a concentric strip, from above and to the right. We can differentiate the result from *dextrocardia* by the pleuritic dulness which occupies the place of the normal heart dulness; from *situs inversus*, by the normal right-sided position of the liver.

Fig. 97 shows a large left-sided pleural exudate dislocating the cardiac dulness to the right; Fig. 99, a right-sided pyopneumothorax pushing the cardiac dulness to the left. From these figures it can be seen that in dislocation of the heart the lung edges may be so completely pushed aside as to make the entire cardiac dulness superficial, as in enlargements of the heart (see p. 183) and pericardial effusions (see p. 186).

If the heart is pushed upward by a distension of the abdomen, the cardiac dulness not only lies higher, but also appears larger—*i. e.*, broader than normal. This is partly due to the retraction of the lung edges away from the heart in consequence of the limitation of space in

[1] "Die physikalischen Symptome der Pleuritis exsudativa," *Habilitationsschrift*, Marburg, Elwert, 1875.

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the thorax (see pp. 175, 176, and 181). The heart dulness may even be twisted by a sort of pendulum movement into a horizontal position, because the apex of the heart, which lies upon the movable portion of the diaphragm, is lifted up to the level of, or even higher than, the base.

TOPOGRAPHIC PERCUSSION OF THE LIVER.

NORMAL LIVER DULNESS.

Clinicians have often attempted to determine a superficial and a deep dulness for the liver just as for the heart—*i. e.*, to bound the anterior surface lying directly against the thoracic and abdominal wall, and to estimate the height to which the liver in the dome of the diaphragm rises into the thorax. The latter determination would be of very little value even if it were possible. The highest point of the liver lies far removed from the anterior thoracic wall; and in large persons with well-developed chests it is too remote for the acoustic range of the percussion blow. Again, although we can obtain a relative dulness (a diminution of the fulness of the tone) above the lung-liver boundary corresponding to the wedge-shaped form of the lower edge of the lung, yet the upper limit of this dulness will almost always be lower than the highest point of the liver. But, as stated above, the estimation of the highest point of the liver is of little value. The liver is applied closely to the diaphragm, and so the upper liver boundary merely coincides with the position of the diaphragm, and we can determine that accurately enough by estimating the lung-liver boundary and the liver edge. If the diaphragm is high, the lung-liver boundary and the liver edge are always high. If the diaphragm is low, the reverse is true. The lower liver boundary, which is generally easy to determine by percussion, usually shows whether the organ is increased or diminished in size. An enlargement of the liver crowds the lower liver edge correspondingly downward; except, though rarely, when the muscular resistance of the diaphragm is diminished—*e. g.*, with circumscribed tumors of the upper liver surface, *abscesses*, or *echinococcus cysts*. Even here the estimation of the so-called "relative liver dulness" is apt to be so unreliable as to be of little value, for the superficial lung-liver boundary is probably always somewhat crowded by such a growth.

Ordinarily, therefore, we are contented with estimating by percussion the part of the liver applied to the abdominal wall and situated beneath the edge of the lung. Under favorable circumstances we can map out a superficial dulness such as is pictured in Fig. 79. At the sharp edge of the liver this dulness is so slight that only very light percussion appreciates the dulled note, while stronger percussion sets the underlying intestines in vibration. In the same way very light percussion is essential near the lung, for otherwise the resonant tone of the adjoining lung tissue obscures the dulness. Stronger percussion, however, brings out very clearly the superficial liver dulness higher up, corresponding to the thickness of the layer of the liver percussed. The upper borders of the superficial liver dulness coincide with the lower borders of pulmonary

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resonance. They can be plainly demonstrated from the front to the back. The lower border of the superficial liver dulness ordinarily corresponds to the left boundary of the superficial cardiac dulness, intersecting the fifth or sixth rib between the left parasternal and the mammillary lines (Fig. 79). In the median line it lies halfway between the navel and the base of the xiphoid process, sometimes even higher. In the right mammillary line it reaches the edge of the ribs or projects slightly below them, and it lies upon the tenth rib at the right middle axillary line. All these measurements refer to patients who are breathing quietly and in the recumbent posture. The inferior edge of the liver behind can not usually be plainly demonstrated on account of the thickness of the muscle layer.

The writer protests against making statements about the absolute height of the liver dulness, because no general value can as yet be attached to such measurements; but in concrete cases the vertical diameters of the liver dulness at the different arbitrary lines should be measured in order to recognize any changes in the liver size during the observation of the same case.

ACTIVE AND PASSIVE MOBILITY OF THE LIVER DULNESS.

The liver dulness also possesses an active mobility with respiration, and a passive mobility with an alteration of the patient's position. The active and passive mobility of the upper edge of the superficial liver dulness coincides with the corresponding mobility of the pulmonary border (p. 173 *et seq.*). The active mobility of the lower liver border corresponds to the respiratory mobility of the dome of the diaphragm, and is much less than that of the pulmonary border. When a patient lies upon his left side, the passive mobility of the lower liver edge is shown by a depression of the right lobe. When he lies upon the right side, the reverse occurs (rotation of the liver about a sagittal axis). The liver is more difficult to percuss in the sitting or standing posture, on account of the increased tension of the abdominal walls. The lower liver edge is occasionally elevated in these positions, probably because the increased intra-abdominal pressure pushes the liver to some extent upward. If the abdominal walls are lax, the weight of the liver produces the reverse effect. (See Lung-liver Boundary.)

PATHOLOGIC DISLOCATIONS AND GROSS CHANGES IN THE LIVER DULNESS.

In "situs inversus" the liver lies in the left upper abdomen, symmetric with its normal position. Its dulness corresponds to the reversed position. Free air in the abdominal cavity nearly obliterates the liver dulness, because the air will be collected at the highest points. (See the Section upon Comparative Percussion of the Abdomen.)

Changes of the Upper Border of the Superficial Liver Dulness.—The upper border apparently lies higher than normal if a pathologic dulness from the thoracic organs (pleural effusions or lung consolidation) is superimposed upon the liver dulness. It is therefore necessary to resort to other methods of examination to determine whether it is a genuine high liver dulness or a normal liver dulness plus a pathologic pulmonary or pleural dulness (see below).

The upper border will be higher if the liver is pushed upward *in*

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toto—*e. g.*, by increased abdominal tension. In this case the liver will ordinarily be rotated about a frontal axis, so that its inferior edge is elevated still more than the lung-liver boundary, and the vertical breadth of the liver dulness appears abbreviated (angular position of the liver). If, at the same time, the liver is enlarged, despite its being canted upward, the lower edge may stand at its normal place or even lower.

Pulmonary retraction will also cause a higher position of the lung-liver boundaries.

A mere enlargement of the liver without marked increase of the intra-abdominal pressure—*i. e.*, without the liver being pushed upward—will not ordinarily cause an elevation of the lung-liver boundaries, because the organ will naturally enlarge in the direction of the least resistance and not against the diaphragm. If such an enlargement of the liver occurs so rapidly that its ligaments are not quickly enough stretched, a marked increase upward will result—*e. g.*, most frequently in unequal enlargements of the upper surface of the liver, which cause a localized pressure (tumors, echinococcus cysts, and abscesses of the convexity). In most of these cases, however, the lower edge of the liver will also be pushed downward. Subphrenic abscesses produce practically identical percussion results with those obtained in tumors of the convexity.

Emphysema, either by crowding the diaphragm *in toto* downward or by filling the pleural sinus more completely with lung, will depress the upper border of the superficial liver dulness to a variable degree, depending upon which of these possibilities is present. *Pneumothorax* effects a similar result. Lessening of the intra-abdominal pressure, and exceptionally *enteroptosis*, will also depress the upper liver border.

Changes in the Lower Border of the Superficial Liver Dulness.—We have discussed some of these in the preceding section. They depend either upon a dislocation of the liver, a change in its size, or upon an association of these conditions. A careful attention to the entire clinical picture will differentiate these different conditions—*e. g.*, the etiology (alcohol, anemia *versus* pleural effusion, ascites, tympanites), the accompanying relations (stasis), a palpatory appreciation of alterations in the consistence (cirrhosis, carcinoma).

If the dislocating cause evenly affects the entire upper or lower surface, the lower edge of the liver will be pushed upward or downward as a whole—*i. e.*, symmetric and parallel to its former position (meteorism, ascites, emphysema). If, on the contrary, the pressure acts unevenly, the organ will, by virtue of its attachments, be dislocated in an asymmetric fashion.

Accumulations of air or fluid in the right pleural cavity exceptionally rotate the liver about a sagittal axis. Conversely, a left pleuritic or left pericardial effusion will sometimes depress the left half of the liver. Similarly, though in a reversed way, a one-sided pulmonary retraction will elevate the liver.

Solid masses beneath the liver (packed intestines, tumors of the omentum, of the colon, of the stomach, and the like) may increase the

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liver dulness downward. Palpation or a carefully repeated examination generally discloses the true condition.

Conversely, intestines distended with air, overlying the liver and hiding part of it, may simulate a high position of the liver edge.

TOPOGRAPHIC PERCUSSION OF THE SPLEEN.

NORMAL SPLENIC DULNESS; HALF-MOON-SHAPED (TRAUBE'S) SPACE.

The spleen lies in the left hypochondrium, between the ninth and the eleventh rib (Fig. 75). Its long diameter ordinarily corresponds to the tenth rib. Its posterior extremity is situated only a few centimeters from the spinal column. Its anterior extremity reaches only to the middle or, at most, to the anterior axillary line. In relation to the course of the ribs, the long axis of the organ runs from behind and above forward and downward. The posterior upper third of the spleen lies hidden under the edge of the lung. The front and lower two-thirds ordinarily lie against the thoracic wall unless, as very frequently happens, intestines have pushed in between the thorax and the spleen.

Percussion can only differentiate that portion of the spleen which is uncovered by lung. Hence, it merely estimates a superficial dulness which can be accurately determined only by very light percussion (see p. 160).

To map out the free part of the spleen as perfectly as possible by means of percussion, the patient should be sitting, standing, or lying somewhat upon the right side (the so-called oblique position), because in the directly recumbent position the posterior portions of the spleen are not accessible to percussion. The right oblique position has the advantage that the contents of a well-filled stomach are pushed aside, and so do not simulate an enlarged splenic dulness.

The normal splenic dulness in the sitting or standing posture is pictured in Fig. 80. The upper boundary of the dulness corresponds to that portion of the lung edge which intersects the eighth and ninth ribs, and runs from the middle to the posterior axillary line. The anterior border of the dulness rarely reaches the anterior axillary line; in adults it is about 5 cm. distant from the costal margin. The dulness reaches as low as the eleventh rib. Measured in the long axis of the body, the height of the dulness varies between 5 and 6 cm. Posteriorly the splenic dulness merges into that of the lumbar region, due to the thick layers of muscles, not to the kidneys (see p. 195 *et seq.*).

The splenic dulness is but slightly dislocated either upward or downward in changing from the sitting or standing to the reclining posture. In such cases it follows the same laws as the liver dulness, and moves as a whole up or down (see p. 190). The anterior boundary then usually remains the same. The splenic dulness is only slightly dislocated forward and downward in the oblique and lateral postures, because, although the left lung then assumes a deeper position, at the same time more of the upper part of the spleen is covered by the lung. A splenic dulness

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which projects beyond the anterior axillary line, even in the oblique and lateral posture, is abnormal.

The beginner should practice splenic percussion upon patients first in one position, then in another, in order to control the results.

It is useless to attempt to map out the portion of the spleen lying above the edge of the lung—*i. e.*, to determine a deep splenic dulness. The spleen is a comparatively small organ, covered outside by the lung, and inside in contact with the stomach and intestines, so that a percussion stroke strong enough to penetrate through the lung would set the tympanitic stomach into vibration. Besides, the differentiation of that part of the spleen covered by the lung has no practical value, because, just as in the case of the liver, any alterations can be easily recognized in the parts lying free (see p. 190).

Percussion of the spleen is frequently not as simple as the above description would indicate. The spleen may be abnormally broad behind, or may lie abnormally high, so that it practically escapes percussion, and still be within physiologic limits of size. With meteorism, when the distended intestines push the spleen upward and backward or crowd between the spleen and the thoracic wall, the vibration of such large air-containing cavities may make it almost impossible to differentiate so thin a solid organ as the spleen. Again, solid or fluid stomach or intestinal contents sometimes simulate a splenic dulness. The right oblique position will generally prevent confusion on account of stomach contents, and repeated examination will show the true state of affairs in regard to the intestinal contents. Despite every care, however, we must confess that palpation is very much more trustworthy than percussion of this organ.

Between the splenic dulness and the left end of the liver dulness there is normally a tympanitic area due to the stomach or intestines. This space is bounded above by the lung border, below by the free costal margin, and is called the half-moon-shaped or Traube's space. It is important in the diagnosis of left-sided pleuritic exudations (Figs. 79, 80, and 95). Percussion cannot always differentiate this space above from the lung; but then a boundary line can always be constructed corresponding to the edge of the right lung.

PATHOLOGIC GROSS CHANGES AND DISLOCATIONS OF THE SPLENIC DULNESS.

Enlargement of the spleen is ordinarily evident by an increase in the splenic dulness forward and downward (Fig. 93). If the splenic dulness projects forward beyond the anterior axillary line, or if its vertical extent in adults measures more than 7 cm., the spleen can be considered increased in size.

Diminution or even a complete disappearance of the splenic dulness happens frequently enough in perfectly healthy men with normal spleens. It has no clinical importance.

Pathologic dislocations of the spleen are of slight clinical importance. They are mostly difficult to demonstrate. Although pleural effusions, ascites, meteorism, and tumors may dislocate the spleen, percussion will rarely be of much

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help, because either another dulness occupies the place of the spleen, or else there is a resonant note (meteorism).

The spleen is sometimes dragged downward and forward, with a marked dilatation of the stomach, by stretching the ligamentum gastrolienale. It will then be more accessible than normally to percussion and palpation. (See later, Examination of the Stomach.)

Large pleural effusions occasioning a dulness in Traube's space, as well as en-

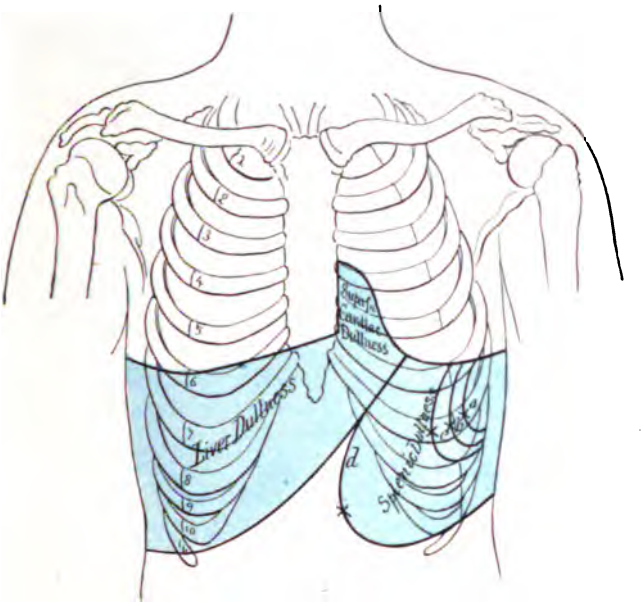

FIG. 93.—The splenic dulness in different grades of splenic enlargement (*a*, *b*, *c*, *d*).

largements of the liver, may cause the hepatic and splenic dulness to merge (see Fig. 95). Under these circumstances percussion may not detect even enormous enlargements of the spleen.

TOPOGRAPHIC PERCUSSION OF THE KIDNEYS.

The anatomic position of the kidneys (Fig. 76) shows how impossible it is to limit their boundaries by percussion. Their deep position prevents percussion from in front, and the thick layers of muscles behind produce a dulness which such a thin structure as a kidney could not increase. If we percuss a patient whose kidney has been removed, we can demonstrate the same vertical line of dulness as in a patient with an intact kidney. It corresponds to the outer edge of the sacrospinal muscles and has nothing to do with the kidney. Large renal tumors afford an intense dulness in the loins, projecting far beyond the boundaries of the sacrospinal muscles. If the tumor has pushed the other abdominal viscera aside and lies against the abdominal wall, it can be demonstrated from in front. Here it is of special diagnostic importance to demonstrate by percussion that the colon is anterior to the kidney

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(see below). Palpation is, however, far more important than percussion in this instance, as in the diagnosis of all other abdominal tumors.

TOPOGRAPHIC PERCUSSION OF AIR-CONTAINING ABDOMINAL VISCERA.

(See also section upon Examination of the Stomach.) Percussion is of no value for distinguishing one of these organs from the other, except under certain quite well-recognized conditions—viz., where the stomach or separate sections of the intestines are filled with air, fluid or solid contents. And then only superficial boundaries (*i. e.*, with light percussion) can be accurately estimated, because stronger percussion transmits the vibrations unexpectedly in all directions. Even superficial percussion is quite untrustworthy, because physiologically the position of the stomach and intestines varies considerably; because their superficial boundaries are rarely sharp; and because although at different portions of the intestines the tympanitic tone varies in intensity and pitch it is impossible to differentiate one region from the other. We can usually distinguish the swollen stomach or the distended colon from the small intestines by ordinary percussion. If this is not possible, the stick-pleximeter percussion or the auscultatory percussion (see p. 158) will differentiate them, provided there is increased tension enough to produce a metallic resonance over the air-containing intestines. Both these methods of examination elicit either a sharp rise in the tone height of the metallic resonance or a sudden cessation of the resonance at the edges of the organs. However, every spot over a hollow organ does not necessarily give the same pitch of metallic resonance. Metallic resonance over an air cavity may be present or lacking; and, again, its pitch may differ over different places of the organ, especially if it is large. If we trace the course of the colon distended with gas, by means of the stick-pleximeter percussion, we often can appreciate entire scales of metallic resonance. Different pitches may be present even over the stomach. Hence, the value of this examination method must be limited, although we may make it easier by dilating the stomach or colon with air. (See Palpation of the Abdomen and Examination of the Stomach.)

The demonstration by percussion of the tympanitic colon anterior to a kidney tumor is very valuable for diagnosis (Fig. 109). It is safer to percuss both before and after distending the colon with water and then with air.

Inspection and palpation generally furnish better evidence of the extent, position, and peculiarity of the stomach and intestines than percussion. (See here special section, Examination of the Stomach.)

TOPOGRAPHIC PERCUSSION OF THE BLADDER AND OF THE UTERUS.

The bladder when empty lies well hidden behind the symphysis pubis. When filled it ascends, even reaching in cases of urinary

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retention above the navel. As it rises it pushes the intestines aside and lies against the abdominal wall in the form of a vertically placed oval tumor, furnishing an intense dulness to faint percussion, which corresponds pretty accurately to its extent and shape. If not very markedly distended and if some loops of intestines lie between it and the abdominal wall, part of the dulness may be deep (plainer to strong percussion), or there may even be no dulness present.

According to Müller's investigations, a bladder must contain between 500 and 600 c.c. before it will produce an appreciable dulness in normally developed women, and 360 to 500 c.c. in men.[1] If the rectum is distended, the bladder dulness projects somewhat more to the right. In lateral positions it is depressed to the deeper side, sinking at the same time somewhat into the depth of the pelvis, and so appearing smaller.

The condition of the bladder can be determined more accurately by palpation than by percussion unless the tension is very great or the abdominal walls very thick.

The pregnant or pathologically enlarged uterus acts like the bladder to percussion, and can be distinguished from it only by careful attention to other clinical relations, by determination of the consistence, by means of palpation, by vaginal examination, and by catheterization.

COMPARATIVE PERCUSSION.

Comparative percussion is concerned with the qualitative changes of the percussion tone over one and the same organ, and the conclusions to be drawn from such changes in regard to the peculiarity of the organ and its surroundings. Topographic percussion, as we have seen, furnishes us with conclusions as to borders—*i. e.*, as to the size and position of organs. In comparative percussion we compare the tone over a certain spot of the body with the normal tone over this spot, or with the tone over adjacent parts of similar organs which are normal. In the case of solid organs percussion, of course, can only bound them—*i. e.*, determine their size, not their structure—and for this purpose topographic percussion is all that is essential. But with the air-containing organs of the abdomen, and especially those of the thorax—the lungs—we may determine countless quantitative and qualitative modifications of the percussion note which will furnish us most important information about pathologic changes at their surface or in their interior.

COMPARATIVE PERCUSSION OF THE THORAX.

The ordinary lung tone is resonant, loud, not tympanitic. The resonance or loudness varies with the thickness of the covering, and is also influenced by the adjacent organs (heart, liver, etc.). Beneath thick muscles or fat—*e. g.*, over the scapula and over the female breast—the percussion tone is less resonant than at other places. The intense dulness of the pulmonary tone in such spots can be modified only by

[1] *Berlin. klin. Woch.*, 1895, No. 13, p. 278.

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very vigorous percussion. Again, the pulmonary tone is necessarily less resonant (see pp. 190 *et seq.*, pp. 162, 178) over areas where the pulmonary tissue is thin—*e. g.*, the tone over the normal apices is faint compared with the loud tone over more voluminous portions of the lung. The examiner's ear gradually accustoms itself to these physiologic differences, so that they are no longer heeded. Marked convexity of the thoracic wall will cause a certain dulness—*e. g.*, in kyphotic and scoliotic patients. If we percuss upon a markedly convex spot of the thorax, a part of the percussion force is lost, because such places yield so little; whereas a flat section of a rib oscillates to percussion in the direction of its greatest elasticity. So we must be careful in drawing conclusions from the tones obtained over kyphoscoliotic chests and over physiologically altered thoraces.

Every structural change of the lung itself has an important influence upon the character of the pulmonary tone, which may become abnormally resonant, tympanitic, or more or less dulled. The change can be appreciated by comparing the tone with that over either symmetric or neighboring areas. Wherever the changes in the tone include an entire lung, the beginner must compare the tone with that of a healthy person. To the skilled these variations from the normal are easily appreciated without such comparison.

APPEARANCE OF A DULLED NOTE WITHIN THE LUNG BOUNDARIES.

In order not to overlook slight dulnesses, it is always a good rule to utilize percussion of varying strengths. To make clear the importance of this precaution, it is necessary to enumerate the different anatomic possibilities which can produce a dulness in the lung tone.

Whenever the acoustic range of the percussion blow includes a smaller amount of air-containing lung tissue, or an area containing less air than normally, the tone will be dulled (p. 162 *et seq.*). This may be caused:

1. By the interposition of airless material between the lung and the thoracic wall: exudations, adhesions, tumors (Fig. 94, I.).

2. By diminution or absence of air in the lung parenchyma itself —*e. g.*, in *atelectasis* (collapse of the alveoli), from compression or from closure of the bronchi with consequent resorption of the air; in *pneumonia*, from the filling of the alveoli with an airless inflammatory exudate; in *carcinoma*, from the newly formed tumor tissue taking the place of the lung tissue. These changes may occur in large areas which reach the surface of the lung (Fig. 94, II.); in small scattered areas (lobular), partly at the surface, partly separated from it by lung tissue still containing air (Fig. 94, III.); or, finally, in still larger areas situated in the depths of the lung (Fig. 94, IV.). Each of these cases will furnish different percussion results.

If we percuss vigorously at *b* (Fig. 94, I.), provided the exudate is not too thick, the lung can be so strongly vibrated that the dulness will be completely overlooked. To differentiate such a dulness as sharply as possible from the surroundings we should here percuss as lightly as

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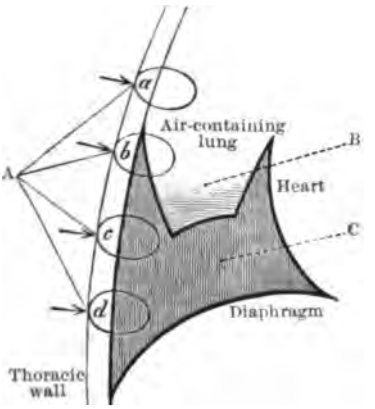
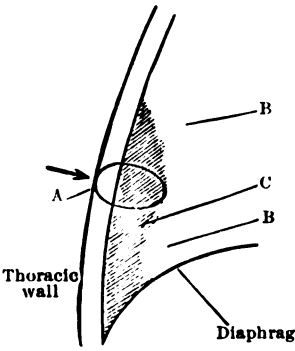
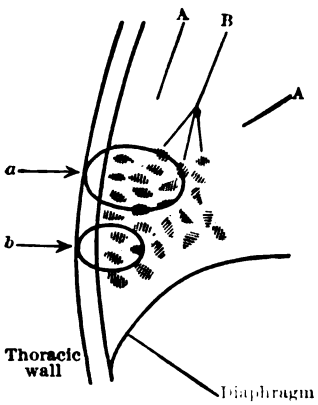
possible. The same holds good for the condition represented in Fig. 94, II. The dulness is superficial in both instances, and can be best attained by carefully regulating the acoustic range of the percussion



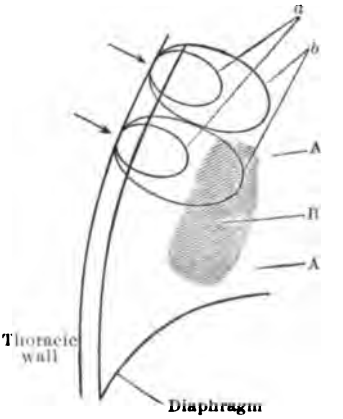
I. Pleural exudation: *A*, Acoustic sphere of action of light percussion; *B*, compressed lung; *C*, pleural exudation.



II. Superficial infiltration area: *A*, Acoustic sphere of action of light percussion; *B*, *B*, air-containing lung tissue; *C*, infiltrated lung tissue.



III. Disseminated small areas of consolidation: *a*, Sphere of action of strong, *b*, of weak, percussion; *A*, *A*, air-containing lung tissue; *B*, small infiltration area.



IV. Larger areas of consolidation lying deeper: *a*, Sphere of action of weak, *b*, of strong, percussion; *A*, *A*, air-containing lung tissue; *B*, infiltrated lung tissue.

FIG. 94.—The different varieties (*i. e.*, methods of origin) of dulling the lung tone. Diagrammatic frontal section through the thorax: I., Pleural exudation; II., III., IV., pulmonary infiltration.

blow so that its longest diameter will not overstep the solid portion, as in Fig. 94, II.

In Fig. 94, III., no very marked dulness can be evoked either by light (*b*) or by strong (*a*) percussion; both types will furnish only a slight dulness, because air-containing tissue will still be vibrated. The results of light or strong percussion will vary with the position of the infiltrated area, whether it is near the surface or not. If the solid

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area as a whole is not very extensive, light percussion will bring out the dulness more plainly, because less air-containing tissue in the interior will be set in vibration.

Light percussion (acoustic range *a*) will demonstrate nothing with a larger, deeply placed area, as in Fig. 94, IV.; but strong percussion (acoustic range *b*) will demonstrate a relative dulness.[1]

Of course, we do not know beforehand whether there is any dulness, or if so what kind of dulness; therefore it is always advisable to employ different strengths of percussion in succession. Then we can draw conclusions as to the cause and position—deep or superficial—of the dulness.

It is not to be expected that percussion can demonstrate every small area of consolidation. In fact, experience teaches us that isolated areas of consolidation, even though superficially located, to be appreciated must have a flattened extent of at least a *few square centimeters*. If they lie deeper, they must naturally be larger. But there are no generally applicable rules. For instance, multiple areas, if scattered thickly enough, need not be nearly so large as single areas to cause a diminution of the resonance—*e. g.*, a lung richly sprinkled with miliary tubercles will sometimes give a relatively dulled tone. On the contrary, even very thickly spread small areas at other times will cause no dulness. The writer has watched cases where the pulmonary resonance was absolutely normal during life, and yet the autopsies showed that the lungs were completely infiltrated with sarcomatous nodules the size of nuts. There are many other important factors in this connection besides the amount of air contained in the lungs. For example, a relaxation of the lung tissue adjacent to solid areas will make the tone more resonant than normal (see p. 210), and this hyperresonance of the tone may offset the dulness from disseminated tumor nodules or inflammatory infiltrations.

In most cases other methods of examination must be resorted to in order to appreciate the causes of dulness. The most important characteristics of the different kinds of dulness of the lung tone will be mentioned in the following sections:

Pleuritic Dulness.

Early in its formation a pleuritic effusion furnishes a dulness in the lower posterior part of the thorax. This gradually rises higher, and pushes further forward, the boundary line running downward and forward (Figs. 95, 97, I., II.). If the effusion increases still farther the dulness increases forward, until finally the greater part of that side of the thorax is dull, except that a slightly resonant tone can be elicited in the upper portions. Exceptionally the dulness may run circularly around the thorax instead of bowing down toward the front.

[1] [Forcible percussion over the lung area, although sometimes necessary, on account either of muscular development or an excess of fat, is generally inaccurate. Even in mapping out the area of deep cardiac dulness, light percussion is usually preferable.—ED.]

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Why this dulness, which represents the position of the border of the exudate, forms such an oblique line has been a mooted question. The ordinary supposition is that the force of gravity determines the position of the exudate, and that its peculiar obliquity is essentially produced by the patient's position during the development of the exudation, and then fixed by encapsulation. If, then, a patient during the entire course of a slowly developing exudation be walking about, we should naturally suppose that the law of gravity would produce a horizontal position for the fluid. As in most cases, however, patients remain in bed in the slightly raised position, the line running downward and forward would correspond to a horizontal one for them. But this theory will not hold, for even if patients do walk about during the formation of an exudation, the same curved line is almost always seen. Therefore another explanation of this appearance must be assumed. Probably the following supposition is correct. Normally the surface of the lungs is kept applied to the costal pleura by the difference between the air pressure and the power of retraction or elasticity of the lungs. Now, it is physically impossible that the elasticity in so irregularly shaped an organ as the lung should be at all points of the surface equal. And wherever the retraction power of the lung is stronger, there it is that more of the intrabronchial air pressure will be borne, because less physical resistance prevents a mechanical separation of the pleura pulmonis from the pleura costalis. The posterior more voluminous parts of the lungs undoubtedly possess the strongest retraction power, so that the exudation behind finds the least resistance to its formation and accumulation. Thus, the boundary line bends downward and forward.[1] In favor of this supposition is the fact that the exudation is not only higher but also thicker behind.

[**Ellis' Line.**—In America the line of pleuritic dulness was first described by Calvin Ellis, at the March meeting, 1873, of the Boston Society for Medical Improvement.[2] He said: "When a pleural effusion is small it may occupy a conical portion of the pleural cavity in the subaxillary region, where respiration and resonance may be wanting. But in a certain number of cases, when the effusion is quite large, if an accurate line be drawn the flatness will be found to describe a curve, gradually approaching the spine toward the base of the chest, leaving a space from 1 to 3 in. broad between the spine and the line of flatness. In this space resonance will still be detected and respiration heard."

In a letter to the editors of the *Boston Medical and Surgical Journal*, January 23, 1874, Dr. George W. Garland reported a series of experiments on lungs of animals, in which he claims to have explained the curved line of dulness described by Ellis.

In a manual entitled *Pneumo-Dynamics*, by G. W. Garland, January 7, 1877, p. 6, the author says that this line was first described by Damoiseau, of Paris, and redescribed by Ellis, of Boston. The latter was the first to trace its true shape.—ED.]

[1] Ellis's or Garland's line is the term generally used in America.
[2] *Boston Med. and Surg. Jour.*, Jan. 1, 1873.

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In dropsical effusions, hydrothorax (see p. 205 *et seq.*), the level of the fluid, to be sure, sometimes becomes horizontal, accurately corresponding to the position of the patient; but, of course, the influence of gravity must be remarkably strong upon a large dropsical effusion. In an early case of hydrothorax, where there is only a slight amount of fluid, the line of dulness follows practically the same curve as in an exudation. Since there are no adhesions, and since the fluid is free and movable, we should suppose that it would follow the law of gravity. The reason why it does not must depend upon the force with which the border of the lungs is pressed against the thorax. Even a large exudate

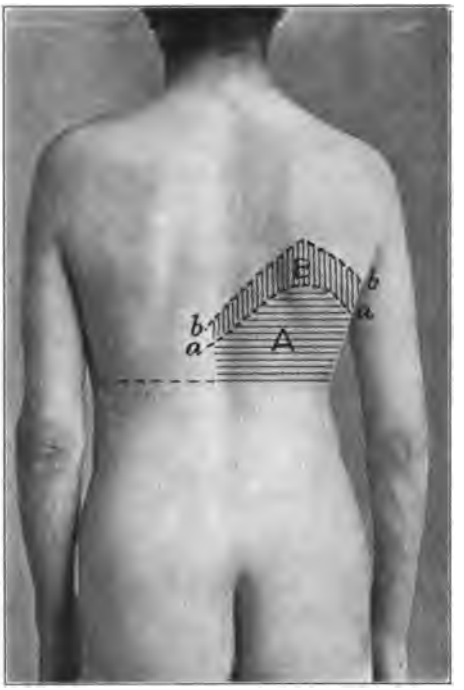

FIG. 95.—Shape of dulness in a fresh pleural effusion. Ellis's or Garland's line: *a*, *a*, represents the upper border of dulness in a small effusion; *b*, *b*, the border in a larger effusion.

usually persists in showing this curved line of dulness, probably because adhesions of the pleural surfaces are formed almost immediately, and these adhesions are only very slowly dissolved by the increasing exudate; whereas in a hydrothorax without adhesions the fluid meets with no marked resistance, and so follows the laws of gravity more completely. Moreover, in moderate-sized hydrothorax we can often enough prove that the change of level due to gravity only very gradually follows, showing that the anterior portion of the lung still opposes a certain resistance to the dislocation of the fluid. The cases where the level of dulness is horizontal are but rarely due to pleurisy, nor are they

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limited to patients who go about during the formation of the exudation. In such instances some abnormal resistances behind—*e. g.*, old adhesions or pulmonary changes—probably oppose the collection of the exudation. All possible anomalies in the form of the dulness may be thus explained.

Besides the peculiarity of the boundary line, large pleuritic exudations are characterized by the marked intensity of the dulness (*flatness*). We seldom meet so intense a dulness (the peculiar thigh tone) in consolidation of the lungs, because ordinarily in the latter case the bronchi still contain air and percussion still produces a certain amount of resonance.

Of still more importance, however, in distinguishing pleural exudations from pneumonia is the demonstration of a *dislocation of the heart and liver* (see pp. 188 *et seq.*, and 192), and, in marked cases, of a decided enlargement of the affected side of the thorax (see p. 33 *et seq.*).

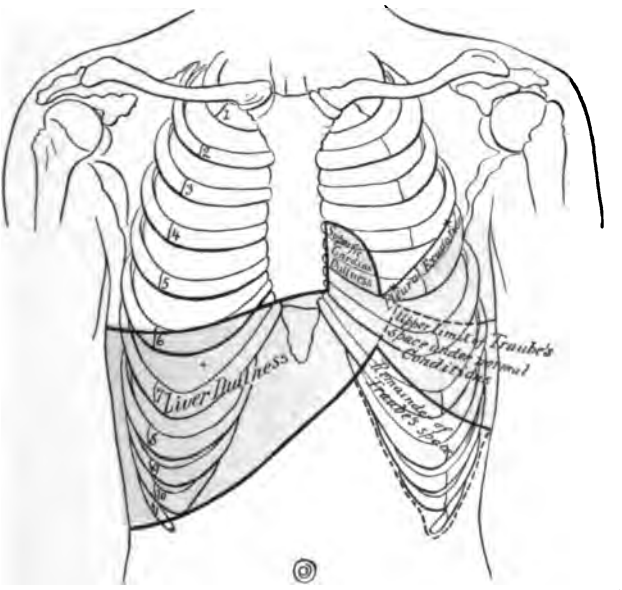

FIG. 96.—Dulness in left-sided pleural exudation. Diminution of Traube's space.

Corresponding to the position of the complementary pleural sinus (Figs. 74, 75, pp. 165 and 166 *et seq.*) the dulness in left-sided effusions reaches lower than the left pulmonary edge. The position of the latter can be determined from the position of the lung-liver boundary. Traube's space, the half-moon space (Figs. 79 and 80) between the spleen, lung, and liver, will then be encroached upon by a dulness from above (see Figs. 96 and 97, I.). Such a dulness can be very easily demonstrated, and when present is diagnostic of a left-sided pleural effusion; but according to the writer's experience left-sided exudations do not always produce this sign, because the complementary pleural sinus is often obliterated by early formed firm adhesions, so that the exudate does not

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push into the sinus. Very large pleuritic effusions may force the diaphragm downward, so that the dulness which corresponds to the convex bowing of the diaphragm below may oftentimes project beyond the rib border as a narrow strip parallel to the costal margin (Fig. 97, I.). This is rarely observed, because the intestines overlap the projection of the diaphragm.

Pleural effusions are rarely, if ever, freely movable with the change of the patient's position; this can be conceived of only if there are no inflammatory adhesions at the borders of the exudation. Such exudations, more frequently serous than purulent, would furnish the same percussion results as a transudation (hydrothorax).

A pleural exudate, even if limited by adhesions, may be slightly mobile. This will be shown by the fluids moving from behind forward inside of the encapsulated space during the upright position of the patient. Sitting or standing increases the intensity of the dulness anteriorly and diminishes it posteriorly without altering its boundaries. The fluid presumably flows between the base of the lung and the diaphragm. This latter peculiarity has some diagnostic importance, because it explains how small exudates can be plainly demonstrated behind immediately after the sitting posture is assumed, while later on the dulness seems to disappear, or, at least, to be diminished in height and intensity.

Light percussion is best suited to determine the boundaries of pleuritic exudations, since the dulness is superficial. Fig. 94, I., shows a frontal section of a pleuritic exudation. It narrows like a wedge at the top and at the bottom, so that at the top and in Traube's space only a very light percussion could correctly determine the boundary.

If a pleuritic exudation becomes still larger, it compresses not only the part of the lung internal to the fluid (Fig. 94, I.), but the entire lung, the pressure spreading in the direction from the base of the lung to the apex; hence, the lung tissue above the exudation is under increased tension, and furnishes at first a hyperresonant and tympanitic note (p. 156), which soon, in consequence of diminished air content, becomes more and more dulled (p. 155). The tone over a pleural exudate is therefore at first contrasted with the normal lung tone, later with a hyperresonant, tympanitic tone, and finally with a relatively dulled tone. In the last instance differentiation is often very difficult but generally possible, because the tone over compressed lung tissue whose bronchi still contain air sounds less intensely dulled than that over the exudate, and frequently possesses some tympanitic quality. The Williams's tracheal tone (see p. 214) can sometimes be obtained when the pulmonary compression is very marked. (See Fig. 97 and the following pages for the percussion relations and for the signs of dislocation in large pleuritic effusions.)

If a pleuritic exudation becomes absorbed except for the fibrinous coating left behind, dulness may still persist. To distinguish such a condition from a fluid exudation necessitates a very careful attention to other symptoms and to other examination methods.

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As a rule, fibrinous thickenings produce intense dulness only when they are associated with signs of thoracic contraction. In such an event the atelectasis of the lungs usually adds its share to the dulness. We have no right to argue against a fluid effusion because the signs remain stationary, as exploratory puncture has frequently convinced the writer of the presence of fluid in cases where the dulness which persisted long after the beginning of the pleurisy had been attributed to fibrinous thickening. Tapping such a chest usually causes a severe pain, because the compressed lung cannot expand; hence, interference is contra-indicated, and one should allow it to remain as a filled cavity. The writer knows a patient with such an exudate who is undertaking high mountain climbing.

The height of the exudation will not always determine its amount. If an exudate is well encapsulated, increase or decrease of the amount of the fluid is much more apt to produce an increase or decrease in thickness than in height, changing the intensity of the dulness rather than its extent. As mentioned above, abnormal relations of retraction or of elasticity are responsible for abnormal positions of exudates—*e. g.*, a marked collection beneath or to the median side of the lung. In such a case neither the intensity nor the extent of the dulness affords a clue to the real condition, but only a careful study of the other appearances, such as dyspnea, dislocation of the organs, widening of the thorax, etc. These are all very important in localizing the point for tapping a pleuritic effusion.

Ferber has plainly demonstrated a dulness after injecting 120 c.c. of fluid into the cadaver of a four-year-old child, and after 400 c.c. into an adult. But in the living, as shown in the results of tapping, much smaller quantities of fluid are often demonstrable by light percussion.

Dulness of Hydrothorax.

In general dropsy the dulness due to the transudation of serum in the thoracic cavity corresponds to that of a pleural exudation. Here, too, with small transudations, the boundary line tends to curve downward and forward (see p. 201). But as soon as the fluid has reached a certain amount it becomes more or less movable, and the upper border of dulness then assumes a horizontal level in every position of the body, although at first it may change its position only very slowly (p. 201). This is an important point in making a differential diagnosis between hydrothorax and pleurisy. Hydrothorax is for the most part bilateral, but frequently more marked upon one than upon the other side. If patients have maintained a constrained position upon one side, the hydrothorax is ordinarily more strongly developed upon the lower side, or even may be limited to that side. Hydrothorax does not often produce any dislocation, because it is commonly bilateral, and because there is ordinarily an accumulation of fluid in the abdomen at the same time. Traube's space is generally obliterated.

Increased abdominal contents, pushing up the diaphragm and the lung boundaries, often obscure the diagnosis of a coincident hydrothorax. If the patient sits up the anterior borders of the lungs will be pushed upward, but this is of very little help, for the sitting posture compresses the abdomen, and therefore also elevates the diaphragm. Hence, we must examine either in the standing or lateral posture. To be sure, a slight hydrothorax may be hidden in the erect posture on account of the depression of the diaphragm (p. 174). A moderate hydrothorax in the horizontal lateral posture would furnish a strip of dulness running along the vertebral column, which could easily be determined by comparing it with the other side (see p. 207 *et seq.*).

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Dulness of a Pleural Exudate when Combined with a Pneumothorax.

If a pneumothorax has persisted for some time, a fluid inflammatory pleural effusion commonly results, which may be either purulent or

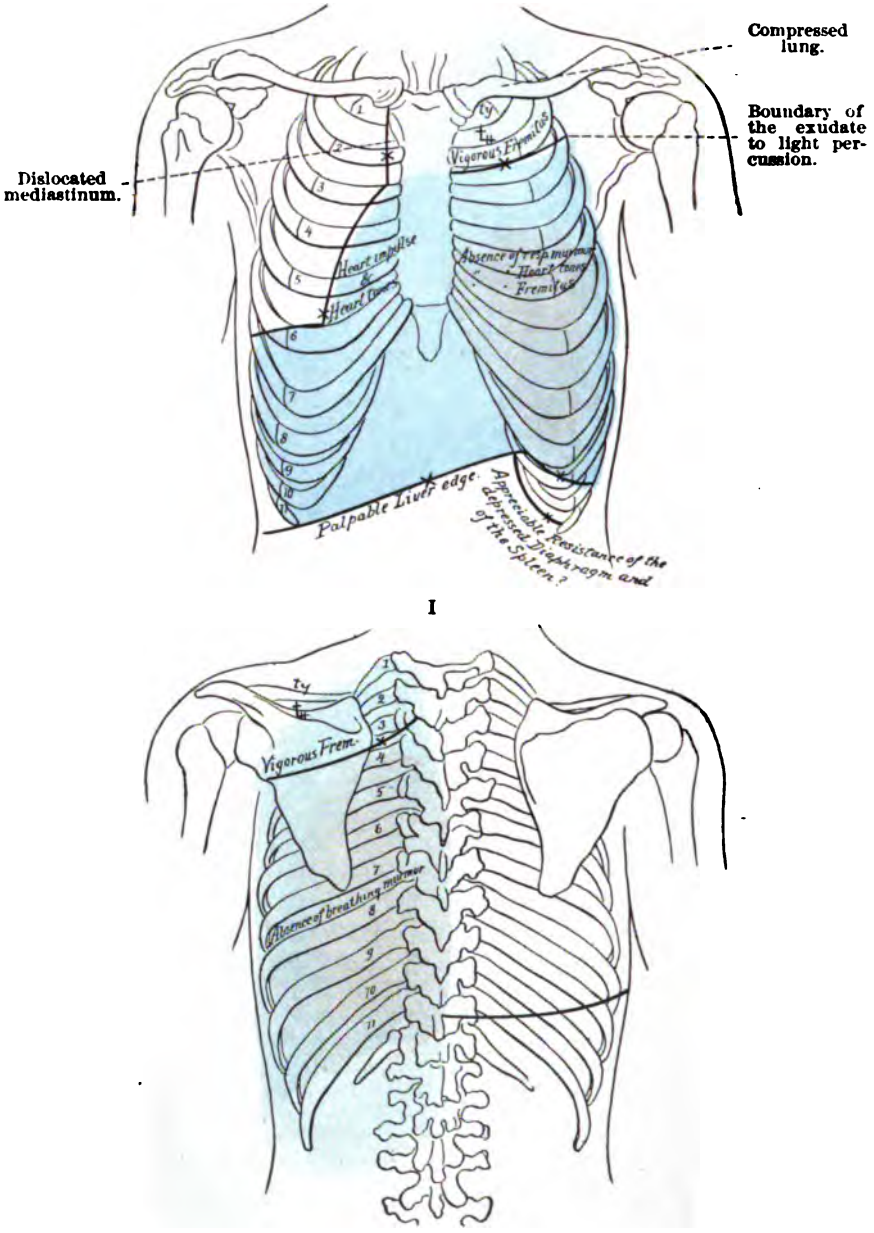

FIG. 97.—Results of examination in a large left-sided pleural exudate. Pronounced dislocation of the heart, of the mediastinum, of the left lobe of the liver, and of the diaphragm. (See p. 348 *et seq.* for explanation of signs.)

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serous (sero- or pyopneumothorax). The fluid then collects beneath the air in the deepest portions of the pleural cavity and furnishes a dull percussion tone. Change of the patient's position produces the most decided mobility of this dulness, and such mobility is very characteristic of this affection. In this condition pressure in the pleural cavity is as a rule, strongly positive, and unless tied down by adhesions the entire lung is separated from the thoracic wall by the air, so that the fluid can perfectly well follow the laws of gravity. The level of dulness is therefore exactly horizontal in every position of the patient. As there is no opposition, any change of posture produces an instantaneous change of the dulness (a contrast to hydrothorax). The fluid does not form a wedge, as in ordinary pleurisy, but all of it is beneath the lung (Fig. 94, I.). The

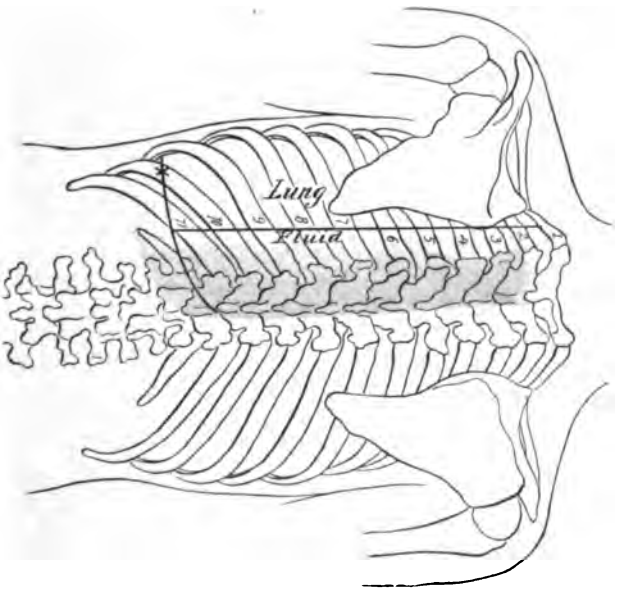

FIG. 98.—Dulness of hydrothorax in right lateral posture.

consequence of this is that a considerable amount must be present before it becomes evident to percussion. In the beginning the exudations are sometimes completely hidden at the bottom of the thorax, especially if left-sided. The high pressure of the pneumothorax depresses the diaphragm and makes it bulge out convexly. Not infrequently we can demonstrate the fluid by means of the succussion murmur (see p. 240) before percussion will show anything. As it increases in size a narrow band of dulness can be made out at the base of the lung, front and back. We are apt to obtain better results from percussion if the patient bends forward, or if he lies upon the well side and we percuss above the spinal column, in the same way as in hydrothorax (Fig. 98). Figs. 99 and 134 show the results of examination in sero- and pyopneumothorax.

If the lung of the affected side is more or less adherent at the onset

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of the pneumothorax, all the relations will be atypical. Even a very small exudation may then be appreciated by percussion. Although the relations of position are atypical, we should still expect a change of position in the dulness of the fluid.

Dulness of Hemothorax.

If blood is poured out into the pleural cavity from traumatism or from the rupture of an aneurism, the resulting dulness will ordinarily correspond to that of hydrothorax. This dulness will usually shift with change of the patient's posture until some inflammation ensues, when encapsulation will very soon fix it. The blood coagulates very slowly (after the lapse of days).

Dulness Due to Infiltration of the Lung (Inflammation; Tuberculosis; Infarction).

An infiltration of the lungs presents a less intense dulness than a pleural effusion, because the bronchi ordinarily remain full of air, and so aid in producing a partially resonant tone. The shape of the dulness is generally different. It is less sharply bounded, because the infiltration gradually projects into healthy lung tissue. In the neighborhood of infiltrations we frequently obtain a tympanitic or hyperresonant note, so that the dulness is often so-called dull tympany (see p. 209 *et seq.*).

In *croupous pneumonia* the dulness is more apt to be located behind and below. Frequently enough it is found at the borders of the lobes of the lung; it may, however, overlap these borders in an irregular way (see Figs. 74, 75, and 76).

In *bronchopneumonic infiltrations* the dulness is situated either behind and below or at the sharp anterior and lateral lung edges, or else in a narrow strip along both sides of the spine.

In *tubercular infiltrations* the dulness is most frequently localized at the apices and at the sharp pulmonary edges, especially at the "lingula" which bounds the superficial cardiac dulness on the left. Miliary tuberculosis generally causes no dulness, but rather a hyperresonant tone (p. 210, 3). Very thickly studded miliary tubercles may, however, produce a diffuse dulness simulating a catarrhal pneumonia (p. 200).

Pulmonary infarctions, if large enough to cause physical signs, commonly occasion a less intense dulness, and generally over the lower posterior portions of the lung.

Dulness from Tumors of the Lungs, of the Pleura, and of the Mediastinum.

There is little characteristic about the dulness of tumors of the lungs and pleura, and this corresponds to their atypical topography. They furnish a marked dulness only when they have reached a considerable size, and then it is apt to be more intense than the dulness of inflammatory infiltrations, because the tumor tissue contains no bronchi filled with air. The more common varieties of intrathoracic tumors arise from the mediastinum. They furnish a somewhat characteristic dulness over the upper end of the sternum; it projects from there laterally into the territory of the lung, where it is generally more or less distinctly separated from the cardiac dulness in the shape of an hour-glass. If these tumors involve the pleura, they are frequently accompanied by pleuritic exudations, with the corresponding physical signs.

Dulness of Pulmonary Cavities.

Tubercular and bronchiectatic cavities of the lung furnish a dull note if the cavity is filled with secretion instead of with air. It is quite characteristic for such a dulness to change into a tympanitic note after copious expectoration. But the dulness generally disappears only in part, because the thickening of the pulmonary tissue in the neighborhood persists.

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Dulness from Pulmonary Edema.

It is ordinarily assumed that pulmonary edema gives rise to a hyperresonant note (see the following page), yet a series of autopsies has convinced the writer that if a pulmonary edema is of long duration, the air in the area infiltrated with fluid becomes absorbed and a very intense dulness results, especially in the postero-inferior portions of the lung. This dulness can be differentiated from the dulness of infiltration by its rapid alteration and by the absence of bronchial breathing, because the bronchi, too, are mostly filled with fluid. Here it is really a sort of fluid or serous infiltration.

Dulness of Atelectasis of the Lungs.

This is in general like the dulness of infiltration. *Obstruction atelectasis—i. e.*, one due to a catarrh and plugging of the bronchus, with subsequent resorption of air from the alveoli—ordinarily furnishes a dulness like that of small areas of infiltration (see above). Later on the atelectasis is commonly transformed into an infiltration. *Compression atelectasis*, from dislocation or enlargement of the heart or from the crowding of the diaphragm upward and the consequent compression of the sharp pulmonary edge, also furnishes a dull tone. This occurs upon a larger scale from fluid accumulations in the pleura, where the part of the lung covered by the fluid, and later that lying above the fluid, is robbed of its air content (see p. 204). Marked cardiac enlargement compresses the lung and often causes a very extensive dulness of the left posterior and inferior portions of the lung. It is frequently wrongly diagnosed as a pleural effusion or pulmonary infiltration.

During the formation of all kinds of atelectasis the tissue is relaxed, so that the normal tone is first transformed into a hyperresonant or tympanitic tone, and finally, on account of the diminution of the air content, into a dulled tone (see p. 155).

Dulness of Pulmonary Retraction.

By pulmonary retraction is understood that chronic change of the lung which results from indurative (shrinking) infiltration and atelectasis. The result of percussion in the retraction associated with pulmonary tuberculosis corresponds essentially to that in tubercular consolidations. The retraction resulting from a pleurisy causes a localized dulness corresponding in shape and extent to the antecedent exudation. If the whole lung was compressed, a diffuse relative dulness of the entire lung will persist. Thick pleuritic adhesions or exudations which remain stationary frequently increase the intensity of the dulness in these conditions of pleuritic shrinking.

Dulness from Diverticulum of the Esophagus.

(See later, Examination of the Esophagus.)

ABNORMALLY LOUD (HYPERRESONANT) AND TYMPANITIC TONES WITHIN THE PULMONARY BORDERS.

We have already described upon pp. 157 and 158 the conditions which may give rise to a hyperresonant or to a tympanitic note over an area characterized normally by a non-tympanitic note.

Hyperresonant or tympanitic notes within the pulmonary borders occur as follows:

1. In *pulmonary emphysema* diffused over the entire lung. The very typical percussion note of emphysematous lungs is called a "box tone." The name is sufficiently descriptive. Some authors consider it merely an abnormally resonant pulmonary note; others, a low tympanitic note.

2. In *relaxation of the pulmonary tissue* from retraction due to a

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limitation of space in the thorax. This occurs in dislocation upward of the diaphragm, in intrathoracic and intrapulmonary tumors, in pleural effusions, in cardiac enlargements, and in pericardial effusions. When the diaphragm is pushed upward the tone may be abnormally loud, and equally so over both lungs. If the relaxation of the pulmonary tissue be localized, as in the other cases mentioned above, the abnormally loud tone will be especially noticeable near the cause of the limitation of space. Thus, above the pleuritic dulness the retracted lung gives an abnormally loud or tympanitic note. In all these conditions the more marked the relaxation of the lung, the more nearly tympanitic this abnormally loud pulmonary tone becomes. If the limitation of space

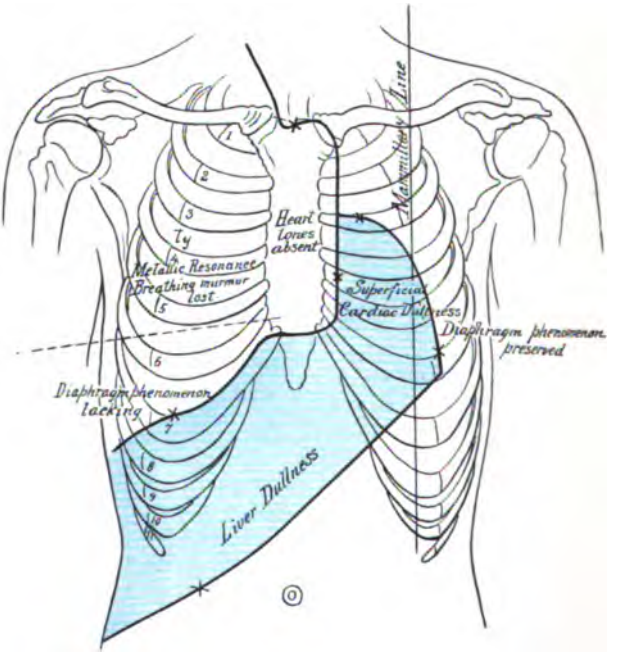

FIG. 99.- Results of percussion in a right pneumothorax—dorsal decubitus (the exudation is therefore not demonstrable above): Loud tone overstepping the territory of the lung; dislocation of the liver, heart, and mediastinum; superficial cardiac dulness restricted at the right.

proceeds still farther, so that the lung is compressed, the tone becomes dull.

3. In *relaxation of the pulmonary tissues from structural changes.* To this type belong diffuse pulmonary edema and the localized inflammatory edema which appears nearby, both as a precursor and as a residuum of the inflammatory processes. Here, too, the normal pulmonary tone becomes first abnormally loud, next hyperresonant, then tympanitic, and finally dull (see above). The relaxation of the pulmonary tissue in beginning obstruction atelectasis, due to the plugging of a bronchus by secretion or by a foreign body, also belongs to this category. Another transition occurs here as well from the hyperresonant to the tympanitic

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tone, and finally, when the atelectasis becomes complete or leads to infiltration, to a dulness.

4. In *pneumothorax* (Fig. 99). In most cases the percussion tone becomes abnormally loud, but only rarely tympanitic. The so-called valve pneumothorax is the most frequent type, and then the air is under such marked pressure that the tone is mostly non-tympanitic (p. 156). It is, however, generally tympanitic in open pneumothorax, where the air exists only under the atmospheric pressure (operative empyema). Another difference between the normal lung tone and the loud tone of a pneumothorax is that the latter projects noticeably beyond the normal lung boundaries, because the diffuse air accumulation penetrates to the complementary pleural sinus. In right pneumothorax the liver dulness is diminished from above, the cardiac dulness from the right (see p. 180 and Fig. 99). In left-sided pneumothorax the splenic dulness disappears and the cardiac dulness is either diminished or, more commonly, also disappears (p. 180). Very frequently distinct dislocation can be observed. Sometimes, though not constantly, the loud tone over a pneumothorax presents a metallic character, which can be appreciated with the stick-pleximeter method of percussion (see p. 157). Again, it may exhibit all sorts of variations in the pitch (see following pages). If the pneumothorax is combined with an inflammatory fluid effusion, and this is the rule when it has persisted for any length of time, the fluid shifts completely with change of the patient's position, and very much more rapidly than in hydrothorax. In every position of the patient we find, then, that the lower part of the thorax is dull; and that above the dulness, separated from it by an exactly horizontal line, is the hyperresonant tone of the pneumothorax (p. 207 and Fig. 99).

5. In *cavities of the lungs* (tubercular, bronchiectatic, and abscess cavities). As a rule, the cavities are at least partly filled with secretion, and they are surrounded by tubercular or inflammatory infiltrated solid tissue, so that percussion over them ordinarily elicits a dull tone (see p. 208). Stronger percussion, especially after expectoration of the secretion, transforms the dulness into a tympanitic tone. A very large cavity containing a great deal of air, or one located very superficially and only surrounded by a thin layer of infiltrated pulmonary tissue, will furnish an abnormally loud tone, and even with weak percussion a tympanitic tone. Smaller cavities (up to the size of several cubic centimeters), and even very large cavities when situated deeply, generally escape demonstration by percussion. The stick-pleximeter percussion method will sometimes, though not often, demonstrate metallic resonance and change of tone height over cavities (see pp. 157 and 158 *et seq.*).

6. Diaphragmatic hernias in the region of the lung, containing intestines, will cause a loud tympanitic tone. They can be distinguished, especially from pneumothorax, by their irregular shape and by the intestinal borborygmus, mostly of metallic character.

7. See p. 691, under Examination of the Esophagus, in regard to the tympanitic note of esophageal diverticula.

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PECULIAR PERCUSSION PHENOMENA OVER THE THORAX.

Metallic Resonance Over the Thorax.

See p. 157 in regard to the origin and peculiarity of metallic resonance.

Metallic resonance is most frequently observed in *pneumothorax* (see the foregoing pages). It is favored by the size of the air space and by the smoothness of the walls, and is most readily demonstrated by the stick-pleximeter method of percussion combined with auscultation. Metallic resonance is, however, not a constant sign of pneumothorax, because it requires a peculiarity in the shape of the air space and a certain degree of tension of the enclosed air. If the air exists under too slight or even under too high a pressure, the metallic resonance disappears. It often requires a certain strength of percussion to make it audible, and not infrequently it can only be appreciated at certain places. In most cases the metallic tone is too weak to be appreciated at a distance—*i. e.*, without coincident auscultation.

The metallic resonance of a pneumothorax frequently disappears after aspiration; or, in case it was not present beforehand, aspiration may bring it out. This is due to the influence of the tension of the enclosed air.

The metallic resonance of a pneumothorax frequently exhibits distinct variations in pitch, depending upon whether we percuss the patient in the sitting or recumbent posture. This is plainly due to the mobility of the fluid in the pleural cavity with change of the patient's posture. A deepening of the tone in the sitting, and an elevation in the recumbent, posture is more frequently noted than the reverse (the so-called Biermer tone change). The height of the metallic resonance varies inversely with the greatest diameter of the air space (see p. 207); therefore we assume that in the former case the diaphragm, under the weight of the exudation in the sitting posture, drops downward. When the tone becomes higher in sitting up, we assume that it is due to the resulting shortening of the air space by the rise of the exudation in the sitting posture. The pitch of the metallic resonance varies in different places; hence, in such an examination we should always percuss the same spot.

In pneumopericardium an abnormally loud tympanitic or non-tympanitic note takes the place of the cardiac dulness, sometimes modified by a dulness from the exudation and sometimes not (see p. 180). A metallic resonance can at times be demonstrated similar to that of pneumothorax. The patient's posture may also influence the loudness and the pitch of this metallic resonance (change of tension of the air, dislocation of the heart and of the exudation).

Pulmonary cavities only rarely furnish metallic resonance, because they do not often fulfil the conditions for its establishment (see p. 158). They are either too small, have too irregular or too thick walls, or are situated too deeply. If a metallic resonance is obtained over a pulmonary cavity, similar alterations in pitch may be brought out; concerning which see pp. 214 and 215 *et seq.*

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Diaphragmatic hernias within the thorax may also give rise to a metallic resonance.

CRACKED-POT RESONANCE OVER THE THORAX (CHINK OF COINS; SPINNING-TOP MURMUR).

(See p. 158 as to its origin.) Cracked-pot resonance may be produced while percussing a normal thorax under entirely physiologic conditions —*e. g.*, over the lungs of a crying child, or, provided the thorax is yielding, over an adult's lungs while he is talking, or during forced expiration with half-open vocal cords. Vigorous percussion, proximity to the trachea, and open mouth all favor the production of this resonance. The noise, under physiologic circumstances, is assumed to arise at the narrowed glottis, because the percussion blow causes the sudden escape of air (see p. 158).

It can also be noted over relaxed or partly infiltrated pulmonary tissue under all those conditions which furnish abnormally loud or tympanitic pulmonary tones—*e. g.*, at the borders of pleuritic exudations and in the neighborhood of infiltrations. Its explanation under these circumstances is not yet entirely clear, whether here, too, it is due to a stenotic murmur (p. 158) arising, as in the crying child, at the glottis, or *in loco*. If the conditions which give rise to the noise even in healthy persons are absent—*e. g.*, speaking, pressure of the half-open glottis, etc.—the writer's experience has convinced him that cracked-pot resonance rarely results from the above-mentioned pathologic conditions (relaxation, infiltration). Cracked-pot resonance has a certain significance in the diagnosis of pulmonary cavities. It is very common, for example, over small superficial cavities (a few centimeters) which communicate with a bronchus, especially upon rather vigorous percussion. It is probably due here to the production of a stenotic murmur at the opening of the bronchus into the cavity. Marked emaciation will favor the production of the murmur. We are inclined to attribute considerable importance to this phenomenon in the diagnosis of pulmonary cavities.

CHANGE IN THE PITCH OF THE PERCUSSION NOTE OVER THE THORAX.

Wintrich's Phenomenon and Williams' Tracheal Tone. —If the patient opens his mouth during percussion the pitch of a tympanitic pulmonary note, or of a metallic resonance over pulmonary cavities, will be raised, while if the mouth is closed the pitch will be deepened. This is *Wintrich's phenomenon*. The patient should breathe naturally, and the note should, of course, be compared during the same phase of respiration, because its pitch also varies with the phase of respiration. (See p. 215, Friedreich's Tone Change.) It is a sign of a cavity, and arises if the cavity communicates with the bronchus and transmits the resonance into the trachea, and from there into the mouth. The pitch in the mouth must vary with its being open or shut, according to the laws of open and closed pipes. If we approach the ear to the patient's mouth we can readily be convinced that the notes' change of

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pitch in reality depends upon an admixture of the percussion tone with the individual tone of the mouth. Hence, Wintrich's change of tone is not absolutely pathognomonic of a cavity. As a matter of fact, we do observe this same sort of tone change in percussing over the supra- and infraclavicular fossæ from infiltration or contraction of the upper part of the lung or from marked compression by pleuritic exudation, because vigorous percussion will readily transmit the vibration through the thickened tissue to the main bronchus and to the trachea. The note which sounds dull to light percussion then becomes tympanitic, and opening and shutting the mouth will evoke the same Wintrich tone change. The name *Williams' tracheal tone* has been given to this phenomenon when due to infiltrated or contracted areas of lung without cavities. If Wintrich's change of tone appears and disappears, depending upon the body posture, we speak of it as the *interrupted Wintrich tone change*, as contrasted with the *simple Wintrich tone change*. Secretion closes the communication between the cavity and the bronchus, and so the sound is not transmitted. This peculiarity will sometimes aid in differentiating the Wintrich phenomena from the Williams' tracheal tone.

Gerhardt Tone Change (Gerhardt's Phenomenon).—If

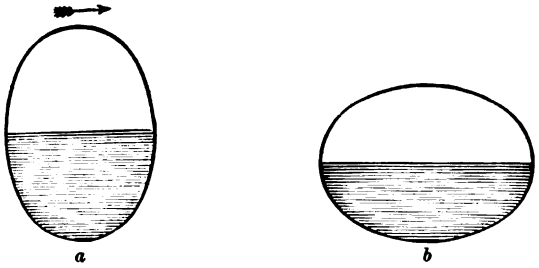

FIG. 100.—Gerhardt's phenomenon.

the pitch of a tympanitic note or of the metallic resonance over a pulmonary cavity changes with the patient's position, we speak of this change as Gerhardt's phenomenon. Fig. 100, *a* and *b*, explains this.

A cavity contains both air and fluid. The fluid is freely movable. The diameter of the part of the cavity containing air is longer in the recumbent than in the erect posture, or *vice versâ*. Hence, percussion will produce a note of deeper or higher pitch according to the patient's position. This would naturally lead us to believe that Gerhardt's phenomenon not only demonstrates the presence of a cavity, but also the direction of its longer axis; in other words, tells us about its shape. From a practical standpoint, however, we should remember that a distinct Gerhardt's phenomenon is rare; that other methods of examination, such as auscultation, are quite as diagnostic; and besides, that slight differences of percussion notes with change of position may be, within physiologic limits, due simply to alteration in the tension of the thoracic wall without any cavity within the chest. As simple a condition as is represented in Fig. 100 is rarely observed upon the autopsy table. The

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cavities have more jagged shape, so that there is more than one long axis to influence the pitch; and besides, the variation of the tension with change of position plays a very important part, because the walls of many cavities adhere to the costal pleura.

Wherever Gerhardt's tone change could be demonstrated exactly as is explained above, the secretion flowing forward in the sitting posture should furnish a dulness at the lower border of the tympany and so clinch the diagnosis.

Friedreich's Phenomenon, or the Respiratory Tone Change.—Inspiration raises the pitch of the tympanitic or metallic note over a cavity and expiration deepens it. This is Friedreich's phenomenon. However, respiration slightly alters the pitch of the note over a perfectly normal lung, so that this phenomenon is not diagnostic of cavities unless it is very marked and perceived at a circumscribed and perhaps suspicious spot. The phenomenon depends upon the variable tension of the lungs in the normal case, and of the cavity-wall in the pathologic case.

Biermer's Phenomenon.—The pitch of the metallic resonance over a sero- and pyopneumothorax is deepened in the erect and raised in the recumbent posture (see p. 212). Biermer's change of tone is in a way identical with Gerhardt's, except that the former author limited his description to the metallic resonance over pneumothorax, and Gerhardt, to the tympanitic note over pulmonary cavities.

COMPARATIVE PERCUSSION OF THE ABDOMEN.

The percussion note over the abdomen becomes more resonant, or louder:

1. From distention of the intestines with gas (*meteorism*). The tension of the intestinal coils is increased and so dulls the tone, but the increased volume of gas seems to overpower the dulness; therefore, the note becomes higher, sometimes deeper, according as the influence of the increase of volume or the increase of tension predominates. The increase of volume tends to deepen the pitch; the increase of tension, to raise it. The more the tension increases, the less tympanitic the note, until eventually it will resemble a hyper-resonant lung tone.

2. From an accumulation of air within the peritoneal cavity, pneumoperitoneum (perforation of stomach or intestine). Here the influence of the volume of air and of the tension acts precisely as in meteorism. If the air accumulation is freely movable it naturally occupies the highest point of the peritoneal cavity and overlaps the liver, sometimes enough to obliterate the liver dulness. If, as commonly happens, an exudation is associated with the accumulation of air in the peritoneal cavity, the inferior portions become dull. This dulness shifts exactly as in pyopneumothorax, and so always presents a horizontal level. The stick-pleximeter method of percussion ordinarily brings out or emphasizes the metallic resonance over an air accumulation in the abdominal cavity.

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The abdominal note becomes dulled:

1. In a diffuse way, from diminution of the gaseous contents of the intestines (starvation, hunger, scaphoid belly of meningitis tuberculosa).

2. In a circumscribed way, from solid or fluid material filling the intestinal coils.

3. From tumors which are situated in the intestines, which lie between the intestine and the abdominal walls, or which, arising from the depths, push the intestines aside. Palpation bounds these much more accurately, so that the only value of percussion is to determine whether the intestine or stomach lies in front of the tumor or not, by the presence or absence of a superficial dulness over the palpable resistance. For this purpose it goes without saying that we must employ as light a percussion as possible, in order to avoid vibrating neighboring or deeper-lying intestinal coils. It is difficult to determine deep dulness over the abdomen, so that percussion has very little value in localizing deep-lying tumors (see p. 196).

4. From a large number of perfectly empty and contracted intestinal coils.[1] These may produce quite an intense localized dulness.

5. From inflammatory infiltrations of the intestinal walls or of the peritoneum. Palpation also appreciates them as resistances resembling tumors. To this group belong the dulness noted in the right iliac fossa in perityphlitis and that of chronic inflammatory and tubercular adhesions. The dulness can sometimes be sharply defined, sometimes not. Light percussion is necessary. Palpation again generally furnishes more accurate data.

6. From fluid effusions between the intestines and the abdominal wall. At a moderately acute stage inflammatory effusions are frequently encapsulated by adhesions, and then furnish a circumscribed, irregularly shaped dulness. Inflammatory serous effusions which are chronic in their development—*e. g.*, tubercular peritonitis, and even acute purulent effusions—are not necessarily encapsulated. We shall obtain from them a more or less intense dulness in the dependent portions of the abdomen, shifting with the movements and forming an approximately horizontal level in every position. This horizontal level of dulness may, however, frequently be interrupted at one or two places, where separate intestinal loops are adherent or where marked tympanites may press them against the abdominal wall. This change in the position of the dulness need not appear instantly in every case, even though adhesions be absent, because the distended intestines are likely to oppose some obstacle to the movement. *Ascites* or *dropsical effusions in the abdomen* (general dropsy, portal stasis) furnish similar percussion results. The dulness is always superficial, so that percussion must be light in order to bound the fluid dulness.

A free fluid effusion in the abdomen must attain considerable dimensions before it produces distinct dulness. F. Müller[2] found in experimenting upon the cadaver that in children one year old 100 c.c. could not be demonstrated; that 150 c.c. gave a relative dulness; and that 200 c.c. give an absolute dulness, shifting with

[1] F. Müller, *Berlin. klin. Woch.*, 1895, No. 13, p. 278. [2] *Loc. cit.*

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change of position. In grown people 1000 c.c. furnished no distinct dulness; but with 1500 c.c. a dulness was perceptible in the dependent portions (lumbar regions); while with 2000 c.c. the dulness reached a hand's breadth and became absolute. He rightly emphasized the greater accuracy of percussion of the living body; and this is in accordance with countless clinical and pathologico-anatomic experiences. The more favorable results in percussing the living depend, the writer believes, on the one hand, upon the greater elasticity of the living abdominal walls, which, therefore, furnish a better localization of the percussion blow; and, on the other hand, upon the fact that during life the movements of the intestines prevent the loss of any fluid between separate coils of intestines.

Very large fluid effusions furnish a dull note over the greater part of the abdomen. But even then a rounded tympanitic area very characteristically persists at the highest point of the abdomen—*i. e.*, the epigastrium—corresponding to the top of the intestinal coils floating in the fluid. Free fluid effusion in the abdominal cavity can thus be differentiated from the equally marked abdominal distention due to cystic tumors. In women such cysts frequently originate in the ovaries; in men, very rarely in the pancreas. With these cysts the dulness is most marked in the middle of the abdomen, because they quickly grow in the middle line, the direction of least resistance, and crowd the intestines backward.

(See the next paragraph in regard to the demonstration of free fluid effusions in the abdomen, with coincident edema of the abdominal walls.)

7. From thickening of the abdominal walls, whether on account of fat-accumulation or of edema. The edema is ordinarily localized in the dependent portions, so that percussion may confuse it with ascites. Palpation of the abdominal walls, and the fact that the dulness does not shift with the changing of the patient's position, will generally prevent a mistake. If the edema is combined with ascites, the movable fluid can sometimes be demonstrated only in the knee-chest position, because the edematous lateral walls of the abdomen furnish so much dulness.

AUSCULTATION.

AUSCULTATION IN GENERAL; INSTRUMENTS.

AUSCULTATION, in its broadest sense, means the examination of the body by the sense of hearing. It should include the appreciation of any sounds the body may furnish, whether heard nearby, at a distance, or with the aid of instruments—*e. g.*, the cough, the voice, etc. In the narrow and more usual sense, auscultation includes only the diagnostic method of listening to the body by means of some specially constructed sound-conducting apparatus or by means of the ear directly applied.

A few experiments and essays antedated the practical listening to the interior of the body. Auscultation was, however, first practically employed by Laënnec. His work upon auscultation was published in the year 1819. In it he not only instituted these methods of examination, but ventured definite conclusions which were based upon several years' study. He also invented the stethoscope. A great number of learned authors have since then developed the scope of auscultation; among

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them notably Skoda, Wintrich, Zamminer, Traube, Bamberger, A. Geigel, Th. Weber, and Gerhardt.

In regard to method, we distinguish between an *immediate* or *direct* and a *mediate* or *indirect auscultation*. In the former the examiner's ear is applied directly to the patient's body; in the latter we listen with the aid of an instrument called the stethoscope.

According to the writer's opinion, the essential for a good stethoscope is not, as was contended by Laënnec, and since his time by many others, its capability of intensifying the tone vibrations. Unless an examiner is hard of hearing, stethoscopes which transmit to the ear vibrations intensified by resonance are by no means the best, because they are bound to alter the tone to some degree. Examples of this type are the instruments of Voltolini, Hüter, and König. In that of Hüter, the opening of the sound funnel is closed by a simple rubber membrane; in that of König, by two rubber membranes with an air-tight space between, whose form can be changed by blowing upon it into a lens-shaped resonating chamber. The microphone, devised to intensify the sounds, has not thus far proved practical.[1] The requisite, then, for a good stethoscope is not so much the capacity to magnify as the capacity to transmit the tones to the ear at least not markedly weakened. This essential is fulfilled by practically all the countless stethoscopes met with in practice, in the use of which we are sure that we hear only what is appreciated by the ear. The stethoscope might therefore be considered superfluous and only immediate auscultation practised. But it has certain advantages, the most important of which is that it enables us to auscultate an exactly circumscribed spot to the exclusion of the neighborhood; in other words, to exclude the sounds of adjacent body parts. In cardiac diagnosis this is absolutely necessary. On the other hand, immediate auscultation offers certain disadvantages in the direction of convenience and cleanliness; besides, in the supraclavicular it is impossible, in the infraclavicular region very difficult, to apply the ear directly. Direct contact can, of course, be avoided by interposing a soft towel between the patient's body and the examiner's ear.

Stethoscopes have been made in the most varied forms and out of the most varied materials. They ordinarily consist of a hollow stem of wood, hard rubber or metal, with an enlarged tip slightly funnel-shaped at one end, and an ear plate, with a hole in the middle, fastened perpendicularly to the other end of the stem.

[1] The so-called *phonendoscope*, recently so extensively advertised and discussed, is in reality nothing more than the old resonance stethoscope, which long ago fell into disuse on account of the alteration of the tone. Its popularity is probably due to the name and to the appearance of the instrument. Like the microphone, its principle of construction is based upon the false assumption that the main trouble in auscultation consists in the difficulty of appreciating the sounds. Anyone who has had much experience will complain rather of an embarrassment of riches in auscultation. The real difficulty lies in judging the significance of the sounds, and this difficulty will not be made any less by using an instrument which magnifies unimportant or artificial overtones.[2]

[2] [Despite Prof. Sahli's objections to the resonating stethoscope, its use in America is so universal that we believe a word in its support might be in place here. The choice of a stethoscope is, after all, quite a matter of custom and convenience. Many good stethoscopes are made in America, especially the Tiemann and Ford models, the favorites in New York City, and the Gannett model, so often selected by clinicians in Boston and Baltimore. The resonance is decided with any of these instruments, but we have not found that it seriously detracts from their practicability. The Bowles' model, a type of phonendoscope, is warmly recommended by many of the younger clinicians. In our opinion it has many advantages, although we still depend upon the other models for most purposes. One especial advantage is that it can often be used when another type of instrument fails—*e. g.*, when a patient is recumbent and too sick to be moved. Then, by slipping it under the patient's back, we can often obtain an approximately accurate idea of the breathing over the bases of the lungs behind. —Ed.]

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The funnel-shaped end is placed against the skin of the patient, and the ear of the examiner is applied to the ear plate as smoothly as possible. The ear plate is in some instruments concave, in others convex. For most observers the former shape seems to adapt itself better to the ear. To carry the instrument conveniently in the pocket, it is desirable to be able to separate the ear plate from the tube by unscrewing or in some other way. There is no advantage in substituting for the ear plate an olive-shaped peg adapted to fit into the external canal of the ear. Experiments have shown that the canal which is bored through the tube in the axis of the stethoscope should have about the same diameter as the entrance of the external ear. In order not to cause the patient pain, the edge of the funnel end must be well rounded. The width of the funnel varies with different instruments. A wide funnel has the advantage of receiving sound waves over a large area, and therefore, in general, of transmitting the sounds more powerfully to the ear. On the other hand, a narrow funnel has the advantage of better isolating the sounds and of modifying them less by resonance. It is of some advantage, therefore, to possess a stethoscope with a wide funnel at one end and a narrow one at the other, in either of which the ear plate can be inserted by means of a conical peg. The funnel should not be too long. The long bell-shaped funnel ends of many hard-rubber stethoscopes really confuse the respiratory murmurs, and often in a very marked way, by resonance. The length of the cylindric part of the instrument is of minor importance.

Flexible stethoscopes transmit the sound from a funnel through a tube or tubes to the ear of the observer. This is the principle of most of the above-mentioned resonance stethoscopes. They are very convenient, especially the biauricular, and furnish a very loud tone; but the resonance is confusing, and the slightest movement in handling gives rise to perplexing murmurs. The same difficulty occurs even in the double-eared Camman's stethoscope (recently devised and warmly recommended by Piel) and in similar instruments in which accompanying noises are hindered by making the sound-conducting tube partly quite firm, partly of very dense tubing. After many attempts with all these instruments, the writer must recommend for practice the ordinary single-barrel stethoscope, for he is convinced that in a method of examination which is already difficult itself, it is not advisable to complicate the affair for the sake of external convenience and to use instruments which possess such essential faults. Flexible stethoscopes are absolutely necessary only for auscultating one's own body.

The Technic of Auscultation.—The stethoscope should be applied very carefully, so that the edge of the funnel makes an air-tight connection with the skin; the ear should be lightly applied to the ear plate without leaning or propping the head upon it. Tilting the stethoscope should be avoided, because it is very painful for the patient and prevents correct auscultation. The best way is to hold the stem with the thumb and forefinger lightly applied near the funnel end, so that the slightest movement can be readily felt and corrected.

AUSCULTATION OF THE RESPIRATORY ORGANS.

The immediate, as well as the mediate, method should be used in auscultating the lungs, for some sounds, such as faint bronchial breathing, are better appreciated by applying the ear directly to the chest, and an exact localization is of less value than in cardiac diagnosis.

It is exactly in these cases (in general rare) where the tones are so weak as to be doubtful that the tone-increasing or resonating stethoscope is useless, for it makes the doubt still greater by its modifications of the sounds. We will mention in a special section the sources of error which may arise from involuntarily moving the stethoscope while auscultating the lungs.

It is advisable to auscult during normal, and then during exaggerated, respiration, and over any suspicious places both during and after cough, as well as to listen in such places to the loud and the whispered voice.

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THE NORMAL VESICULAR RESPIRATORY MURMUR.

A characteristic sighing or sipping noise, perhaps resembling a very soft "f," is heard over a healthy lung throughout inspiration; during expiration there is either no sound, or at the beginning a very short, faint noise. The latter is rather difficult to describe; it is apt to be lower pitched than the murmur during inspiration, and is slightly rustling, or over certain places slightly blowing, in character. Its duration corresponds normally to less than a fifth of that of the inspiratory murmur. These two sounds constitute the normal breathing murmur, the so-called vesicular breathing. Its presence shows us that the lung parenchyma at the spot of auscultation not only contains air, but also breathes—that is, during inspiration air enters the alveoli. The inspiratory murmur is evidently the essential characteristic of vesicular breathing.

Numerous theories have been suggested in explanation of this vesicular breathing, but as yet there is no one definitely accepted. Laënnec, the inventor of auscultation, assumed that vesicular breathing was caused by the rubbing of the inspiratory air stream against the walls of the fine bronchi or infundibula. But if friction exists it must act, not between the walls and the air, but between the outer layer of air, which rests nearly motionless against the walls, and the more central actively moving current. Laënnec's theory, however, did not lay any emphasis upon the frictions taking place at the outer boundary of the air stream, and might be stated to-day by saying that vesicular breathing is the acoustic expression of the friction caused by the entry of air into the pulmonary parenchyma. It has also been objected that the current of air entering the bronchi during inspiration is not rapid enough to cause friction noises.

Baas has advanced a more recent theory, which many authors believe disproves Laënnec's. Vesicular breathing he regards as a modification of the blowing noise arising in the larynx and trachea during respiration, transmitted through the bronchi to the interior of the lungs, and thence through the air-containing lung to the observer's ear. The following experiment was made in proof: An inflated lung was placed upon the larynx of a living man, the laryngotracheal murmur was auscultated through this lung, and it was expected that the blowing murmur would be transformed into a sipping vesicular breathing by the air-containing lung. The author has never heard anything but a weakened blowing tracheal murmur in performing this experiment. It is, in fact, difficult to imagine how the tracheal murmur, with an expiratory portion the equal of or stronger than the inspiratory portion, could be both qualitatively and quantitatively modified by the interposition of an air-containing lung. Nor does clinical evidence support Baas' theory, for, as soon as a bronchus is plugged by secretion or by a foreign body, no vesicular breathing can be heard over the pulmonary area supplied by that bronchus until the bronchus becomes free again. This surely proves that inspiratory filling of the lung with air is necessary to produce vesicular breathing. The pathologic modifications of vesicular breathing similarly disprove the Baas theory—*e. g.*, we hear an intensified vesicular

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breathing over circumscribed areas of lung when for some reason the breathing is more vigorous (vicarious); and, on the contrary, a localized diminished breathing wherever for any reason the corresponding lung area is limited in its movement (through lack of room or adhesions). Again, despite the increase of the laryngeal breathing noise in laryngeal stenosis, diminished vesicular breathing is heard over the lung. Further, the thicker the layer of lung, the more intense is the vesicular breathing, although according to this theory it should be diminished. Finally, systolic vesicular breathing, a respiratory murmur synchronous with the cardiac systole and not in the least dependent upon a laryngotracheal breathing murmur, speaks with certainty against the Baas theory, and decidedly favors the idea that vesicular breathing depends upon the pulmonary excursion. The writer considers these arguments sufficient to discountenance Baas' theory.

Now, after all this critical discussion, what is the real explanation of vesicular breathing? In the writer's work upon the origin of vesicular breathing he demonstrated upon a man with congenital fissure of the sternum[1] that vesicular breathing arises from inflation of the lung tissue, even with the exclusion of every laryngotracheal murmur. This man had a hernia of the lung in the region of the sternal fissure which protruded externally to a marked degree when the patient strained—*i. e.*, increased his intra-abdominal tension. When this pulmonary hernia was auscultated at the same time that the patient strained, the most distinct vesicular breathing was heard, which was due to an expiratory filling of the pulmonary alveoli, and, since the laryngotracheal murmur was excluded by the closure of the glottis, we obtained a certain proof that the murmur of vesicular breathing is due to the local inspiratory movements of the pulmonary parenchyma. The writer has recently had an opportunity to make a similar examination of the pulmonary apices of an emphysematous subject, which became inflated like balloons when the patient strained, and over which could be heard distinct vesicular breathing. In these examinations he could not determine whether the vesicular murmur was produced by the distention of the pulmonary tissue or by the friction in the interior of the inspiratory air stream in the smallest bronchi and alveoli. That is a fine point of no great significance. Stretching of the lung tissue may cause the murmur. The distention of separate lung alveoli doubtless does not happen at the same instant, and may produce a series of vibrations lasting over the entire inspiratory period, which together may cause the vesicular murmur.

To explain the normal brief expiratory murmur we must assume either that the weak remnant of the laryngotracheal breathing murmur is transmitted from the bronchi, or else that the expiratory murmur arises like the vesicular inspiration, locally, through movements in the lung. If the expiratory murmur has a plain blowing character like that of the laryngotracheal murmur, the former hypothesis may be entertained. It should then be classed under the so-called physiologic bronchial breathing (see p. 222 *et seq.*). If this blowing character is

[1] *Correspondenzblatt für Schweizer Aertze*, 1892.

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lacking, the expiratory murmur probably comes from within the lung, especially because expiration, just as inspiration, may be modified by local lung changes. The prolonged expiration in catarrh (see p. 225 *et seq.*) can be explained only by assuming such a localized origin. The elastic retraction of the lung in the beginning of expiration must be strongest and quickest; hence, the expiratory murmur is normally to be heard only at the very beginning of expiration.

Systolic vesicular breathing (mentioned above) is occasionally to be found both in healthy and in diseased individuals. Its origin is unknown; it has no pathologic significance, is heard only in the neighborhood of the heart, in slightly marked instances merely as a systolic accentuation of regular vesicular breathing. It certainly depends upon variations of the intrathoracic negative pressure connected with the systolic diminution in the size of the heart (meiocardia). Many so-called accidental heart murmurs (see later) are nothing more than systolic vesicular breathing.

PHYSIOLOGIC BRONCHIAL (MIXED) BREATHING MURMUR.

Over certain areas of the normal lungs the respiratory murmur sounds bronchial. Such a murmur corresponds in most essentials to the laryngotracheal breathing heard over the larynx (p. 220). In contrast to the vesicular, bronchial breathing presents a blowing character and approaches to a definite pitch. We can reproduce the sound quite perfectly by vigorously inspiring and expiring with the mouth fixed as if about to pronounce the syllable "ha." We can simulate different pitch by changing the position of the mouth. In physiologic bronchial breathing expiration lasts longer and is more strongly accentuated than inspiration, exactly the reverse of the relation between them in vesicular breathing. The rima glottidis is narrowed during expiration, and that is a sufficient cause for an expiratory stenotic murmur or for an accentuation, as well as for its prolonged duration.

Physiologic bronchial breathing is nothing more than the laryngotracheal murmur which originates at the upper air passages and is transmitted through the bronchi to certain areas over the lungs. Its intensity varies considerably in accordance with the individual peculiarities of acoustic transmission. In many men the laryngotracheal murmur is audible only over the neck, while all over the lung pure vesicular breathing can be heard. Physiologic bronchial breathing is most frequently audible anteriorly over the superior portions of the lungs, and posteriorly in the interscapular space. This is due to the location of the trachea and great bronchi. The right bronchus is wider and a more direct continuation of the trachea than the left; hence this breathing is naturally more evident upon the right side and sometimes also over the upper part of the sternum. Vesicular breathing is almost always to be heard at the same time with the physiologic bronchial breathing; but if the respiration is especially faint, then the physiologic bronchial breathing will alone be heard—*e. g.*, a thick thoracic wall renders the appreciation of the vesicular breathing difficult. A so-called *mixed breathing* is, however, the general rule—*i. e.*, during inspiration

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the vesicular, and during expiration the bronchial, character predominates. *Forced breathing, dyspnea, thinness of the thorax, any changes in the larynx and trachea which favor the production of a strong stenotic murmur* (compression of the trachea from the side by a goiter,[1] and the like)—all these will intensify physiologic bronchial breathing. A suspicion of physiologic bronchial breathing may very rarely be heard all over the lung, but more frequently along the spine and over most of the sternum.

Physiologic bronchial breathing may depend in some persons upon certain peculiar positions of the mouth. It may be considerably influenced, intensified, diminished, or even caused to disappear by altering the position of the mouth.

ALTERATIONS OF VESICULAR BREATHING UNDER PHYSIOLOGIC AND PATHOLOGIC CONDITIONS.

INCREASED (PUERILE) AND WEAKENED VESICULAR BREATHING.

The intensity of the vesicular breathing varies with the depth of the respiration and with the spot auscultated. It is weaker at the apices and borders, where the pulmonary layer is thin, than over the thick parts of the lung. Thick thoracic walls also weaken it. Children present an exceptionally loud and strong vesicular, so-called *puerile* breathing, with which physiologic bronchial breathing is often especially plainly mixed. Beginners are apt to mistake it for a pathologic phenomenon.

Pathologic increased vesicular breathing is best designated as *sharp* or *increased vesicular breathing.* The writer emphasizes these two adjectives because the expressions *sharp* or *increased* vesicular breathing and *rough* vesicular breathing, though fundamentally different, are often used synonymously. If ordinary vesicular breathing is represented by the sound of a soft *f*, increased vesicular breathing roughly corresponds to the consonants *ff*. The physiologic increased breathing (mentioned above) must first be taken into account before we decide that increased vesicular breathing is pathologic.

Pathologically increased vesicular breathing depends most frequently upon a *catarrh of the finer bronchial tubes.* The latter are narrowed by the swollen mucous membrane, and this stenosis presumably causes the increased breathing. This explanation would naturally support the friction theory of vesicular breathing.

Another cause for pathologically increased vesicular breathing is the more active breathing of a certain portion of the lung. This happens wherever a lung part is retracted or relaxed, and is generally accompanied by an abnormally loud or tympanitic percussion note (see p. 210). Thus, we frequently hear increased vesicular breathing in *the first stage of croupous pneumonia,* in *the neighborhood of infiltrations,* or *in the neighborhood of affections of the thoracic cavity which limit the space,*

[1] In such a case the expression "physiologic bronchial breathing" signifies merely that the phenomenon does not depend upon any pathologic changes in the lung.

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etc. In pleurisy with effusion and in pneumothorax the respiratory murmur is diminished or absent over one-half of the thorax, despite the pronounced retraction of the lung. But over the other chest-half the breathing is *increased* vicariously. The same condition is often noted in pneumonia and in pulmonary tumors.

Increased vesicular breathing is of special importance in demonstrating multiple small infiltration areas which furnish no other auscultatory or percussion signs. The increase depends partly upon the retraction and relaxation of the neighboring pulmonary tissue, partly upon the accompanying localized catarrh. For this reason increased vesicular breathing localized over one apex is an important sign for recognizing incipient tuberculosis, and especially so because at the apex, where tuberculosis most frequently makes its first appearance, vesicular breathing is normally weak.

Weakness, diminution or absence of vesicular breathing is observed under manifold conditions. Careful attention should be given to the physiologic variations before we can assume any pathologic processes. Any cause which limits the inspiratory distention of the pulmonary parenchyma will diminish and finally abolish the vesicular breathing—*e. g.*, any obstacle to respiration situated in the larger air passages. If the obstacle is located in the larynx or trachea, the respiratory murmur will be diminished over both lungs. If located in one bronchus, it will be diminished over the entire pulmonary territory supplied by that bronchus. This furnishes us with a means of estimating the degree of laryngeal stenosis in croup and of determining any extension of the croupous process down into one or the other bronchus. A localized diminution or abolition of the vesicular breathing is important for estimating the position of a foreign body in the bronchus.[1] The respiratory murmur is diminished in certain forms of catarrh which cause an exceptionally marked stenosis of the affected bronchi. The murmur is abolished in obturation atelectasis.

Even if the air passages are free, diminished breathing will be observed when any sort of mechanical obstruction prevents the normal alveolar distention—*e. g.*, firm pleural adhesions which limit the lung excursions. Multiple small infiltrations often diminish or abolish the vesicular breathing because, to a certain extent, they make the pulmonary area between them, which is still pervious to air, stiff and nonexpansive. Increased vesicular breathing is, however, more commonly heard over multiple small infiltrations.

Such a localized diminution of the respiratory murmur, whether depending upon a circumscribed catarrh stenosing the bronchi, or upon fixation of the lung by small areas of infiltration, or by pleural adhesions, is very important in diagnosing circumscribed tubercular areas. Sometimes, though rarely, even extensive tubercular infiltration does not show the expected bronchial breathing, but diminished vesicular breathing. This peculiarity of many tubercular infiltrations, as contrasted with other kinds of pulmonary consolidation, can be explained

[1] [The *x*-ray will often aid here. - ED.]

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by the frequent narrowing or contraction of the bronchial lumen in tuberculosis.

Diminished breathing is observed in pleurisy over the affected side even at the places where the lung lies against the thoracic wall, because either the exudation or the pain limits the excursions of that side.

An emphysematous lung makes very slight excursions, on account of its permanent inspiratory position; hence the respiratory murmur is frequently diminished. But a coincident catarrh of the bronchi would tend to intensify the respiratory murmur, and so the final result will naturally depend upon which factor is in excess. Diminished breathing is one of the cardinal symptoms of *pleurisy*, *pneumothorax*, and *pulmonary tumors*, for two reasons: In the first place the excursion of the affected side is limited; and in the second place the transmission of the breathing to the examiner's ear is impaired by the interposition of fluid or solid tissue.

VESICULAR BREATHING WITH A PROLONGED EXPIRATION.

Normally we hear only a faint murmur or none at all over the lung during expiration. But under some circumstances the expiratory murmur may be prolonged until it lasts even longer than inspiration.

Prolonged expiration is a frequent, though not constant, accompaniment of increased vesicular breathing. It occurs in bronchitis, probably because the swollen mucous membrane opposes an obstacle to the respiratory current, and produces a stenotic murmur with expiration. Expiration, as is well known, is less powerful and slower than inspiration, and the stenosis slows it still more. If the catarrh is localized at certain places, the prolonged expiration will also be a local phenomenon. The prolonged expiratory murmur in the catarrh of emphysema and in asthmatic attacks is much more extensive, being diffused over the entire lung. In such cases the entire expiratory movements of the thorax are slowed (p. 90 *et seq.*). Prolonged expiration is therefore a symptom of catarrh. If it is localized, it has the same significance in the diagnosis of diseased areas (infiltration, tuberculosis) as has increased or diminished vesicular breathing (pp. 223 and 224).

ROUGH OR IMPURE AND COG WHEEL VESICULAR BREATHING.

Rough vesicular breathing is an impure, slightly uneven murmur heard during inspiration, as if strange accompanying noises were admixed with the normal vesicular murmur. In order to prevent confusion with sharp or increased vesicular breathing (p. 223), the term impure breathing is more fitting. Increased breathing is exquisitely pure and generally very intense, whereas rough breathing is more frequently weak and faint.

Impure as well as increased breathing is a sign of bronchial catarrh, and may or may not be accompanied by prolonged expiration. Either the partial impermeability of the bronchi produces unequal respiratory excursions of the lung area in question or else accompanying noises derived from the presence of secretion complicate the pure vesicular

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murmur. If these accompanying noises can be plainly isolated, we call them *râles* (see p. 232), but if they remain indistinct and blended, the vesicular breathing becomes impure or rough.

The *so-called cog-wheel respiration* is somewhat like impure breathing, but is characterized by jerky intermittent pauses in the inspiratory murmur or by accompanying noises which plainly separate the individual portions of the murmur from each other. In contrast to impure breathing, these individual portions retain their smooth sighing or sipping character. The acoustic peculiarities of cog-wheel respiration convince us that it is caused by the air current being forced into the alveoli with effort and intermittently overcoming some obstacles. Cog-wheel respiration localized over a definite area of the lung is also a sign of catarrh, and so it is natural to suppose that in this case this intermittent obstruction to the respiration is due either to valve-like swellings of the mucous membrane or to accumulations of secretion which must be pushed aside by the air current. Hence its relationship to impure or rough breathing. Naturally cog-wheel inspiration may be an increased or diminished breathing and may be associated with prolonged expiration. Sometimes the expiration is also cog-wheel in type.

Hensen[1] has called attention to the fact that in many cases of cog-wheel breathing the intermissions are synchronous with the pulse. He explains this phenomenon by the supposition that hyperemia of the lung is responsible for this form of cog-wheel breathing. This pulsating character, however, may be explained equally well by the previous statements if we bear in mind that every systole of the heart produces a negative variation of the intrathoracic pressure and, consequently, accelerates the inspiratory air current.

There is another kind of cog-wheel respiration not at all dependent upon the pulmonary or bronchial condition, but upon uneven or intermittent action of the inspiratory muscles. This occurs by no means rarely in partial paralyses and in fatigued conditions of the respiratory muscles. It can be distinguished from the form described above by its more even distribution over the entire lung.

PATHOLOGIC BRONCHIAL BREATHING.

Pathologic bronchial breathing is a blowing murmur heard over morbid areas, and is practically identical with the sound we have already described under the term physiologic bronchial breathing—*i. e.*, with a laryngotracheal murmur resembling the syllable "ha." From experience we have learned that it is to be heard wherever the pulmonary parenchyma is airless (whether on account of external compression or on account of infiltration), wherever there are pathologic pulmonary cavities which freely communicate with the bronchus, and wherever the bronchi themselves are dilated, either diffusely or in the form of a sac.

One of the oldest explanations of the origin of bronchial breathing over solid portions of the lung assumed that the vesicular breathing disappeared and that the laryngotracheal murmur was transmitted more

[1] *Arch. f. klin. Med.*, 1902, vol. lxxiv., p. 237.

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plainly to the surface of the thorax through the thickened lung parenchyma than through the air-containing tissue. But this explanation is not tenable, because we can prove that a solid organ does not transmit this murmur as well as does the air-containing lung. However, although the hepatized tissue itself does not transmit the laryngeal murmur better than the normal lung, nevertheless the air-containing bronchi incased in a solid tissue are better conductors than when incased in air-containing tissue.

This modified explanation is generally accepted as correct, but it will not hold good in every case, for in pneumonia bronchial breathing is heard not only over the infiltrated area, but also in its vicinity and upon the opposite (healthy) side in the neighborhood of the spinal column. With such a result we sometimes erroneously diagnose a double pneumonia, and learn at the autopsy that the bronchial breathing must have been transmitted from the affected side. This transmission of bronchial breathing shows how imperfect the preceding theory is, since it shows

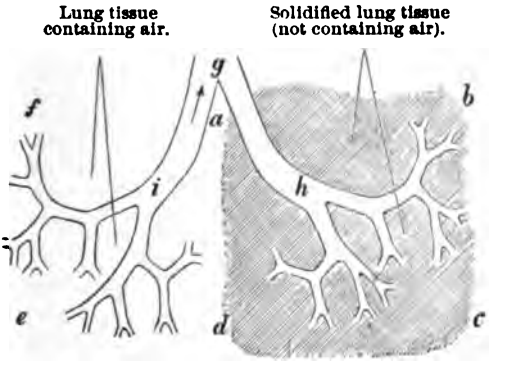

FIG. 101.

that bronchial breathing can be well transmitted in air-containing tissue. Then, if the bronchial breathing is really as intense in the normal bronchi as in the infiltrated tissue, how can we explain why in sound lungs it should not be able to reach the examiner's ear? The vesicular breathing certainly does not conceal the bronchial breathing, because both in transmitted bronchial breathing and also under other circumstances (mixed breathing, see p. 231) we can appreciate at the same time both a bronchial and a vesicular murmur. Personally the writer can explain transmitted bronchial breathing only by assuming that the infiltrated lung not only transmits the laryngeal murmur to the surface through the bronchi, but magnifies it. It is evident that such an increase in a portion of the lung immovable on account of the infiltration would be produced best by resonation or consonation (Skoda). The variations observed in the pitch of bronchial breathing (p. 229) show that real resonating phenomena can appear in the bronchi of infiltrated portions of lung. This question of resonance will be discussed under Consonating Râles.

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Fig. 101 explains another cause of the increase of bronchial breathing over infiltrated areas. Here *a, b, c, d* represents an infiltrated, *a, f, e, d* a non-infiltrated, bronchial area with the tributary bronchi *g, h* and *g, i*. As a consequence of the infiltration the air current between *h* and *g* ceases, whereas that from *g* to *i* persists, so that at the point *g* the current conditions are decidedly altered. The current of air *i, g* blows over a quiet column of air and might very likely produce bronchial breathing similarly to the noise made by blowing over the opening of a key.

Pathologic bronchial breathing is sometimes, although rarely, heard louder during inspiration, and not, as is the laryngeal murmur, during expiration. This fact, it seems to the writer, argues in favor of the latter theory of the origin or increase of bronchial breathing, and cannot be explained by mere conduction and resonation.

The result of this discussion should lead us to conclude that better transmission of the laryngotracheal murmur is not sufficient to explain the presence of pathologic bronchial breathing over pulmonary consolidation, but rather that the laryngotracheal murmur comes much better to the surface in solid tissue with open bronchi, and that in the thickest portions it is even intensified.

There is no appreciable acoustic difference in the bronchial breathing whether the pulmonary consolidation consists in an infiltration of the alveoli with inflammation products, or in an atelectasis from compression of a pleuritic exudation, pneumothorax, hydrothorax, or pericardial exudation. Naturally this compression of the lungs must be limited to the alveoli alone and not involve bronchi, at least those of any size. For as soon as an effusion increases enough to compress the larger bronchi, the bronchial breathing, which is frequently heard with difficulty on account of the interposition of the effusion, becomes weaker and weaker and finally disappears.

Obturation atelectasis, due to the plugging of a bronchus and a consequent resorption of air from the alveoli, prevents the production of bronchial breathing, and in spite of the thickness of the pulmonary tissue and of the consequent dulness no respiratory murmur can be heard unless transmitted from the neighborhood.

Even very tiny areas of consolidation too small to dull the percussion tone may cause bronchial breathing. This shows how valuable a sign it is in diagnosis, and is also another argument against the decided value of the transmission theory of bronchial breathing.

Bronchial breathing is also heard over air-containing cavities which communicate with the bronchi, and over dilatations of the bronchi themselves. Here it is probably due to a better transmission of the laryngeal murmur, especially if the cavity or the bronchiectasy lies superficially. Besides, such cavities are almost always surrounded by infiltrated tissue, so that resonance contributes considerably to the increase of the laryngeal murmur. Finally, we must not forget that under some circumstances a cavity itself breathes—*i. e.*, a respiratory current of air streams through

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the entrance of the cavity into the bronchus and back again. And this would cause a blowing respiratory murmur just as in the larynx.

Sometimes it requires very deep respirations to make the bronchial breathing audible. Again, closure of the bronchus—*e. g.*, by secretion—will interrupt the bronchial breathing. Hence, sometimes after coughing the bronchial breathing will reappear. A good device for bringing out faint bronchial breathing, if a patient cannot be persuaded to breathe deeply, consists in having the patient count aloud as long as possible during one respiration, while the examiner auscultates. The next inspiration will be a maximum. This method has also the advantage of demonstrating bronchophony (see p. 241). The ear applied directly to the chest will sometimes appreciate weak bronchial breathing better than with the interposition of the stethoscope (p. 219).

DIFFERENT KINDS OF PATHOLOGIC BRONCHIAL BREATHING.

Bronchial, far more than vesicular, breathing has a definite pitch (p. 222), which may vary quite decidedly. These variations can easily be reproduced by fixing the mouth in the position for saying the syllables "ha," "he," "hi," "ho," "hu," and then inspiring and expiring.

The pitch of bronchial breathing depends in certain cases upon the conditions of resonation, and with them upon the width of the bronchi or of the cavities. But up to the present time, except perhaps in distinguishing so-called amphoric breathing, the pitch has not occupied an important place in diagnosis. By *amphoric* or *cavernous* breathing we mean a very deep and soft and generally not very loud bronchial breathing, heard especially over large cavities (lung cavities and pneumothorax). Besides its low pitch it has a very characteristic metallic resonance, apparently due to the overtones from the resonance in the cavity. Amphoric breathing is, therefore, sometimes associated with a metallic percussion tone. We may reproduce it by whispering the syllable "hu" or by blowing over an empty jar; whence the name. Provided that we do not call any deep bronchial breathing *amphoric* (a frequent mistake), amphoric breathing is a pretty safe sign of a cavity. The metallic resonance accompanying it is the essential guide. Like metallic percussion, amphoric breathing arises for the most part only over cavities which are at least 6 cm. in diameter (according to the ordinary estimate); but it is sometimes found over smaller cavities.

The pitch of amphoric breathing—*i. e.*, of its metallic resonance—may vary in accordance with laws similar to those governing the metallic percussion resonance. (See the paragraphs upon the Percussion Tone Change, p. 213 *et seq.*)

Metallic breathing can sometimes be distinguished from amphoric breathing. It is a murmur with high metallic overtones and without the deep basal tones. It has no especial significance different from that of amphoric respiration.

Amphoric or metallic respiration may rarely be heard over pulmonary consolidation, even without the formation of a cavity. We do not know the explanation.

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The respiratory murmur may in rare cases assume an amphoric or metallic character on account of the resonation set up in neighboring physiologic air-containing cavities, such as the stomach or the distended intestines.

Amphoric respiration may be heard over a pneumothorax from resonation if, as is most common, it is a closed, or valve, pneumothorax.

In marked dyspnea certain positions of the mouth may add an amphoric resonance to pathologic or physiologic bronchial breathing. But such amphoric breathing can be appreciated at a distance, and, besides, is distributed exactly like physiologic bronchial breathing, so no confusion need result.

METAMORPHOSED BREATHING MURMUR.

Metamorphosed breathing is essentially bronchial in type. In the more common form inspiration begins sharp, blowing and bronchial, gradually becoming much softer, and sometimes ending in amphoric breathing. In another variety the pitch of the bronchial element changes during inspiration or during expiration or during both. Either type, if heard continuously, is a practically safe sign of a cavity. Probably after the inspiration has lasted for a certain time it distends the cavity and its orifice enough to modify the pitch of the respiratory murmur. A similar metamorphosed breathing murmur might arise if the inspiratory current pushed aside the secretion of a partially occluded bronchus. Laënnec's "souffle voilé" is another variety. It begins as vesicular and then changes into bronchial or mixed breathing (see p. 231). The vesicular element in this case is probably transmitted. It occurs principally over tuberculous infiltrations.

The writer once succeeded in demonstrating two different metamorphosed breathing murmurs over adjacent areas (one higher during inspiration, the other deeper), and in making a diagnosis, which was later confirmed by autopsy, of two small abscess cavities situated near together.

INDEFINITE BREATHING MURMUR.

This is neither vesicular nor bronchial, but sounds like the expiratory portion of normal breathing. The term indefinite is applied also to breathing which is very faint and difficult to hear. Both bronchial and vesicular breathing may be diminished enough by a pleural exudation to become transformed into indefinite. Sometimes the breathing becomes indefinite because it is masked by other louder noises (râles or friction sounds). The true nature of indefinite breathing can often be brought out after the patient breathes deeply. Loud, distinct breathing is never indefinite, nor is the mixed murmur (to be described below). The only diagnostic significance of indefinite breathing is that of a very weakened murmur whose vesicular, bronchial, or mixed origin cannot be determined.

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MIXED BREATHING MURMURS (BRONCHOVESICULAR BREATHING).

There are two main types of bronchovesicular breathing:

1. Vesicular inspiration with bronchial expiration.

2. Mixed inspiration—*i. e.*, inspiration which is both vesicular and bronchial—with bronchial expiration.

The conditions of origin of bronchial and of vesicular breathing differ so manifestly that at first sight it would seem difficult to explain these mixed murmurs. Practically, however, the vesicular and bronchial elements never arise from one spot, but each is transmitted from some distance and heard in combination with the other; and so a mixed murmur entitles us to assume that there exists in close proximity both normal pulmonary tissue and tissue so altered as to produce bronchial breathing. Thus, bronchovesicular breathing is to be heard: (*a*) over portions of the lung containing scattered small infiltrations; (*b*) over normal pulmonary tissue near large infiltrations; (*c*) in the neighborhood of the upper boundaries of dulness of a pleural effusion which compresses the inferior while permitting free breathing of the superior portion of the lung; (*d*) over cavities surrounded by healthy lung tissue, etc. The importance of bronchovesicular breathing in disclosing small areas of consolidation is evident, because such areas need not produce any dulness.

Physiologic bronchial breathing is almost always combined with vesicular breathing. This is important in differentiating physiologic from pathologic bronchial breathing. But the latter may also be mixed with vesicular breathing, and the distinction between physiologic and pathologic mixed breathing is as difficult as it is important. Physiologic mixed breathing is especially common over certain areas, mentioned above, whereas pathologic mixed breathing may be heard over any part of the lung. Even where physiologic mixed breathing is diffused over a large area, we can easily determine that the nearer we listen to the greater bronchi or the roots of the lungs the stronger the bronchial element becomes; and, conversely, the farther from them, the weaker. If the bronchial element increases with the distance from the lung roots, we may infer that it is pathologic mixed breathing. Physiologic mixed breathing is much more affected by the intensity of the laryngeal murmur and by the position of the mouth than is pathologic. The character of bronchovesicular breathing often varies at different examinations, at the one time plainly vesicular, at another bronchial. This is supposed to be due to secretion plugging a bronchus, at one time in the infiltrated area, at another in the normal area. In the former case the bronchial, in the latter the vesicular, component is weakened or disappears.

There is still another type of mixed breathing which is of diagnostic importance. Mixed breathing is sometimes heard over a considerable area of the lung, loudest near the hilus (physiologic type); or, conversely, near the pulmonary edges. If accompanied by a chronic cough, but with no other evidence of pulmonary infiltration or tuberculosis, it would argue very strongly for a bronchiectasis.

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The device for recognizing weak bronchial breathing (p. 229 *et seq.*) also aids in differentiating mixed from pure vesicular breathing.

RALES (Rhonchi).

Under this term are included all sounds which are caused by the motion in the bronchi, not only of air, but of secretions or of other fluid, semifluid or solid materials. Moist râles depend upon fluid, dry upon solid, material within the bronchi. To determine râles exactly and carefully analyze them we must auscultate the patient while breathing deeply as well as while breathing quietly, and also during and following a cough. No other auscultatory phenomena are so markedly altered by such a variation in the type of examination. Coughing, for instance, will oftentimes cause râles either to appear or disappear. If a patient is difficult to examine, if he cannot be persuaded to breathe properly, tell him to count aloud as long as possible, and the next inspiration will naturally be full and deep. We can often bring out a very few râles, the demonstration of which over the pulmonary apices is very essential in the diagnosis of pulmonary tuberculosis, by auscultating the patient early in the morning, before he has expectorated the secretion accumulated during the night. Râles can often be felt by the hand placed upon the chest.

Transmission of Rales.—Râles can also be heard by transmission at a certain distance from their place of origin. But their intensity ordinarily decreases so quickly that the determination of their origin rarely offers difficulty.

Oral Rales.—It frequently happens that râles may be heard at some distance when the patient breathes with his mouth open. As a general rule these are large râles, and can also be heard at some distance proceeding from the thorax even when the mouth is closed. This transmission of râles through the mouth is particularly marked in the tracheal and pharyngeal râles of moribund patients. These râles owe their origin to the flooding of the trachea and pharynx by the fluid of the pulmonary edema, but may also be caused by failure to swallow the pharyngeal mucus. By auscultating the trachea and the larynx, it may be determined whether the râles arise here or in the depths of the lung. It occasionally happens that a deep area in the center of the lung produces oral râles which are not transmitted to the surface of the thorax. This symptom has a certain diagnostic significance.

MOIST OR BUBBLING RALES.

The sound of moist or bubbling râles can be imitated by blowing through a glass tube into a vessel of water. The expansion of air into bubbles causes a characteristic crackling noise, which will vary with the caliber of the tube and with the strength of the bubble. A wide tube will produce large bubbles, with a sound resembling the so-called *large* or *coarse bubbling râles;* a narrow tube causes fine bubbles, the sound resembling the *small* or *fine bubbling râles.*

It is much easier to appreciate by the ear the difference between the fine and the coarse bubbling râles than it is to define it scientifically. *Coarse bubbling râles* are fewer in number, more intense, and of a lower pitch. *Fine bubbling râles* are more numerous, less intense, and of a higher pitch. This distinction does not, however, entirely fulfil the conditions, because accelerating the air current will increase the number of the bubbles without altering their character. On the other hand,

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approaching the ear close to or distant from the tube will increase or diminish their intensity also without altering their character. Neither does the essential distinction depend upon the pitch, for in many cases no distinct pitch can be recognized. Therefore, some other difference between coarse and fine bubbling râles must exist. The writer imagines some difference depends upon the size of the mass set in motion—*i. e.*, in coarse bubbling râles the energy of the vibration is greater than in fine bubbling râles, because the moving mass is larger. And this influences not only the intensity of the vibrations, but, in consequence of the law of inertia, the duration of the tone itself. The coarse râles last longer. (See Duration of the Percussion Tone.) If this explanation is correct, râles may be few in number, and, when they arise close to the ear, intense; and yet retain their fine bubbling character.

The term "bubbling" was employed because it was formerly supposed that moist râles arose from the bursting of air-bubbles in fluid secretion. Of course, we know now that the contents of the bronchi are not sufficiently fluid for such an explanation; and so we suppose that the râles arise from membranes of secretions being formed in the bronchial lumen, and then torn apart again, partly by the movement of the air and partly by the movement of the lungs. The *coarse bubbling râles* would therefore correspond to the thicker, and the *fine* to the thinner, layers of secretion. We always auscultate over a more or less extensive bronchial area; hence many different individual noises may give rise to the resultant "much bubbling" noise. Moving or bursting of real bubbles probably occurs only in quite isolated cases, and then really only when the lung is saturated with thin fluid—*e. g.*, in pulmonary hemorrhage, pulmonary edema, or drowning.

Moist râles may be heard during expiration as well as during inspiration, although during inspiration they are almost always much plainer, possibly because the movement of the lung is quicker.

Very fine bubbling or moist râles are also named *crackling* or *subcrepitant râles*, the latter from a certain similarity to *crepitant* râles (pp. 237 and 238).

A complete series of transitions exists between the coarse bubbling râles as one extreme and the fine moist râles as the other. The rattling tracheal or pharyngeal râle, generally heard in the moribund, even at a distance, belongs to the former variety. Fine moist râles more commonly originate in the fine tubes; coarse, in the larger tubes or in pathologic cavities. The former are generally more numerous because there are more fine tubes.

Moist râles arise only when the bronchi contain fluid or semi-fluid material; in most cases, therefore, they signify a bronchial catarrh, to be located in the large or small bronchi, according to the size of the râles. Fine moist râles have in general a more serious import, because a catarrh of the finer bronchi often leads to bronchopneumonia, and because such a catarrh frequently depends upon local changes of the pulmonary parenchyma (such as inflammatory infiltration, tuberculosis or infarction). This is especially true if the fine moist râles are

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heard localized over a definite area of the lung. The obstinate persistence of such localized râles over the same area while the rest of the lung is permanently free, even without any other signs, justifies the diagnosis of a serious process, either a tubercular or a lobular pneumonic infiltration or an infarction. On the contrary, an ordinary innocent catarrh is much more diffused, because an otherwise healthy mucous membrane presents practically the same fostering soil throughout its entire extent. Coarse bubbling râles heard over areas where no large bronchi are to be found signify an especially serious affection, because they must arise either from pathologically dilated bronchi or from cavities. When heard over the apices, they are thus important signs of tubercular cavities; when heard over the postero-inferior portions of the lungs, they more frequently depend upon bronchiectasis. Coarse moist râles are, however, frequently transmitted to quite a distance, a possibility which must be kept in mind when they are heard over areas somewhat remote from the roots of the lungs. Such a possibility may, however, be excluded if at the same time no coarse râles are heard over the larger bronchi. Moist râles of any size, when obstinately localized over a circumscribed area, point to a serious focal lesion of the lungs.

Mixed bubbling râles arise when the small as well as the large bronchi are affected. They also arise from the larger bronchi and from pulmonary cavities.

Pulmonary hemorrhage and pulmonary edema, unless the larger bronchi are also flooded with fluid, present quite uniformly fine bubbling râles, which are frequently heard both with inspiration and expiration (continuous râles). The expectoration in these cases is ordinarily very foamy.

The sticky mucous secretion of an ordinary catarrhal bronchitis generally produces *dry* râles. Moist râles have a more serious significance; their appearance is associated with a fluid secretion deficient in mucus—*e. g.*, they are especially common in intense inflammatory processes of the mucous membrane or of the pulmonary tissue, in edema, stasis, and hypostasis. Moist râles constantly present over the apices of the lungs almost always mean tuberculosis. In such a case a thin fluid secretion, such as the moist râles suggest, signifies a marked purulent and destructive process.

DRY RALES (CRACKLING OR SNAPPING AND MUSICAL RALES).

Dry râles are produced by the movement of a viscid secretion. They have a more manifold character than moist râles. The latter are always composed of a more or less regular succession of separated noises. The former present either a quite isolated sound, a crackling which resembles to some extent a moist râle, or a prolonged, distinctly musical sound (the humming, whistling, sonorous or sibilant râle). The difficulty in moving the tenacious secretion probably accounts for the lack of regularity in the succession of the dry râles.

To explain the origin of the crackling dry râle, we should remember that the secretion of the mucous membrane of the bronchi is stretched

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out in the form of threads or membranes, is torn loose either by the air current or by the movement of the lungs, and later, by virtue of its tenacity, again sticks fast to another portion of the bronchial wall without necessarily giving rise to another series of noises. Thus, they differ very slightly in their origin from the moist râle, except that on account of its viscidity the secretion is probably only seldom stretched and then torn loose from the mucous membrane, whereas in moist râles this process is constantly repeated. Such a conception practically tabulates the dry crackling râles as a subdivision of the moist râle.

Very possibly certain forms of crackling râles are not real râles—*i. e.*, they do not depend upon the movement of secretion, but upon a jerky current of air over a surface roughened and irregular from infiltration in the neighborhood—*e. g.*, in tubercular apices.

Musical dry râles arise from the vibration of threads or membranes which are drawn out across the bronchial lumen. They are set in vibration (but not torn away by the air current) just like the strings of a fiddle or the tongues of a pipe. Again, some of the walls of the finer tubes may be so swollen by inflammation as to be almost occluded, and form a sort of whistle. Finally, the swelling of the mucous membrane may be situated at the angle between two communicating bronchi, thus acting like the tongue of a pipe. These various causes explain the diversity of musical râles, which sometimes resemble the purring of a cat, sometimes the tone of a violin, a harp, or a bass viol, sometimes the snoring of a sleeper, sometimes a shrill whistle. The deeper sonorous râles generally arise in the larger bronchi; the higher, whistling or sibilant râles, in the smaller.

The names crackling, sonorous, or sibilant râles—more briefly, crackles, snores, or whistles—are well used to characterize the sounds.

When dry râles are obstinately localized at one spot they have the same serious significance as moist râles, even without other auscultatory or percussion phenomena (see pp. 233 and 234). Pulmonary tuberculosis for a long time may be evidenced only by the sounds of a dry catarrh at one apex.

Dry râles are often transmitted to a considerable distance. Sonorous râles are often appreciated by the palpating hand, and resemble a pleuritic friction (see p. 239).

RESONANT (Consonating) AND NON-RESONANT (Non-consonating) RÄLES.

Râles which arise in bronchi surrounded by solid—*i. e.*, airless tissue (infiltration, atelectasis)—are transmitted, intensified or modified by resonance, exactly as is pathologic bronchial breathing (see p. 226 *et seq.*). They then become exceptionally sharp and distinct, and are called *resonant* or *consonating râles*, in distinction to the non-resonant râles heard over normal pulmonary parenchyma. The terminology is here somewhat confusing, for a dry musical râle must naturally be resonant, but is not modified by a resonance in the sense mentioned above. To avoid this confusion it would be advisable to return to Skoda's terminology, *consonating râles.*

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Dry musical râles (sonorous and sibilant), even when they arise in aërated pulmonary tissue, have so distinct and well recognized a musical character that conditions of resonance or consonance cannot alter them to any extent, and only a very skilled ear can distinguish any difference between consonating and non-consonating musical râles. On account of the marked preponderance of a ground-tone, consonance in such râles is evidenced more by an increased intensity than by any qualitative modification of the timbre. Merely from the increase in intensity we can draw no further conclusions. But moist and also *crackling râles* are very plainly affected by consonation. The entire effect is increased in intensity, and certain high partial tones are especially intensified by resonance. The result is difficult to describe clearly, but can easily be demonstrated upon an appropriate patient. In the discussion of bronchial breathing we showed that actual resonating phenomena do appear in the bronchi, and so there is no reason to doubt that a râle may resonate. But the better conduction through infiltrated tissue is not alone sufficient to make the râles resonant; nor is loudness of any avail—*e. g.*, tracheal râles loud enough to be heard even at a distance show no trace of resonance. The resonating character depends for the most part on an admixture of the higher overtones. Certain types of stethoscopes will add a distinct resonance to râles (see p. 244). A pulmonary cavity which presents a metallic note to percussion and an amphoric breathing to auscultation also furnishes consonating râles, with a metallic resonance.

The so-called noise of *falling drops* (gutta cadens), heard especially in tubercular destruction of the lung, is apparently nothing but a metallic consonating crackling râle in a cavity (see, however, a similar sound with a different import, p. 240 *et seq.*). After this discussion of its origin, the diagnostic significance of the consonating râle will require fewer words, for it should now be clear that the demonstration of the consonation of râles has exactly the same significance as the demonstration of pathologic bronchial breathing. And the writer would emphasize to the beginner the importance of distinguishing consonance and non-consonance of the râles in the diagnosis of infiltrations. If bronchial breathing, consonating râles, and dulness of the percussion tone were always present together over pulmonary infiltrations, the presence of one or the other of these signs would be sufficient, and we could then dispense with the others. Unfortunately, it is not so simple; for, as we have already seen, bronchial breathing is very often heard without any dulness, and just as frequently consonating râles without bronchial breathing. In gross, the conditions essential for consonating râles and for bronchial breathing are the same; but either may be present alone, because quite distinct sound elements must be increased by resonation to exhibit the consonating character in the râles, on the one hand, and in the respiratory murmur on the other.

From a practical standpoint there is one thing more to emphasize. A râle must possess a certain strength to be able to consonate plainly; hence, with very superficial breathing we frequently hear over

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an infiltration or cavity non-consonating râles, which with deeper breathing are transformed into consonating râles. Lobular pneumonia is often very difficult to demonstrate in quite sick patients, especially because the superficial breathing characteristic of this disease is scarcely strong enough to produce plain bronchial breathing. With mixed bubbling râles certain ones may be consonating and others non-consonating, because parts of the mixed breathing murmur are not strong enough to bring out the consonation.

CREPITANT RALES OR CREPITATION.

During the first stage of pulmonary infiltration, when anatomically the lung is in the state of engorgement, when the pulmonary tissue is still full of air, when percussion discloses no dulness, and when auscultation shows no bronchial breathing, there is to be heard over the affected part of the lung the so-called *crepitant râle*, or *crepitation* (crepitatio indux). A similar noise (crepitatio redux) is to be heard during the resolution stage of pneumonia when the solidified lung again becomes aërated. This characteristic sound is to be heard both in *croupous* and *catarrhal pulmonary inflammation*, in certain stages of *tubercular infiltration*, and in *hemorrhagic infarctions* and in *beginning pulmonary edema*. In most cases only audible during inspiration, *crepitation* possesses a certain acoustic similarity to a very fine moist râle (subcrepitant), and can be very well imitated by rolling one's hair between the fingers, near the ear.

Formerly crepitation was considered to be a true, very fine, moist râle, arising when the air current set into motion fluid secretion in the finest bronchi and alveoli. But this supposition has been disputed; in the first place, because we can reproduce crepitation by blowing up or by pressing between the fingers a cadaver's dry lung; and again because in perfectly healthy individuals with no real secretion in the alveoli or bronchi, crepitation is sometimes heard, especially at the inferior boundary of the lung, or after they have been breathing superficially for a long time and then take a deep breath. The fact that it is heard only with inspiration is a further argument against its origin from the movement of secretion. It is now apparently universally conceded that crepitation does not arise from this cause, nor, as was earlier supposed, as a consequence of a great number of microscopic explosions of air bubbles in the fluid contents of the alveoli, but from the tearing apart of the approximated alveolar walls by the inspiratory stream.[1] In pulmonary engorgement related to the first stage of pulmonary edema we can well imagine that the increasing swelling and soaking of the alveolar walls would approximate them, and the presence of some fluid secretion or exudation in the alveoli would only favor each expiratory adhesion.

[1] [Prof. Sahli has entirely disregarded one theory of the origin of crepitation—viz., that it is in reality a very fine pleural friction rub and does not come from within the lung. This theory seems to us less plausible, but so good an authority as Osler says, "Whether this is a fine pleural crepitus or is produced in the air cells and finer bronchi is still an open question."—ED.]

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Still, fluid plays here a secondary rôle. In pulmonary edema, if the transudation increases, genuine fine bubbling râles become gradually associated with crepitation, and later on, when the fluid reaches the greater bronchi, even coarse moist râles are developed. An excellent opportunity is thus afforded to study the difference between crepitation and the fine moist, or so-called subcrepitant, râles. They are easily distinguished by remembering that the former occurs during inspiration and possesses a resonating character.

When associated with bronchial breathing, crepitation points to the beginning of an infiltration, and has, therefore, a serious significance. On the contrary, however, the crepitatio redux at the termination of a pneumonia is a longed-for sign of beginning resolution, and a demonstration that the alveoli have once more become permeable to air. Crepitation may in certain cases be heard over some spot or other of the chest during the entire course of croupous pneumonia, because this disease almost never develops simultaneously.

Actual crepitation may exceptionally be heard with expiration, probably depending upon the fact that under unusual conditions some of the alveoli become filled with air during expiration instead of during inspiration. This is simple enough to understand if we suppose that, in consequence of adhesions or of partial rigidity from small areas of infiltration, one portion of the lung does not breathe as well as an adjoining part, and that the good breathing part, on account of some obstacle opposing its emptying (secretion or swelling of the mucous membrane), sometimes pumps the expiratory air into the poor breathing part. The latter will then be distended while the rest of the lung expires, thus giving rise to a crepitation which is expiratory in relation to the general respiration, but inspiratory in relation to the affected part itself. In lobular infiltrations at the sharp pulmonary edges the writer has not infrequently found this expiratory crepitation.

CARDIOPNEUMATIC RALES (Cardiac Rales and Cardiac Crepitation).

Before concluding the discussion of the ordinary râles occasioned by respiratory movements, we should mention the fact that in rare cases with coexistent catarrh of the bronchi, râles may be produced by the movements of the heart itself. These are due in part to the direct mechanical vibration of the adjoining portions of the lung; in part, however, to the systolic and diastolic variations of intrathoracic pressure from alterations in cardiac volume (auxo- and meiocardia). Such variations cause real respiratory excursions of the adjoining pulmonary parts. (Already noted under Systolic Vesicular Breathing, pp. 221 and 222.) Cardiac râles are most frequently systolic when the lung, partly infiltrated or filled with cavities, is adherent to the pleura near the heart, and then set into active motion by the cardiac action.

In a similar way, provided the essential characteristics are present in the lungs, *crepitation* may be occasioned by the heart action (cardiosystolic râles.

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PLEURAL FRICTION RUB.

The two pleural surfaces move against each other normally without producing any noise; but when the surfaces have become roughened, as in pleurisy, in tuberculosis, in tumors, or with the abnormal pleural dryness of cholera, a characteristic friction rub is developed. The area over which the friction rub is audible varies with the extent of the fibrinous deposition or roughness. The excursions of the pleural surfaces are more extensive at the pulmonary edges; therefore, a friction rub is more often heard there than at the hilus of the lung. Friction rubs are rarely heard over the apices of the lungs, where the movement is more a distention by centrifugal inflation than a sliding. Depending upon the type of the roughness, the rub will be either inspiratory or expiratory, or both. The kind of noise produced is practically manifold. It may be imitated by placing the flat of the hand against the ear and slowly rubbing the back of the hand with the ball of a finger of the other hand. The character of the rub varies with the amount of pressure. Some, exceptionally loud, simulate the creaking of new leather or sonorous râles, whereas others, much fainter, resemble the noise caused by rubbing two pieces of silk stuff together, and still others resemble crepitation, cog-wheel breathing, or even faint bronchial breathing. Another peculiarity of friction rubs is that they are usually jerky and not smoothly spread over each phase of the breathing.

Though a mere beginner recognizes without difficulty a decided rough pleuritic rub, yet even an expert is oftentimes in doubt over the nature of a sound which resembles a sonorous or crackling râle as much as it does a friction rub. One way of determining is to try the effect of coughing. It will often entirely change the character of a râle, and sometimes even cause it to disappear, but does not affect a rub. On the other hand, pressure of the stethoscope or of the flat hand intensifies a rub, but does not affect a râle. A third source of help is palpation, which appreciates a friction rub more readily than a râle. The confusion between a soft rub and faint bronchial or cog-wheel breathing can be cleared up only by an expert's repeated and accurate examinations. Another peculiar rub, which fortunately is quite rare, can be distinguished from crepitation only by the fact that it can be just as plainly heard during expiration as during inspiration, and that it is more jerky than the crepitation. Friction rubs heard over the precordia, which originate from the pleura, from the pericardium, or from the sharp pulmonary edge overlapping the heart, may be synchronous with the cardiac action as well as with the breathing. They are called *pleuropericardial*, *pseudopericardial*, or *extrapericardial friction rubs*, and are liable to be confused with true pericardial rubs. (See Pericardial Friction Rubs for Diagnosis.)

When a fluid exudation complicates pleurisy, the friction rub disappears, because the layer of fluid prevents the two pleural surfaces from touching and rubbing. The rub may persist, however, along the edge of dulness, rather more commonly in front than behind. Such a persistence proves that the two pleural surfaces touch and rub against each

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other somewhere above or below the adhesions, encapsulating the fluid exudation. The reappearance of a rub within the boundaries of the dulness is a favorable sign, because it shows that the fluid is diminishing and the pleural surfaces are again in contact. The dulness may then be due, at least in part, to thick layers of fibrin. Loud friction rubs may, of course, be transmitted some distance from their place of origin; but if they are plainly felt by the palpating hand over the area where they are loudest heard it is pretty good evidence that they arise very nearby.

RESPIRATORY MURMURS IN INTERSTITIAL PULMONARY EMPHYSEMA.

When air from within the lung is forced into the interstitial pulmonary connective tissue by the rupture of one or more alveolar walls, bubbling sounds and sometimes murmurs are produced by each respiratory excursion. These sounds generally recall coarse or mixed moist râles, and for the most part are resonant in character. The resonance, which may even take on a metallic character, probably depends upon the resonation in the cavities formed by the bubbles. Sometimes these sounds simulate the familiar emphysematous crackling of the skin (see p. 52), but they are too coarse to be confused with crepitation (p. 237 *et seq.*). When large emphysematous bubbles are situated beneath the pleura, they may sometimes decidedly weaken the respiratory murmur. Interstitial emphysema may be present at any spot; it may spread over the entire lung, and from there into the subcutaneous connective tissue. Sometimes the diagnosis of interstitial pulmonary emphysema will be first securely established and the confusion with râles prevented by the demonstration of the precordial murmur of emphysema, and of the characteristic crackling of the skin.

NOISES AUDIBLE IN PNEUMOTHORAX.

PLEURAL SPLASHING NOISE (Succussio Hippocratis).

Hippocrates first described the characteristic splashing noise produced by shaking a patient with sero- or pyopneumothorax. The noise is similar to that made by shaking a large flask half-filled with water, and arises in the same way. Sometimes it is audible at a distance; sometimes only to be heard when the examiner places his ear against the chest. The patient himself sometimes notices the noise during all violent movements. The splashing sometimes presents a distinctly metallic timbre, according to the shape and size of the pneumothorax cavity and to the tension of the contained air.

Similar succussion noises may evidently arise in any other physiologic or pathologic body cavity containing both air and fluid; for example, when air is contained in the pericardium or in the peritoneum with a fluid effusion. It is also occasionally heard in a large pulmonary cavity, and quite frequently in perfectly healthy people with stomachs distended with large amounts of fluid and air. In order to differentiate the Hippocrates succussion from these phenomena, and especially from the last-mentioned physiologic murmur from the stomach contents, we must utilize the results of other methods of examination and endeavor to localize the origin of the splashing as accurately as possible by the proximity of the ear, and also by carefully examining with a filled and a fasting stomach. Frequently the examiner can appreciate, with his hand applied to the

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thorax of the patient, a distinct blow of the fluid wave within the pleural cavity at the moment of the shaking.

WATER-WHISTLE NOISE (Pulmonary Fistula Noise).

This is a characteristic gurgling noise which often sounds metallic and reminds one of a coarse bubbling râle. It is heard over a pneumothorax occasionally when the air or the fluid is removed by aspiration, and so the atmospheric pressure diminishes the pressure in the pleural cavity. In a valvular pneumothorax the murmur will arise as soon as the pressure within the pleural cavity is diminished to below that of the atmospheric pressure; then air immediately escapes from the patent pulmonary fistula. If this fistulous opening is situated below the level of the fluid, the noise is produced by the rising of the air bubbles. On account of its acoustic character and of its mode of origin, Unverricht called it a water-whistle noise, and Riegel called it a pulmonary fistula noise. The same noise will be noted even without aspiration if in a patient with a pyopneumothorax perforating into the lung air is sucked in with inspiration during the expectoration of the exudate, or, again, if with coughing air is squeezed out of the lung by the fluid in a pyopneumothorax which opens through the thoracic wall and which also has a pulmonary fistula. The sign is of some diagnostic value in recognizing the occurrence of a pulmonary fistula.

NOISE OF FALLING DROPS IN PNEUMOTHORAX.

In pulmonary tuberculosis, as we have seen upon p. 236 *et seq.*, the so-called noise of falling drops is always to be considered as a simple metallic crackling râle; but in pneumothorax, on the contrary, it is possible to hear actual falling drops. When such a patient changes from the recumbent to the sitting posture, it is quite possible that the shaggy prominences of the pleura covered with fibrin, which were previously moistened, free themselves from fluid drop by drop, and in this way produce a metallic dropping noise.

AUSCULTATION OF THE VOICE SOUNDS OVER THE CHEST [Bronchophony].

Transmission of the voice sounds from the larynx and trachea through the lung to the chest surface is subject under normal conditions to the laws which govern the transmission of the laryngotracheal respiratory murmur. The voice sounds are therefore heard best over the areas where we hear physiologic bronchial breathing—*i. e.*, near the hilus of the lung between the shoulder blades, and over the upper end of the sternum. But even in these regions articulation is faintly appreciated, while over the peripheral portions of the lung only an indistinct murmur can be detected. This comparatively distinct transmission of the voice over the areas of physiologic bronchial breathing is known as *physiologic bronchophony*. At best the voice cannot be heard distinctly, but sounds as if someone were talking at some distance in a hall of poor acoustic qualities. The whispered voice is usually heard a little more distinctly. Pronounced bronchophony will at least transmit individual sounds and the rhythm of syllables. Very faint bronchophony is known as "bronchial whispering."[1] Pathologic bronchophony is due to the same conditions and is subject to the same laws as pathologic bronchial breathing. It is heard over areas of

[1] [The term "bronchial whisper" should not be confused with "whispered bronchophony."—Ed.]

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compressed or infiltrated lung with patent bronchi, over bronchiectases, and over pulmonary cavities. Bronchophony may acquire a metallic sound over cavities. Pathologic bronchophony, like bronchial breathing, owes its origin partly to better conduction of sound by bronchi which are surrounded by an infiltrated area and partly to resonance. Physiologic bronchophony varies in different individuals; hence, in order to detect any pathologic change, it is usually advisable to compare symmetric portions of the chest. It should, however, be remembered that physiologic bronchophony is ordinarily louder on the right side, just as is physiologic bronchial breathing. Both the spoken and the whispered voice should be tested, and the ear which is not used for auscultation should be closed, so as to avoid any sound transmission through the air. Pathologic bronchophony has exactly the same diagnostic significance as consonating râles and pathologic bronchial breathing (see above). All these signs, in virtue of their common origin and uniform significance, may be included in the term "consonating phenomena." Each one should, however, be looked for, because, in spite of their uniform cause and significance, any one of the three may be imperfectly developed; whispered bronchophony, for instance, may be heard over an infiltrated area, although bronchial breathing is indistinct. If deep breathing is painful or impossible, the demonstration of bronchophony may be of great value.

Extreme bronchophony, or *pectoriloquy*, and *egophony*, a form of bronchophony with a peculiar bleating sound, are sometimes considered as distinct and separate phenomena from bronchophony. But the writer does not consider it justifiable to attribute these signs to different conditions, because the various types of bronchophony blend in such a manner as to be indistinguishable one from the other. Neither does he consider it justifiable to attribute any special diagnostic significance to pectoriloquy as a sign of cavities, nor to egophony as a sign of a pulmonary compression which is sufficient to cause flattening of the bronchi. Pectoriloquy may be so decided over an infiltrated area that the examiner can appreciate the articulation although he cannot actually distinguish the word. Egophony may also be audible over such an area. All sorts of theories have been suggested to explain the occurrence of the bleating sound in egophony, but none of them has satisfied the conditions from the standpoint of physics. In any case egophony is simply a quantitative increase of ordinary bronchophony. If the breathing sounds amphoric and if the percussion note is metallic, the bronchophony is apt to be either amphoric or metallic. By examining for bronchophony with the naked ear the degree of vocal fremitus can be accurately determined at the same time. In fact, the skin of the ear can appreciate the vibration even more delicately than can the hand.

VARIOUS ERRORS IN PULMONARY AUSCULTATION.

Even an expert finds auscultation of the lungs much more difficult than auscultation of the heart, and to a beginner the former is especially

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difficult on account of many possible errors. First of all there is the so-called hair crepitation, caused by the displacement of the hairs under the stethoscope with respiration. This occurs in persons with even a moderate growth of hair on the chest. The crepitations are synchronous with the respiratory movements, and an expert will quickly notice that the sound is just as marked during inspiration as during expiration, and that it is intensified by a careless use of the stethoscope. This source of error may be so annoying that it is necessary to wet the hair thoroughly with water or oil.

Muscle sounds may also lead to error. In auscultating the apices the muscle sounds produced in the trapezius as it takes part in the respiratory excursion may be very disturbing. They may simulate rough breathing or even râles. It is important to be able to recognize this condition, so as to avoid error. Muscle sounds may also be produced by fibrillary contraction of muscles—*e. g.*, "shivering" due to the sudden exposure. These sounds may be appreciated long before shivering is apparent to the eye. Such sounds may be differentiated from the respiratory murmur by asking the patient to hold his breath; if they persist they are not respiratory. Besides these active muscle sounds there is a so-called "passive variety," which may be produced by the displacement of bundles of muscle fibers under the edge of a stethoscope by light movements of the instrument or by the respiratory excursion of the chest itself. These sounds may be recognized by intentionally moving the stethoscope, and so reproducing the sound even when the patient is not breathing. Again, such sounds will not be heard when examining with the naked ear or when very gently applying the stethoscope. Other noises originate from similar conditions, as, for instance, that produced by pressing the stethoscope over the lobulated fat in the female breast. These sounds frequently resemble râles very closely. They disappear when the stethoscope is used properly, and reappear when a little pressure is exerted, even when the patient stops breathing.

In auscultating portions of the body which are difficult of access a beginner is often liable to produce *typical friction sounds* artificially, especially so if he is in an uncomfortable position, so that his stethoscope is not accurately or firmly applied, so that the patient's skin beneath it moves with respiration. Hence, it is a good plan for him to confirm his examination by listening with the naked ear or by carefully readjusting his stethoscope.

There are a great many other opportunities for producing extraneous sounds and so confusing the examiner. We might emphasize the necessity of persuading patients that their chests should be entirely uncovered for a proper examination, that they should not scratch themselves during the examination, and should remain absolutely quiet. It is perfectly useless to attempt an examination, particularly of the apices of the lungs, unless the patient's upper chest is entirely uncovered. A patient's false modesty, his fear of catching cold, or the examiner's laziness—none of these reasons can possibly excuse a careless examination.

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It is desirable for every physician to accustom himself thoroughly to the use of one form of stethoscope. Even if one is accustomed to a poor instrument, he will make much better use of it than at his first attempts with a better one. Stethoscopes differ decidedly in their reproduction of consonation—*i. e.*, bronchial phenomena. The popular hard-rubber stethoscopes, for example, with comparatively long, open bells and thin, smooth walls increase the resonation very decidedly. Mixed breathing through one of these instruments sounds much more bronchial than through a cylindric wooden stethoscope. Râles assume a more consonating character when heard through the former, a fact which the writer considers a practical proof that consonating râles are produced by resonation and consonation.[1]

Another source of error in auscultating the chest may be mentioned. If the stethoscope bell is carelessly applied to the chest-wall, so that part of its edge does not rest there firmly, but is slightly elevated, thus allowing the air free entrance to, and exit from, the bell, the examiner will frequently hear a very good imitation of bronchial breathing. The sound is, of course, due to resonation. Probably the noise of the patient breathing through his mouth is transmitted and exaggerated by the open bell. It is quite similar to the sound we hear when we place a large sea shell with its opening near the ear; when external sounds ordinarily unnoticeable are so much exaggerated by resonation, that children frequently imagine they hear the roaring of the sea.

AUSCULTATION OF THE HEART.

We almost always need a stethoscope to auscultate the heart properly, because, for the sake of diagnosis, we wish not only to appreciate the sounds, but also to localize them as accurately as possible. We often need to auscultate the heart both in the recumbent and in the erect posture, and sometimes in other postures. Hence, it is a good routine to auscultate every patient's heart both while he is lying down and while he is sitting or standing up. (See Mitral Insufficiency and Aortic Insufficiency.)

NORMAL AUSCULTATION SIGNS OVER THE HEART.

The only sounds we hear over a healthy individual's heart are the so-called "*heart tones.*" The word "*tone*" is not used here in its strict acoustic sense, because the sounds heard are more properly *noises*, and only quite rarely possess a distinctly recognizable pitch. French authors call normal heart tones "bruits normaux"; but the Germans and the English have retained the term "tone" despite its acoustic

[1] [The use of the single-barrel stethoscope preferred by Prof. Sahli is not common among the profession in America. Undoubtedly many of his reasons for this preference are well founded; but we think that a good bi-aural stethoscope will transmit the sounds quite as plainly, always provided that the examiner is thoroughly accustomed to the resonation of his own instrument. So many different stethoscopes have been advised by different authorities, that it seems hardly fitting for us to recommend especially the type with which we are personally more familiar.—ED.]

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inaccuracy, and have reserved the word "geräusche" (noises, murmurs) for certain pathologic sounds to be discussed later. (See p. 260 for the distinction between tones and murmurs.)

We normally hear two heart tones over the entire precordia, and with increased cardiac activity they may often be heard at some distance from the cardiac region. One tone follows the other with a definite rhythm. They can be imitated by pronouncing the syllables "lubb-dupp," or by tapping a closed book quite lightly with the tips of the fingers. A short pause exists between the first and the second tone, and a longer pause ensues before another first tone occurs. The first tone (the so-called systolic tone) is synchronous with the apex beat as felt in the fifth intercostal space—*i. e.*, the beginning of systole. The second sound is synchronous with the beginning of diastole, and is therefore called the diastolic tone.

Physiologists have proved that each heart tone is made up of several factors, and that the reason why at any point of the heart we hear only two tones is because all cardiac tones are either systolic or diastolic; in other words, all systolic tones are massed together, and so are all diastolic tones. In reality, six different tones arise over the heart—four systolic and two diastolic.

The four systolic tones originate:

1. Over the left ventricle, at the mitral valve.
2. Over the right ventricle, at the tricuspid valve.
3. Over the beginning of the aorta.
4. Over the beginning of the pulmonary artery.

The two diastolic tones originate:

5. Over the aortic semilunar valves.
6. Over the pulmonary semilunar valves.

Hence, no diastolic tone originates over the auriculoventricular valves—*i. e.*, over the ventricles.

It has been clearly understood for years that the diastolic tones could be produced only by diastolic tension of the semilunar valves. The origin of the systolic tones have, however, baffled investigators for some time. Without going into the historic presentation of this question, the present theory, which has apparently stood the test of physiologic and clinical observation, will be briefly mentioned. The systolic tone, which is heard at the apex, originates, according to this theory, principally from the systolic tension of the auriculoventricular valves, which are closed at the end of diastole. This view is supported by clinical observations. In addition, experiments upon the bloodless heart have proved that the muscular sound of the heart muscle also takes part in the production of this systolic tone. The expression "muscular sound" should not be construed in the same way as when that term is applied to the skeletal muscles. The muscle sound of a skeletal muscle is one whose pitch depends directly upon the number of individual contractures, which together produce the condition of tetanus. The contraction of the heart is, however, single and not tetanic; hence, its muscular sound is very different from the skeletal muscle sound. The muscular sound of the

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heart is in reality a vibratory phenomenon caused by the sudden systolic contraction of the heart—in other words, a systolic tension tone, exactly of the same character as and coincident in time with the tones produced at the auriculoventricular valves and over the large arteries (aorta, pulmonary artery). This conception therefore means that all systolic tones of the heart are identical in character and are due to tension of its walls—*i. e.*, including its valves. The systolic tones of the aorta and pulmonary artery are referable to the systolic tension of the conus arteriosus and the closed semilunar valves. They are produced by the sudden increase in intracardiac pressure at the beginning of systole. It is incorrect to maintain, as has been frequently done, that the first tone is produced by systole forcing the blood into the aorta and into the pulmonary artery, because Martius' experiments proved that the first tone is synchronous with the beginning (closure or tension time) of systole; and because at that moment the arterial openings are still closed.

Clinically, it has been quite generally accepted that the systolic tones originate not only at the auriculoventricular valves and over the ventricles, but also at the great vessels (aorta and pulmonary artery). This opinion has been attacked upon physiologic grounds. Nevertheless, the writer considers that the clinical view is strongly supported by cases of mitral and tricuspid insufficiency which furnish a systolic tone over the auscultation area of the great vessels, while no such tone can be made out over the ventricles. Such cases are not very rare.

Systolic tones which are heard over the left ventricle are commonly called "mitral tones," those heard over the right ventricle, "tricuspid tones." We thus limit the tones to that part of the sound which originates at the valves alone, in order to interpret clinically the pathologic alterations in the systolic tones. The so-called muscular tone has thus far proved of little diagnostic significance because we cannot readily distinguish it from the proper valvular tones. Certain impurities in the first tone, usually referable to the auriculoventricular valves, may, however, be caused by the muscular tone. Perhaps finer methods of examination may be devised, so that we may be able to utilize these muscular tones in the diagnosis of diseases of the heart muscle (see p. 254).

In order to make an exact diagnosis of the condition of the different chambers of the heart we should separate the elements of the tones and distinguish them individually, although the above-mentioned six tones of the heart are so intimately merged into a mixed systolic and a mixed diastolic tone that an inexperienced ear can only perceive the same tone over the entire heart.

The fact that the projections of the arterial openings and of the valves upon the chest-wall are at some distance from each other (Fig. 102) enables us, according to the spot auscultated, to appreciate at will sometimes one elementary tone of the heart better, sometimes another. We do not, however, best accomplish such an acoustic separation by auscultating each valve exactly at the point of its projection upon the chest-wall, for these points of projection are situated

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rather near each other, as is shown in Fig. 102. Hence, the sounds at each point are quite confused. In practice, except for the pulmonary valve, which is auscultated exactly over its point of projection, we employ the following device: We set the stethoscope over the valve to be auscultated, and then move it away from this point of projection until we reach a spot where the tone we wish to auscultate can still be distinctly heard, but where the other tones, on account of their greater distance, are as faintly heard as possible and so no longer cause confusion. This device is founded upon the well-known law, that the intensity of sound varies inversely with the square of the distance from its point of origin. By carefully noting the result of auscultation in valvular disease (including the substitution of murmurs for the tones of

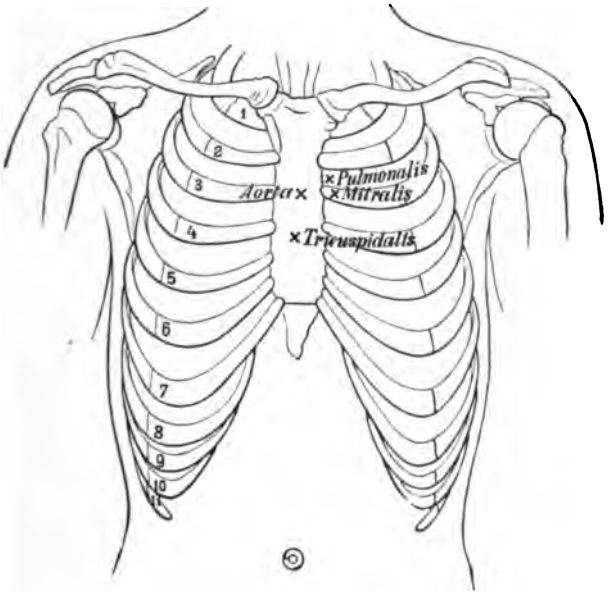

FIG. 102.—Projection of the heart valves.

the affected valve) and by comparing such results with the changes subsequently found in the valves at autopsy, we are able to select empirically the points at which each valve tone can be best auscultated separately.

The following relations between the sites of the valves (Luschka) and their respective auscultation points have been demonstrated by extensive experience:

The *mitral valve* is situated beneath the junction of the third left costal cartilage with the sternum; its *tone* is heard best at the apex beat.

The *tricuspid valve* is situated halfway between the points where the left third and right fifth costal cartilages join the sternum. Its *tone* is heard best over the lower end of the sternum.

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The *pulmonary valves* are situated in the second intercostal space, somewhat to the left of the edge of the sternum. Their *tones* are best heard at this point.

The *aortic valves* are situated about the middle of the sternum, at the level of the third costal cartilage. Their tones are best heard in the right second intercostal space, near the edge of the sternum.

The *mitral valve* is best heard at the apex beat, because the valve itself is overlapped by the lungs and by the right ventricle with the tricuspid valve, while at the apex beat, the overlying lung tissue is very thin, and the left ventricle, in which the sound is produced, is not covered there by the right ventricle, or, at least, to a less degree than farther above. Further, eccentric situation of the apex prevents the other heart tones from being so readily transmitted there.

The above statements explain why the rhythm of the heart tones differs at different points over the heart. The writer will state the facts, and then attempt to explain them. Over the auscultation points for the aorta and pulmonary artery and in the vicinity the heart tones exhibit an iambic rhythm (lubb-dúpp, lubb-dúpp)

Over the lower part of the sternum, however, and as far as the apex beat—*i. e.*, over the auscultation points for the auriculoventricular valves (the ventricles)—the heart tones produce a trochée (lúbb-dupp, lúbb-dupp)

This may be explained as follows: No diastolic tone is produced at the auscultation points for the auriculoventricular valves. The second tone is heard only by transmission from the aorta and pulmonary artery; consequently, it is proportionately diminished. Hence, the accent upon the first tone (trochée). The conditions are different over the great vessels, where both a first and a second tone are produced. Naturally, the first tone is relatively weak because it is produced by a moderate increase in pressure upon the arterial semilunar valves acting against considerable arterial pressure at the beginning or closure time of systole. On the other hand, the second tone is much stronger and accentuated because it is produced by the rapid and forcible closure of the elastic semilunar valves acting under the pressure of the aorta and pulmonary arteries. This accounts for the iambic rhythm heard over the great vessels.

DISTINCTION BETWEEN SYSTOLE AND DIASTOLE BY MEANS OF AUSCULTATION.

One of the most important requisites for the diagnosis of cardiac lesions is the ability to distinguish the systolic from the diastolic phase of the heart's action; in other words, to recognize accurately the systolic and diastolic tones. An experienced clinician does not find this especially difficult under normal conditions, because of the usual accentuation of the tones. The diastolic tone is accentuated over the great vessels, and the systolic tone is accentuated at the apex and over the tricuspid valve.

The sequence of the heart tones at the base of the heart and at the

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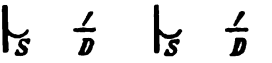
auscultation points for the auriculoventricular valves is shown by the following scheme (where the beginning of systole is indicated by a vertical line):

Great vessels: 

Auriculoventricular valves:

Some cases, however, furnish exceptions to the rule of accentuation in that the first and second sounds are heard more or less equally distinct. Under such circumstances the peculiarity of the pauses furnishes the most efficient means of differentiation. Both physiology and clinical experience have proved that systole is shorter than diastole; therefore the systolic tone is the one preceded by the longer interval of silence. In other words, if the heart tones are arranged in pairs, according to the interval of silence, and practice leads us to do this instinctively, the first tone of each pair will be the systolic, and the second tone the diastolic. In the following scheme of the heart tones the two heart tones are equally accented, but can be readily distinguished by the relative lengths of the pauses between them:

Even this method may be unavailable, because both the pauses are sometimes equal in length (pendulum rhythm). Then the clinician usually depends upon palpating the apex beat. This is a practicable method provided the heart action is not too rapid and the apex beat sufficiently forcible. If, however, the heart is too rapid, the time between the beginning of systole and diastole is too short to associate the sensation of touch with the perception of sound.

Comparing the heart tones with the carotid pulse is even less reliable than comparing them with the apex beat, as the apex beat is synchronous with the first tone, while the carotid pulse corresponds more accurately to the expulsion time.

Systole can never be determined from the radial pulse unless the heart action is very slow. If rapid, the radial pulse is delayed (0.22 seconds, according to Landois), and so coincides more nearly with diastole than with systole. Students should therefore avoid this method if possible.

Systole and diastole can, as a rule, be readily distinguished by one of these methods, but under certain pathologic conditions where the heart action is very rapid, especially if it is irregular, the differentiation may be extremely difficult. Decision must be deferred in some cases until the heart action has quieted down, either spontaneously by rest or with the aid of drugs (digitalis).

In many heart lesions systole may be determined by certain well-characterized pathologic heart murmurs, particularly those which are

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accentuated toward their termination and which occur only immediately before systole—that is, are presystolic (see p. 269 *et seq.*).

In the above description the writer has enumerated the different methods for recognizing the phases of the heart's action in the order of their practicability. The most desirable and the most commonly employed in practice is the determination of systole by the accentuation of the tones and their relation to the pause. Students should practise this method assiduously, and not form the habit of determining systole by feeling the carotid pulse, or, worse still, the radial pulse. The method recommended affords excellent practice in training the ear, and is of great importance to physicians who have never studied music.

ABNORMAL AUSCULTATION SIGNS OVER THE HEART.

Only those abnormal results of auscultation, the determination of which is peculiar to auscultation, will be discussed here. Abnormalities in rhythm can usually be determined quite as readily from the pulse and the apex beat, and will be discussed in the sections treating of the Pulse and the Apex Beat. The same sections describe the significance of the conditions under which the rhythm, as determined by auscultation and palpation of the apex beat, differs from that obtained at the radial pulse. (See p. 101 and the section upon Reduplication of the Apex Beat.)

ALTERATIONS IN THE LOUDNESS OR INTENSITY OF THE HEART TONES.

The intensity of the heart tones depends partly upon conditions within and partly upon conditions without the heart. The thicker the chest-walls the less distinct the heart tones. Hence, in corpulent or muscular individuals, and particularly in women with well-developed breasts, the heart tones are apt to be faint, while in emaciated individuals they are generally strong. Edema of the chest-wall may also weaken the heart tones. Similarly, the heart tones are less audible if the heart is pushed away from the chest-wall, as a result of emphysema, of fluid in the pericardium, or of precordial emphysema. If the pericordial exudation is extensive the heart tones are frequently quite inaudible.

They are intensified, however, if the lung is retracted from the heart as a result of pulmonary contraction or in consequence of an encroachment upon the intrathoracic space (kyphoscoliosis—high position of the diaphragm, or displacement of the heart).

The heart tones are intensified by a consolidation of the pulmonary edges about the heart. The factors operating here are probably the same as those which produce bronchial breathing over consolidations (p. 226 *et seq.*). The heart tones are more perfectly transmitted to the surface, on the one hand, through patent bronchi surrounded by consolidated lung, and, on the other hand, by resonance of the air in the bronchi. Resonance is also responsible for the heart tones being intensified in pneumopericardium and in adjoining cavities of the lungs, also,

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again, in certain degrees of distention of the stomach with air. In all these conditions the tones may acquire a metallic ring.

In other cases, however, the heart tones are intensified on account of conditions within the heart itself. The intensity depends especially upon the strength of the heart. As a rule, therefore, the tones are louder in strong individuals or when the action of the heart is excited, while they are less audible or even inaudible in weak or very sick individuals, during collapse, and in serious affections of the heart muscle which diminish the capacity for work. Many exceptions to these rules occur in individual cases, because, as we have already seen, the intensity of the heart tones depends upon many other factors besides the mere strength of the heart. The elasticity and smoothness of the valves is one in point, and it may perhaps explain why the heart tones are occasionally very loud in weak individuals with low blood-pressure, and in anemic persons.

If the heart tones are markedly increased they are audible not only over the precordia, but also at a greater or less distance from the heart, in the intrascapular space, in the head, in the epigastric region, and occasionally even at some distance from the patient's body.

Increase or diminution of individual tones is of greater significance than uniform increase or diminution of all the heart tones. In order to recognize changes in the intensity of individual tones, we must clearly appreciate the relative intensity of the different heart tones under physiologic conditions. The corresponding tones of the right and left heart are generally conceded to be of equal intensity. This is true despite the greater power developed by the left ventricle, because the intensity of the tone depends not so much upon the absolute value of the pressure acting upon the valves as upon the difference between the pressures acting upon the two sides of the valve at the moment of closure, and the rapidity of the increase in tension. Again, although the mitral and aortic tones of the left heart are naturally louder than the corresponding tones of the right heart, this difference in intensity is neutralized because the aortic and mitral valves are situated at a greater distance from the chest-wall than are the valves of the right heart. Even under normal conditions the corresponding tones of the right and left heart are, however, occasionally of unequal intensity; hence, an increase or diminution of one or the other tone cannot be considered pathologic unless the difference is very marked.

The second aortic tone is increased in hypertrophy of the left ventricle provided the valves themselves are not diseased and the hypertrophied ventricle is sufficient to produce an increase in blood-pressure. This occurs particularly in arteriosclerosis and in chronic nephritis. The second pulmonic tone is increased similarly in the hypertrophy of the right ventricle which accompanies mitral lesions, or in any other condition obstructing pulmonary circulation (emphysema). Accentuation of the second pulmonic tone is therefore an important sign of a compensated mitral lesion. Diminution or weakening of a second pulmonic tone which has been previously accentuated occurs when the compensa-

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tion in these lesions becomes disturbed as soon as the right ventricle fails to exert sufficient power. Under the above conditions, where the ventricle develops more force we might expect that the first aortic and pulmonic tones, as well as those over the auriculoventricular valves, would be intensified. Although not usually the case, this is occasionally true. As has been mentioned above, the intensity of the first aortic and pulmonic sounds depends less upon the absolute pressure exerted by the ventricles at the beginning of systole than upon the difference in pressure on the two sides of the closed valve and the rapidity of the increase in tension. Similarly, the intensity of the auriculoventricular tones depends not so much upon the absolute force developed by the heart as upon the degree of tension and the rapidity of its increase at the valve. Greater working power does not necessarily increase these two factors, provided sufficient blood enters the ventricle during diastole and at the same time places the valve under considerable tension.

Diminution of individual heart tones is caused by more or less extensive destruction of the respective valves. Thus, marked deformity of the mitral valve causes diminution of the mitral tones, and marked destruction of the aortic valve causes diminution of the second aortic tone. At the same time we must not forget that many erroneous views are held about this point. The loss of a portion of the membrane of a valve does not necessarily cause absence or even marked diminution of the corresponding tone, because the remaining portions of the valve can still produce the tones; and, besides, not only the valve itself, but also the surrounding structures contribute to the production of tones. For the first tone the walls of the ventricle and the conus arteriosus, and for the second the walls of the first portion of the aorta and pulmonary artery, play an important part.

The following rule may therefore be accepted: If a valve is affected we shall hear not only the murmur it causes, but also its corresponding tone. An experienced clinician can frequently detect both the murmurs and the heart tone. This is best accomplished by holding the ear at a little distance from the stethoscope, so that the sounds are heard rather faintly. The respective tones may then be diminished by a valvular lesion, but are not necessarily so. It has been frequently stated that the diagnosis of aortic insufficiency depends upon an absence of the second aortic tone. This view is absolutely incorrect. A practised ear can hear the second aortic tone in most cases of aortic insufficiency, although frequently it is diminished. Absence or marked diminution of the second aortic tone may, however, aid in some cases in the diagnosis of aortic insufficiency.

It is interesting and quite important diagnostically to remember that with a marked insufficiency of the auriculoventricular valve not only the auriculoventricular tone, but all the tones of that side of the heart become very much diminished or disappear entirely. The absence of the first tone is not due to the impossibility of producing a first tone in an extensive destruction of the valve, for, as was noted above, the systolic tone originates in all that part of the heart put into

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a state of tension during systole—namely, the walls of the ventricle and the arterial openings. For example, a marked mitral insufficiency may show no systolic tone at any point over the left heart, and not merely the first, but also the second, tone over the aortic area may be entirely absent. The cause of this peculiarity is at first glance rather difficult to understand, but it may be explained as follows: On account of the existence of mitral insufficiency the closure time of the heart disappears, but its place is usurped by a period during which the heart does contract about its contents, but the rapidity of increase of tension is very slow, because the blood in the ventricle immediately escapes into the auricle. Consequently, the mitral valve is never brought into a state of rapid and strong tension, because, as soon as the blood escapes into the auricle, both sides of the valve are subjected to about the same degree of pressure. Hence, the mitral tone is very faint or entirely lacking on account of the absence of a closure period. A further result of the regurgitation at the beginning of systole is that the walls of the ventricles, the conus arteriosus, and the closed aortic valves are not sufficiently nor rapidly enough stretched to produce a strong systolic tone. Consequently, either no systolic tone or only a rudimentary one is heard over the ventricle and over the beginning of the aorta. In addition, a marked mitral regurgitation may also cause diminution or absence of the second aortic tone, because the blood regurgitated into the auricle by the ventricular pressure rushes back again into the ventricle at the beginning of diastole, thus reducing below the normal the difference in pressure upon the two sides of the valves, and the tension of the valves. Other things being equal, the same reasoning applies to the right heart and to tricuspid insufficiency. All the heart tones may be diminished or entirely absent in a combined mitral and tricuspid insufficiency. The absence of the heart tones in insufficiency of the auriculoventricular valves is of double diagnostic significance. In the first place, it aids in determining the degree of insufficiency. . Mitral insufficiency with no tone over the left heart must necessarily be a serious lesion. This law applies only during the period of compensation. During disturbance of compensation the heart tones may be enfeebled on account of diminished systolic force. On the other hand the absence of heart tones may occasionally prove the existence of an auriculoventricular insufficiency which could not otherwise be diagnosed on account of the absence of a mitral murmur. If, for example, a compensated mitral stenosis is diagnosed, and it is noted that the heart tones are absent on the left side, the stenosis is probably combined with an insufficiency of the mitral valve.

Finally, in regard to the audibility of heart tones in valvular lesions accompanied by murmurs, it is to be noted that the statement so frequently made, that the tones are masked by a murmur, is based on an erroneous conception. The same change which disturbs the functions of the valves and produces the murmur may coincidentally prevent the production of a tone or diminish its force. The tones cannot, however, be masked by the murmur. Provided the tone actually exists, its sound, like a sharp blow in the midst of the slow, pulsating sound of

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the murmur, can always be detected by an experienced clinician, especially if he employs the device of holding the ear at a little distance from the stethoscope.

In the graphic representations of the results of auscultation the writer has expressed the intensity of the heart tones by the heaviness of the metrical signs and accents used to represent them. (See, for example, Fig. 114, increased second pulmonic tone.)

ALTERATIONS IN THE TIMBRE OF THE HEART TONES.

The timbre or quality of the heart tone may vary even under normal conditions. Whether the normal heart tones are simply noises or whether they resemble musical sounds depends upon a number of factors difficult to analyze, especially upon the elasticity of the valves and of the walls of the larger vessels. However, a drum-like tympanitic or singing quality is rather rare in healthy individuals. Occasionally the tones even in healthy individuals are not clear; they are not like a single short blow, but produce a rough, irregular sound. The cause of such an impurity in the tones of a normal heart has not been determined.

Marked variation in the timbre of the heart tones occurs under pathologic conditions. Thus, in arteriosclerosis the second aortic sound is not only accentuated, but possesses a peculiar ringing quality. Moreover, as has been mentioned upon pp. 250 and 251, as a result of resonance of air contained in cavities, the heart tones are not only intensified, but assume a peculiar metallic character.

The tone called "cliquetis métallique" is a noticeably rattling systolic tone heard over the ventricles when the heart action is stimulated, not only in healthy individuals, but also in heart disease, in attacks of nervous palpitation, and in every form of cardiac hypertrophy. Occasionally it can be heard at some little distance, and is probably due to the violent vibration transmitted to the chest-wall, or possibly the stomach, in consequence of the accelerated cardiac action.

Impurity or roughness of the heart sounds is observed in healthy persons, but it is more commonly due to some disease of the heart. It is frequently due to slight changes in the valve which do not necessarily disturb its function (roughness or rigidity of the curtains), but in some cases it is the result of a valvular lesion which is not sufficiently pronounced to produce the characteristic murmur. Under such circumstances an impure tone might be regarded as a rudimentary murmur; because, if the cardiac action is accelerated—*e. g.*, by directing the patient to get up and to sit down again several times in succession—the rough tone is frequently replaced by a true murmur. Autopsy findings occasionally suggest that the roughness of the systolic tone is due to changes in the muscle sound, the result of structural changes in the heart muscle (fibrosis).

APPARENT OR ACTUAL REDUPLICATION OF THE HEART TONES.

Normally only two tones are heard over every point of the heart, a systolic and a diastolic tone; but under both physiologic and pathologic

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conditions three or even four tones may be distinguished. This peculiarity may depend upon either one of two causes: either there is only an apparent reduplication—*i. e.*, under normal conditions all systolic tones and all diastolic tones occur at the same moment, but in any given case such a coincidence may be disturbed—or else there may be an actual reduplication of the sounds, due to the production of abnormal tones.

Division, Splitting, and Reduplication of Heart Sounds ($\frac{2}{4}$ time).—When two tones with a very short interval between them are heard in place of one heart tone the tone is said to be split or reduplicated. If the two tones occur very close together, we speak of a *division* or *splitting* of that heart tone, while we limit the term *reduplication* to those cases where the two tones are separated by a longer interval. The three tones do not follow each other in $\frac{3}{4}$ time, but the heart rhythm preserves the normal $\frac{2}{4}$ time. Reduplication of the first sound gives an anapest, ⏑ ⏑ — (lubb-lubb-dúpp); reduplication of the second sound, a dactyl, — ⏑ ⏑ (lúbb-dupp-dupp).[1] The double tones in splitting, which is only indistinctly separated from reduplication, are repeated so rapidly that the examiner obtains the impression of a single tone accented at the beginning or at the end. This is commonly expressed by connecting the metric signs ⏑⏑ and ⏑⏑.

This splitting or reduplication may be caused (*a*) by an imperfect coincidence of the correlated tones of the right and left heart, or (*b*) by the production of abnormal tones.

(*a*) **Splitting and Reduplication as a Result of Imperfect Coincidence of the Heart Tones.**—It is not surprising that various conditions should disturb the coincidence of the heart tones, considering the great number of factors influencing the course of the heart's action (nervous influences, variations in pressure in the different parts of the heart chambers and in the rest of the circulation). It is, on the contrary, more remarkable that any such coincidence of all systolic and all diastolic phenomena should occur at all; and suggests a very perfect balance of the cardiac mechanism.

Faulty coincidence, producing a reduplication or splitting of the first tone, is occasionally caused by a failure of the two ventricles to contract at the same moment; under such circumstances the splitting of the tone is audible over the entire heart. Splitting or reduplication of the first tone is explained in most cases by an actual increase in the number of heart tones, and will accordingly be discussed in the following subdivision (*b*, p. 257).

Faulty coincidence, producing a splitting of the second tone, is frequently observed in healthy individuals at the height of inspiration

[1] The normal accentuation of systole over the auriculoventricular valves and diastole over the great vessels is effaced by the splitting of the tones. Hence, reduplication and splitting of the first tone produces an anapestic rhythm, not only over the great vessels but also over the auriculoventricular valves. In the same way splitting of the second tone produces a dactyl, not only over the auriculoventricular valves but also over the great vessels.

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and in mitral lesions—both insufficiency and stenosis. A common explanation of the splitting of the second tone is that the aortic and pulmonary semilunar valves do not shut at the same instant. This lack of coincidence, under the above-named conditions, is supposed to be due to an abnormal difference in pressure between the aorta and pulmonary artery. High pressure closes the valves sooner than low pressure. The second part of this explanation is certainly incorrect, because even under physiologic conditions the pressure in the aorta differs enormously from that in the pulmonary artery, and because, furthermore, Ceradini has demonstrated that the closure of the semilunar valves occurs instantly and independently of the degree of pressure in the artery as soon as blood ceases to flow out of the heart. The second tone is not caused by the closure, but by the sudden tension of the closed semilunar valves, and takes place at the moment when, after a period of rest, the ventricular diastole begins. So far as any lack of coincidence of the left and right tones are concerned, splitting or reduplication of the second tone might be referred to lack of coincidence in the beginning of diastole, but such an assumption is quite unnecessary. The following explanation is more plausible: The semilunar valves, which are closed at the end of systole as a result of the marked diastolic drop in pressure within the ventricles, are suddenly forced back and made tense against the ventricle by the pressure in the aorta and pulmonary artery at the beginning of systole. This produces the diastolic tone. Any factor which tends to prevent a rapid diastolic drop in pressure within the ventricles will probably delay the corresponding second tones, while any factor which favors a drop in pressure will hasten it. As a matter of fact, such factors are present under those conditions where reduplication or splitting of the second sound exists.

Inspiration in a healthy individual, with moderately rapid respiration (see p. 119), detains the blood in the dilated pulmonary vessels, and so delays the ventricular filling. In consequence, the difference in pressure between the aorta and the left ventricle is rapidly increased, producing sudden diastolic tension of the aortic valves, and thus causing the second aortic sound to occur before the second pulmonic, as evidenced by reduplication or splitting of the second tone. The conditions are similar in mitral stenosis, where the flow of blood into the left ventricle is obstructed by the narrowed valve; consequently, the aortic valve is put under tension sooner than the pulmonic. In mitral insufficiency, on the contrary, the left ventricle is filled very rapidly during diastole, on account of the congestion in the auricle; consequently, the aortic valve is put under tension somewhat later and the second aortic tone is delayed. If this explanation is correct, the second aortic tone is hastened during inspiration of a healthy individual, as well as in mitral stenosis, but delayed in cases of mitral insufficiency. This is actually the fact, because in mitral stenosis and in healthy individuals the second part of the double tone can be perceived in the left intercostal space as a loud second pulmonic tone, while in mitral

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insufficiency, in the same place, the first part of the double tone is more distinctly heard.[1]

Reduplication of the second tone so frequently accompanies mitral lesions that it should be considered as of some diagnostic significance, provided that it can be proved to be independent of any definite phase in breathing.

(*b*) **Splitting and Reduplication as a Result of the Production of New Tones.**—In some cases there is reason to believe that the reduplication of tones is not due to failure of coincidence of the normal tones, but is caused by the production of new abnormal tones.

It is conceivable that *splitting* or *reduplication of the first tone* over the auriculoventricular valves occurs where mechanical conditions prevent the individual valve segments from being put under tension at exactly the same moment, so that they produce separate systolic tones.

In many other cases the reduplication or splitting of the first tone, which is heard only over the great vessels, might readily be assumed to consist of the normal first tone, which is increased by the tension of the closed semilunar valves, plus a second part of the tone which is produced during the expulsion time by the pulse wave in the aortic or pulmonary artery. As a matter of fact, there is no reason why the pulse wave which can produce a tone in the carotid artery should not produce such a tone in the aorta. Such a splitting of tone does not occur normally, because the closure time is so short that the tone of closure time and that of the expulsion time are merged into one. Accordingly, the reduplication or splitting of the first tone as heard only over the great vessels would depend upon a prolongation of the closure time, and thus possess some clinical significance. This explanation agrees with the physiologic reduplication of the first tone heard over the great vessels, particularly during those phases of respiration where arterial pressure is elevated (according to p. 119); that is, with slow respiration, during inspiration, with rapid respiration during expiration.

In many cases the *reduplication* or *splitting of the second tone* is also due to the production of new tones and not merely to lack of coincidence. Circumstances are conceivable where marked secondary elevations (dicrotism) due to elasticity or reflected waves of the aortic pulse may cause a supernumerary second tone. Furthermore, a new diastolic mitral tone may produce the rhythm of reduplication instead of a three-time rhythm (see p. 259).

We can usually recognize when splitting or reduplication is due to an actual increase in the number of tones by the fact that the splitting is then heard most distinctly exactly over one valve and not at some point between. The writer does not think this peculiarity is of any special diagnostic value.

[1] To prove that this explanation is correct, we should determine whether the inspiratory can be changed to an expiratory reduplication in healthy individuals breathing very slowly; because slow respiration produces exactly an opposite effect upon the ventricular filling from rapid respiration.

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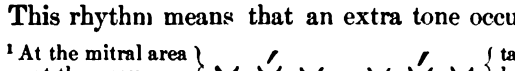
Triple Rhythm ($\frac{3}{4}$ Measure).—1. *The triple rhythm of the heart tones in mitral stenosis* consists of three approximately equal tones, heard either exclusively or most plainly at the apex and over the mitral area. There is no such grouping of two of the three tones, described above as reduplication (splitting or doubling), but the heart, instead of beating as normally, in $\frac{2}{4}$, beats in $\frac{3}{4}$ time. The accent ordinarily falls upon the second of the three tones.[1] With this rhythm the typical murmur of mitral stenosis may or may not be audible. If inaudible, it may be brought out by simply accelerating the cardiac action, and we then find that the murmur usurps the place of the first of the three tones, thus proving that the latter is an abnormal presystolic tone. The second is the normal systolic tone and corresponds to the apex beat; and the third is consequently the diastolic tone. The localization of the extra tone (the first stroke of the triple rhythm) at the auscultation area of the mitral valve is another argument for its origin from this valve. It is therefore an abnormal presystolic tone. The thickened mitral valve remains tense during ventricular diastole, and so the auricular contraction plus the valvular tension produces the tone. The adherent mitral valve in this case forms a diaphragm between the auricle and the ventricle during diastole; and just as the systolic vibration of this diaphragm occasions a systolic tone, so does a presystolic (diastolic) tension from the auricular contraction occasion a new presystolic tone. This sort of triple rhythm is readily appreciated in typical cases by the presystolic character of the first tone; and it is of diagnostic importance in recognizing cases of mitral stenosis not accompanied by a presystolic murmur (see below). One should, however, be careful not to confuse triple rhythm with another very common peculiarity of mitral lesions—viz., reduplication of the second tone, due to the imperfect coincidence of the second aortic and second pulmonic. The rhythm and the localization over the mitral valve are quite different (see p. 255) and especially distinctive of the presystolic tone.

It must, however, be stated that the diastolic mitral valve tone of mitral stenosis is not always presystolic, for it may appear at another period of diastole, namely, when tension of the mitral valve occurs not only during the presystolic contraction of the auricle, but also at any earlier period of diastole, from the recoil of the blood at the adherent mitral valve. In such cases exactly the same rhythm results as in reduplication of the second tone (p. 255 *et seq.* and p. 257); and the significance then can be determined only by its appearance over the mitral valve, as contrasted with its ordinary appearance over the base or at the apex.

2. *Gallop rhythm* is a triple rhythm heard, for the most part, over the entire heart; the tones follow at apparently equal intervals, and the second ordinarily is accentuated at the apex and the third at the great vessels; thus:

This rhythm means that an extra tone occurs during diastole—*i. e.*,



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before the normal first tone. We can ordinarily distinguish it from the presystolic tone of mitral stenosis, because it is generally to be heard equally plainly all over the heart.

Its explanation is not yet quite certain. We have no physiologic grounds for believing the theory that it is due to the fact that the contraction of the ventricle consists of two strokes. Potain[1] formerly held that the auricular contraction made the ventricular wall tense and so produced the first of the three tones; and Kriege and Schmall's[2] cardiographic representations supported his theory. Potain, however, has since[3] modified his explanation. He now considers that the extra tone which occurs during diastole, or before systole, is not due entirely to the contraction of the auricle making the heart-walls tense, but also to a sudden, abnormal passive tension of the ventricular wall during diastole, which tension is occasioned in the first place by the *vis a tergo* of the entering blood, and in the second place by the auricular contraction. The extra tone frequently coincides with presystole; but sometimes it approaches more nearly the preceding diastolic tone than the following systolic tone, and so makes the rhythm quite different. In addition to the normal systolic shock we can frequently feel or see a diastolic or presystolic shock of the anterior heart-wall, coinciding with the extra tone. This corresponds to the Kriege-Schmall cardiogram, so that the above explanation seems quite probable.

Such an explanation makes *gallop rhythm* the expression of an abnormally stimulated heart activity, which produces either an abnormally quick diastolic relaxation and a consecutive sudden passive tension of the ventricular wall from the entering blood, or an increased contraction of the auricle. Conditions which apparently are directly opposed, produce a gallop rhythm—viz., *cardiac insufficiency*, which is always connected with an irritability of the heart; and the *stimulated cardiac action* of *health*, of *exophthalmic goiter*, and of *nephritis with high-tension pulse*. Potain distinguishes between a gallop of the right and of the left heart, according to whether the rhythm is heard more plainly over the right or the left side.

Gallop rhythm, then, is a diastolic phenomenon. Potain's "*systolic gallop rhythm*" is better called a doubling of the first tone (p. 257). It is heard over the great arteries, and is due to a prolongation of the closure time.

PENDULUM OR FETAL RHYTHM OF THE HEART TONES (Embryocardia).

By this is understood a rhythm in which the pauses between the systolic and diastolic equal those between the diastolic and the systolic tones—*i. e.*, the short and the long pauses are equalized. Pendulum rhythm has been principally observed with increased tension in the arterial system (*nephritis*). Pawinski's[4] experiments seem to prove that it depends upon a prolongation of the systole, and essentially of its

[1] *Union méd.*, 1875, Nov. 11 and 18; 1876, Jan. 6 and 27; Feb. 29, Mar. 11.
[2] *Zeits. f. klin. Med.*, 1891, vol. xviii., Parts 3 and 4. [3] *Sem. méd.*, 1900, p. 22.
[4] *Deutsch. Med. Woch.*, 1891, No. 4.

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closure time, due in turn to the increased arterial pressure the ventricle has to overcome before it can open the semilunar valves.

Von Huchard designated as *embryocardia* a characteristic form of cardiac action observed in severe infectious conditions and in the terminal stages of heart disease. On account of the great rapidity of cardiac action, the duration of both pauses, as well as the resonating character of both tones, appears almost entirely equalized. Embryocardia should therefore be defined as tachycardia plus pendulum rhythm. Stokes described the same phenomenon some time ago under the name of fetal-heart tones. Its explanation is still unknown.

HEART MURMURS.

These are variable sounds heard either in addition to the tones or sometimes usurping their place. They are nearly always due to some diseased condition, but they are occasionally heard in health.

An exact distinction between *tone* and *murmur* is not essential here. Acoustically, the heart tones depend as much as the murmurs upon non-periodic sound vibrations; so that it is not correct to describe tones as consisting of regular, and murmurs of irregular, sound vibrations. An approximate periodicity or regularity of the vibrations in these sounds, and thereby an approach to a definite pitch, is doubtless far more applicable to murmurs than to tones—*e. g.*, many murmurs are best described as being musical. For practical diagnosis the distinction between *tone* and *murmur* depends chiefly upon the method of origin. The *tones* of the heart (and of the vessels) arise from a single sudden disturbance of equilibrium of the sound-producing body; the *murmurs*, on the contrary, from several repeated disturbances of equilibrium. Any evident duration in the *tone* must depend upon continued vibrations of the part due to its inertia; in the *murmur*, on the contrary, upon repeated fresh vibrations due to new applications of the moving force. Thus, a tone may be compared to the sound produced by striking once upon a drum; and a murmur, to the sound produced as long as one blows in a pipe. Acoustically, they are noises in either case, though they may approach more or less nearly to a musical tone or resonance. *Murmurs*, then, can be differentiated from *tones*, whether of the heart or of the vessels (see 284 *et seq.*), essentially by their prolongation and their lengthened resonance.

Some of the murmurs heard over the heart originate from within, some from without; the former are called *endocardial*, the latter *paracardial*.

Endocardial Murmurs.

These are of two kinds: *valvular murmurs*, depending upon a disturbance of the cardiac valves, and *accidental murmurs*, having no connection with a disturbed valve function.

Endocardial murmurs are more or less prolonged noises (thus contrasting with the heart tones, which are brief and sound sharply cut). They are very diverse in character, generally blowing or puffing, sometimes scraping, musical, even singing or whistling. The heart tones are expressed symbolically by the metric signs ⊥ and ⌣ and the

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endocardial murmurs by the crescendo and decrescendo signs < and >. Two elementary forms of murmurs may be differentiated:

1. > Decrescendo murmurs, which begin sharply and gradually fade away.

2. < Crescendo murmurs, which begin gradually and end sharply.

Two combined forms may be derived, thus:

3. <> Murmurs which begin, and fade away, gradually.

4. X Murmurs which begin and end sharply, and which in the middle are of minimum intensity.

In most cases the *endocardial murmurs* are audible only to auscultation; but exceptionally they may be heard at a distance, or even appreciated by the patient himself. These so-called distance murmurs have generally a musical, singing, or whistling character.

We are indebted to the experiments and researches of Corrigan, Kiwisch, Heynsius, Thomas Weber, Chauveau, Marey, Thann, Nolet, and others[1] for the physical explanation of the peculiarities of endocardial murmurs such as those mentioned above. Normally, the blood flows through the heart chambers without noise, so that a murmur must be attributed to some abnormality of the blood-current in the heart, as the following experiment illustrates:

If we have a glass tube (*a-b* in Fig. 103, I.) through which a stream of water is flowing, no murmur can be heard at the point *c* so long as the current is moderate. If, now, the rapidity of the current is increased by supplying more water, a continuous blowing sound can be appreciated at *c*, which simulates to a certain extent an endocardial murmur. Such a simple experiment explains the *first essential* for the origin of endocardial murmurs, namely, *the rapidity of the current*, which, other conditions remaining the same, naturally depends upon the hydraulic pressure causing the stream.

By narrowing or widening the caliber of the tube at a certain place (*c* in Fig. 103, II., III., and IV.) we possess another means of producing a murmur in the silent current, always provided the rapidity of the stream is sufficient. If in such a tube the murmur is not heard, it may be produced, or if very faint, it may be intensified by increasing the rapidity of the flow, although the same rapidity in the straight tube would furnish no sound. This, then, explains the *second essential* in the formation of endocardial murmurs—viz., *alterations in the caliber of the blood-channel.*

In order to clear up these fundamental principles let us assume, as is pictured in Fig. 103, III., that the fluid flows from a narrower into a wider part of the tube. Experimental physics teaches us that under such circumstances the fluid exerts a certain suction power upon its surroundings. This, in our supposed case, to a certain extent narrows the widened portion of the more or less elastic tube. Hence the difference in the lumen is lessened; the suction ceases, the walls of the tube bulge again, and the process is repeated over and over again as long as the current flows. Thus the current occasions lateral vibrations of the widened part of

[1] An excellent historical presentation of our knowledge of heart murmurs is found in Rosenstein's section on Heart Diseases, in *Ziemssen's Handbuch der spec. Pathol. u. Therapie*, vol. vi.

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the tube-wall; and, secondarily, of the narrower part, which alternate with those of the widened part and follow the same rhythm. Thomas Weber claims that the murmurs arise from these vibrations of the tube-wall, and that they are transmitted just as well against as with the stream. If the narrowing is sharply localized, as in Fig. 103, II., the result is the same. And even as in Fig. 103, IV., where a widened tube is suddenly and permanently narrowed, the origin of the vibrations of the wall are exactly the same, whether the current is in the direction of the lower or of the upper arrow. In the former, suction takes place as in III. In the latter, the fluid streams from the wide into the narrow tube, and the action of the pressure occasioned by the narrowing is like that of the suction in III. It is easily understood that a sufficiently rapid current will produce murmurs even in tubes of uniform caliber, as in I. For friction is always to be found in the external layers of streams, and so the layers quiescent against the wall rubbed upon by the more active middle-current layers, act quite like an infinite number of microscopic stenoses. Weber has proved in these experiments that the murmurs over the wider part of the tube always appear stronger than over the narrow. And this is easily understood when we remember that, according to Pascal's law (the

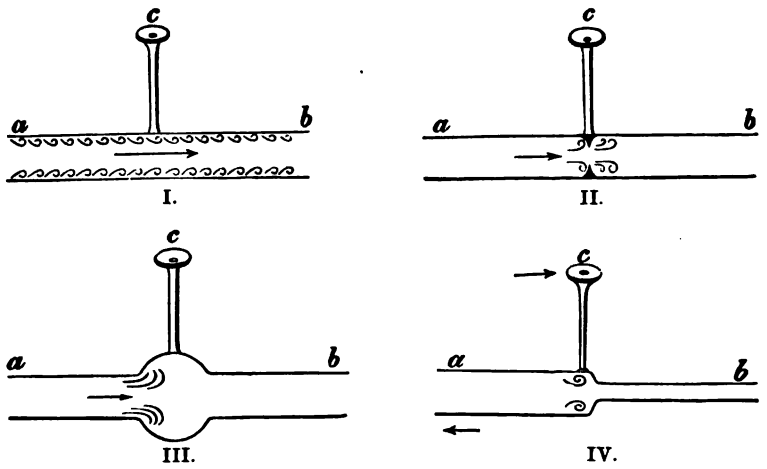

FIG. 103.—The origin of current murmurs: *a*, *b*, Current of water; *c*, stethoscope.

law of the hydraulic press), the vibrations in the wall causing the sound increase in proportion to the size of the surface vibrating between fluid and wall. This is of great importance in the explanation of the relations of transmission of the cardiac murmurs. It can be demonstrated experimentally only upon rubber tubes, since stiff tubes (of glass or metal) transmit the sound too perfectly and too far.

The following experimental facts adduced by Weber are of sufficient pathologic interest to be mentioned:

1. Murmurs arise more readily if the walls of the tube are thin than if they are thick.

2. If the inner surface of the tube is roughened, friction increases and murmurs arise more readily, requiring a less rapid current.

3. A much greater rapidity of current is necessary to give rise to murmurs in glass or brass than in yielding or distensible tubes (rubber tubes, intestines, veins).

4. Quicksilver causes murmurs more readily than water, water more readily than milk, milk more readily than blood mixed with water, heavy fluids more readily than light fluids, thin fluids more readily than tenacious fluids.

5. Sometimes the vibrations of the tube become so strong that they can be appreciated not only by the ear but also by the sense of touch. They feel like sand running over the finger.

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6. With a certain grade of rapidity of the current and narrowing of the tube a finer, more singing tone (musical murmur) can sometimes be heard.

7. Increased or diminished tension of the wall (by means of increased pressure of the fluid) has little influence upon the murmur so long as the rapidity remains the same.

8. If we gradually narrow a tube through which fluid is running, a murmur will appear after a certain degree of this compression. Increasing the compression will increase the intensity of the murmur up to a maximum, then diminish it, and finally cause it to disappear.

The diagrams make it plain that under the conditions which occasion current murmurs, whirling movements of the fluid arise at the point in question. These can be plainly demonstrated by using a glass tube and suspending a light, soluble powder (lycopodium) in the flowing liquid. It has been supposed that these whirling movements were the causes of the current murmur.[1] But such a supposition is only partially correct, because the only parts which produce actual sound must be in permanent vibration, and fluids remain in permanent vibration only with difficulty. Moreover, the approximately estimated number of vibrations of the current murmur is irreconcilable with the possible number of vibrations of the columns of fluid in question. It is, however, clear that the whirling movements are an integral part of the effect, and that they are essential to the origin of permanent vibrations of the tube-wall. They must exist together. Thomas Weber has aptly compared the part of the whirling movements to the rôle of the moving violin bow, the part of the vessel-wall vibrations to the rôle of the sounding string.

Valvular Murmurs.—Valvular Murmurs in General; Organic and Functional Valvular Murmurs.—To explain their elementary relations, it is better for the moment to neglect the part played by the current rapidity.

Valvular murmurs may arise at any valve when the blood-current, either during systole or diastole, flows into an adjoining chamber, through a narrow orifice. Such a result may happen in one of two different ways: either the valves do not open completely, forming a *stenosis*, or they do not close completely, and so allow a blood-stream flowing in an abnormal direction to escape through a narrowed opening, thus forming an *insufficiency* or *regurgitation*.

Anatomically, a *stenosis* is caused by adhesion of the individual segments of the semilunar or of the auriculoventricular valves, or else by a shrinkage of the orifice. *Insufficiency* of the valves arises from the shrinkage of the curtains in their long axes, from partial destruction or perforation of the valves, from tearing free of the entire curtain, or from the deposition of irregular new growths, which mechanically prevent a perfect closure of the valves. In any of these cases we speak of an *organic* or *anatomic valvular lesion*.

Without being anatomically diseased a valve may, however, be incapable of closing if the orifice at which it is inserted becomes dilated. This is called a *relative insufficiency*. The curtains no longer suffice to close the widened orifice, or the position of the papillary muscles is so distorted by the ventricular dilatation that the valves can no longer perfectly close. Widening of the orifice in relative insufficiency of the semilunar valves—*i. e.*, dilation of the walls of the aorta or the pulmonary artery—depends upon increased blood-pressure, upon loss of elasticity of their walls (arteriosclerosis), or upon deposition of histo-

[1] Heynsius is the chief advocate of this theory.

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logic elements in them (inflammatory swelling). On the contrary, relative insufficiency of the auriculoventricular valves, for the most part, dependent upon a diastolic distention of the ventricle, signifies too great an influx of blood from the veins, or, with insufficient cardiac power, an incomplete emptying of the ventricle. An innervation disturbance of the papillary muscles (chorea) may occasion relative insufficiency of the auriculoventricular valves. If such relative insufficiencies are not permanent, but depend upon some transitory disturbance of function (transitory dilation), they are called *functional insufficiencies.*

The blood-current producing the murmur in a valvular stenosis is directed normally; in an insufficiency, abnormally—flowing backward. So, without further explanation, it is apparent that a *murmur of stenosis* is heard during that phase of cardiac action in which the valves are opened; an *insufficiency murmur*, on the contrary, during that phase in which the valves should shut.

Compare here the following scheme:

	Systolic murmurs	Diastolic murmurs
Insufficiencies	of mitral, of tricuspid.	of aorta. of pulmonary.
Stenoses	of aorta, of pulmonary.	of mitral. of tricuspid.

Valvular lesions are, as a rule, accompanied by murmurs; yet not rarely decided lesions, which perhaps were only suspected *intra vitam* from some other clinical conditions, are found post mortem in hearts that never gave a murmur. This is easily understood when we recall that a definite minimum of current rapidity for each grade of flowing mass is essential to cause a murmur. Mitral stenosis, *e. g.*, may occur without a murmur, because the diastolic current is relatively too slow, since diastole lasts much longer than systole. For this reason a valvular lesion must be considered as a possibility in every case of heart disease whether associated with a murmur or not.

Significance of the Timbre (Quality of the Sound) and of the Loudness (Intensity) of the Valvular Murmur.—We were formerly inclined to attribute a definite diagnostic significance to the timbre of an endocardial murmur and to draw conclusions from it as to the conditions of the altered valves. It has, however, been proved that all conclusions based upon the rough, blowing, musical, whistling or singing character of the murmur are entirely untrustworthy. Such a quality varies so much with the accidental configuration of the damaged valves that we deem it of very little importance. Nevertheless, the musical or scratching character of a heart murmur certainly argues against its being of accidental nature (see p. 274 *et seq.*).

Even the *loudness of the murmur* does not necessarily correspond to the degree of a valvular lesion as some physicians are still inclined to believe, for, as the experiments with the tubes prove (see p. 263, Law 8), a certain rapidity of current is requisite to bring out a maximum murmur through a definite narrowing. If the narrowing of the tube is too slight the murmur is weak; if the narrowing is

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too pronounced the murmur is still weaker, probably on account of the great loss in energy. In every valvular lesion, whether insufficiency or stenosis, as the defect increases the murmurs increase in intensity until a maximum is attained; then, despite further increase in the valvular defect, the murmur diminishes again. Now, it is not possible in a given case to determine from the character of the murmur whether the valve lesion—*i. e.*, the narrowed channel—has already overstepped or has not yet reached its acoustic maximum, so once more no safe inference can be drawn from the loudness of the murmur. Much more trustworthy conclusions are to be deduced from carefully heeding the various cardiac functions and from other methods of examination, particularly the estimation of the size of the heart.

How little the loudness of the murmur shows the severity of a valvular lesion is proved by the clinical fact that cardiac invalids with exceptionally loud murmurs may live for years, whereas others, with scarcely audible or even no murmurs, may very soon succumb to their malady.

Murmurs vary a great deal in the same patient, depending upon the kind of cardiac activity. Even a very brief medical experience teaches that the patient's condition is by no means always the best if the murmur is scarcely audible, but frequently the worst. The first essential for murmurs mentioned in our theoretic discussion—viz., current rapidity—easily explains this seeming inconsistence. If the patient's condition is good, a vigorous current is flowing through the diseased valve, and the murmur is therefore strong and loud. If by paralysis of the cardiac power the patient's condition is impaired, the current which causes the murmur is diminished, the murmur weakens, and finally even disappears. This sometimes makes the diagnosis of valvular lesions very difficult, especially because we generally examine patients when things are not going well with them. We must therefore often withhold our opinion until our treatment has improved the patient's condition sufficiently to bring out the murmurs; or we may temporarily increase the current rapidity and so bring out a suspected murmur by causing the patient to execute some active movement, and so augmenting the heart action.

The influences external to the heart which modify the loudness of the valvular murmurs are the same as those affecting the heart tones (see p. 250).

We should always auscultate a patient in different positions, particularly standing and lying down. This is especially important in the determination of murmurs, because position has a great influence upon their character and intensity. Frequently a certain murmur can be heard only in the recumbent, another only in the erect, posture. This cannot always be satisfactorily explained by the fact that the blood-current and the blood-pressure are decidedly influenced by posture. We will refer under the individual valvular lesions to the physical relations which apply to this point.

A very loud murmur is only rarely accidental; so that although

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the loudness of a murmur is not necessarily a measure of its severity (p. 274 *et seq.*), still it is an argument against its accidental nature.

The Localization of Valvular Murmurs in Simple Valvular Lesions.—The essential requisite for an exact diagnosis of a valvular lesion consists in localizing the murmur—*i. e.*, in recognizing at what valve it arises. This is not always easy, because murmurs are heard not only where they arise, but by transmission elsewhere. In general the same rules apply to the localization of murmurs as to the localization of tones (p. 246 *et seq.*)—*i. e.*, murmurs of the mitral should be heard at the apex; of the tricuspid, at the lower end of the sternum; of the pulmonary, in the second intercostal space to the left of the sternum; and of the aortic valve, in the second intercostal space to the right of the sternum.

Exceptions to these rules are so numerous that some explanation seems necessary. If the heart chambers at either side of a narrowing are of unequal size, a stronger (louder) murmur arises over the larger than over the smaller chamber (see Weber's experiments, p. 262). If the murmur originates at some distance from the chest-wall, it may be heard more plainly at a spot upon the thorax quite remote from the point of projection of its place of origin. This depends upon its so-called *transmission* through the medium of the blood-stream, either with or against the current. If transmitted in the direction of the stream, the murmur is louder than if transmitted against the current, just as "the wind carries the sound."

1. *Systolic murmurs arising at the aortic valve* are ordinarily heard loudest at the aortic area (the auscultation spot for the aortic tones), because here the aorta lies near the thorax-wall, and because the blood-current causing the murmur is directed to that side. They are transmitted, however, strongly upward to the carotids; this latter fact may be utilized in doubtful cases in order to distinguish between mitral insufficiency and aortic stenosis. Not infrequently systolic aortic murmurs are heard plainly over the left ventricle as well—*i. e.*, at the apex. This is simple enough to understand, because they arise also in the left ventricle (p. 262).

2. Ordinarily, the diastolic murmur of *aortic insufficiency* (see p. 328) is not heard as plainly at the aortic area as further below, nearer the apex, perhaps most frequently at the middle of or to the left of the lower part of the sternum. The reasons are: first, that the aortic valves are situated deeply, at a distance from the aortic area (see p. 248); second, that the murmur arises principally in the diastolic dilated left ventricle (p. 262); and third, that the current causing the murmur is directed from the aorta to this chamber. The murmur of aortic insufficiency is also frequently to be heard over the neck vessels.

3. The systolic murmur of *mitral insufficiency* (see p. 321) is ordinarily heard best, not over the mitral area, but at the auscultation spot of the mitral tone—*i. e.*, at the apex. Despite the location of the valve and the better transmission of the murmur through the blood-current which is directed upward, the upper part of the left ventricle is so com-

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pletely covered by the right ventricle and lung that the murmur is heard plainest at the apex. Another reason is, that at the beginning of systole the left ventricle is much larger than the auricle, and so a louder sound is heard over the ventricle (*i. e.*, apex) than over the auricle. There are instances, however, where the systolic mitral murmur is best heard at the base of the heart, in the vicinity of the left auricle, to the left of the sternum. Probably this occurs chiefly when there is a marked dilatation of the left auricle. This dilatation is responsible for the fact that the murmur arises strongly over the left auricle and that the latter chamber is more closely applied to the chest-wall, the lung having been pushed aside, which naturally facilitates the appreciation of the murmur over this area. Besides, the current causing the murmur is directed to the region of the left auricle. Marked dilatation of the left ventricle also pushes the maximum point of the systolic murmur from the heart apex upward, because such a dilated left ventricle crowds the right ventricle and the lung and applies itself more completely to the thorax.

4. In *mitral stenosis* (see p. 324) the direction of the current causing the murmur is an especial reason for the sharp localization of the diastolic murmur of this valve lesion (even more than the mitral tone, see p. 248) at the apex. If faint, the diastolic murmur of mitral stenosis can very often be heard only just outside the apex. A real presystolic mitral murmur is heard loudest over the left ventricle (p. 269 *et seq.*) for the same reason, and even more, because the left auricle during presystole is small and the left ventricle large. But a pure diastolic mitral murmur (p. 270) is frequently heard best over the auricle because it arises at the beginning of diastole, when the left auricle is wide, and the left ventricle, in comparison, is still narrow.

5. The *systolic murmur of tricuspid insufficiency* (p. 335 *et seq.*), in spite of the direction of the current upward, is heard most distinctly at the tricuspid area, the lower end of the sternum or somewhat to the right of it, because here the right ventricle as well as the right auricle lies near the thoracic wall (Fig. 74). Other systolic murmurs, excepting perhaps the systolic murmur of the rare pulmonary stenosis, are not plainly transmitted to this spot.

6. The *diastolic murmur of tricuspid stenosis* (see p. 337), a rare lesion, has its maximum intensity at the tricuspid area, the lower end of the sternum, because the current from the affected valve above is directed toward this spot, and because both cavities, right auricle and right ventricle, here lie close to the thoracic wall.

7. The *systolic murmur of pulmonary stenosis* (see p. 340) is auscultated at the pulmonic area, the second left intercostal spaces and over the right ventricle.

The murmur of pulmonary stenosis is frequently heard over the greater part of the anterior surface of the heart, because the right ventricle is ordinarily so dilated in this lesion that it occupies most of this area; and because (p. 262) the murmur arises not only at the stenotic valve, but also over the right ventricle. This is a point which should be carefully heeded to prevent confusion in diagnosis.

The pulmonary stenotic murmur should be plainly transmitted into

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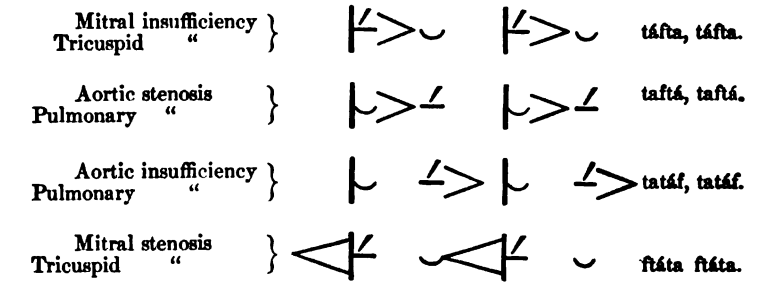
the interior of the lung; it should also be heard very distinctly beneath the left clavicle (where aortic murmurs are only faintly transmitted), or behind, between the shoulder blades, and, in contrast to the murmur of aortic stenosis, it is either not transmitted at all or only very slightly into the neck vessels.

8. The *diastolic murmur of pulmonary insufficiency* is auscultated at the pulmonic area. Like the murmur of aortic insufficiency, it can, however, be heard more plainly over the lower end of the sternum, because it arises essentially in the right ventricle (p. 262), and because the blood-stream producing the murmur is directed downward. Unlike the aortic murmur, it is not transmitted into the neck vessels.

Some of the above rules do not apply if the heart is pushed from its normal position to any extent. This must happen frequently, because valvular lesions cause marked dilatations and alterations of individual heart chambers.

Exact Time Relations of the Valvular Murmurs to the Heart Tones; Real Diastolic Murmurs, and Modified Diastolic or Presystolic Murmurs; Prediastolic Murmurs; Distinction Between the Systolic Murmurs Arising Over the Arterial Orifices and Those Over the Auriculoventricular Orifices.—The heart tones are frequently heard, as well as murmurs, both over the intact valves and over the diseased valves which give rise to the murmur. In the first place the tones may be transmitted from one of the unaffected valves; in the second place, the walls of the heart and of the great vessels participate in forming the tones; and in the third place, the diseased valve is hardly ever destroyed, and so its tone may still be produced (p. 252, *f*). By listening to the tone, a skilled examiner can determine the phase of cardiac action to which a certain murmur belongs. Everything which occurs between the beginning of the systolic tone and the next succeeding diastolic tone belongs to systole; and everything which occurs between the diastolic tone and the next systolic tone belongs to diastole. So a murmur, though very close to the systolic tone and quite remote from the diastolic tone, is still diastolic so long as it occurs before the beginning of the systolic tone—*e. g.*, a presystolic murmur is really diastolic.

The murmurs of the different valvular lesions, together with their tones in their proper time relations, are represented in the following scheme:



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The vertical lines mark the beginning of systole. This scheme shows us that the acoustic picture of a valvular lesion is a very varying one. The beginner can represent the tones sufficiently accurately by the syllables "ta-ta," the murmurs by the letter "f," as in the scheme.

Although it is plain that each pair of these eight valvular lesions are acoustically related, yet there is less confusion than one might suppose, because the murmur of every lesion has a definite point of maximum intensity and every lesion occasions distinct circulatory disturbances. A differentiation is thus possible in almost all cases.

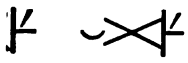
Most murmurs are represented by the decrescendo sign, because their intensity is greatest at the beginning of the phase of cardiac action to which they belong. The reason is that the blood-current is then most rapid. The diastolic murmurs of the auriculoventricular stenoses are the only exceptions, because the auricular contraction increases the current rapidity and they are represented by the crescendo sign.

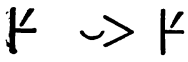
It should be noted that an actually uniform crescendo may occur during the progressive contraction of the auricle and the concomitant dilatation of the ventricle. The murmur really increases during the course of the auricular systole on account of the increasing difference between the sectional area of the contracted auriculoventricular orifice and that of the ventricle. (See Weber's *Laws of Origin of Murmurs*, p. 262.)

Tricuspid stenosis is so rare as to be practically neglected, so that a *presystolic accentuation at the end of the murmur* is practically pathognomonic of mitral stenosis. In puzzling cases this very characteristic accentuation at the end of the murmur sometimes aids in distinguishing the phases of cardiac action. Although theoretically it is not a uniformly regulated crescendo, the duration of the whole sound is so short that the ear receives the impression that it is.

Presystolic accentuation of the diastolic murmur.

Pure presystolic murmur (most common).


Diastolic murmur accentuated at the beginning and at the end of diastole.


Pure diastolic murmur (least common).

The *pure presystolic murmur* is still more common in mitral stenosis. This is represented by a short crescendo sign, for there seems to be an actual pause between the diastolic tone and the murmur. Another modification consists of an *accentuation both at the beginning and at the end of the murmur.* This depends on the fact that the blood flows into the empty ventricle with greater rapidity at the beginning of diastole than during its middle period, when the difference of pressure between the auricle and ventricle has been to a great extent equalized. The last

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modification, the *pure diastolic murmur*, is the least common. The murmur follows the diastolic tone directly and then decreases in intensity without any subsequent presystolic accentuation. These four varieties of murmurs due to auriculoventricular stenosis can be illustrated by the preceding scheme.

All these modifications of the murmurs obviously depend upon the varying relations of the factors which influence the intensity of the murmur at each moment—*i. e.*, upon the degree of the stenosis and upon the rapidity of the blood-current. Therefore it is easily understood that in one and the same case, depending upon the rapidity of the cardiac action, we hear one modification at one time and another modification at another time.[1]

We have yet to mention a characteristic sort of valvular murmur, designated as a *prediastolic murmur*, to be heard, of course, during systole—not in the beginning, but only toward its end. It may be expressed symbolically thus:

and reproduced by the sound "tá-fta." It arises at the auriculoventricular valves, and is limited to the end of systole, probably because at the beginning, and perhaps even during the greater portion of systolic contraction, the valves remain closed, and only relax and bend toward the auricle when the intraventricular pressure has reached a certain height—*i. e.*, shortly before or during the time of expulsion. This may be due to an incomplete contact of the curtains, or to an anomaly in the cordæ tendinæ or papillary muscles. If this supposition is correct, a prediastolic murmur would argue for a moderate grade of auriculoventricular insufficiency, which would be differentiated from the ordinary insufficiency by the fact that the closure time is in part or entirely retained. Ordinary systolic murmurs may be either accidental or organic, whereas prediastolic murmurs are always pathognomonic of real auriculoventricular insufficiency. It is, however, also conceivable that an insufficiency of the auriculoventricular valve produces a prediastolic murmur because the origin of the murmur is especially favored by the decided dilatation of the auricle at the end of ventricular systole. (According to Thomas Weber's *Fundamental Laws*, p. 262.)

We must call attention, finally, to the difference first noted by v. Noorden between systolic murmurs arising at the greater vessels and those arising at the

[1] It cannot be decided absolutely in a given case that these modifications are safe diagnostic signs. The question should be tested at autopsy, in order to determine whether (supposing, of course, a normal current rapidity—*i. e.*, a compensated case) the pure diastolic murmurs correspond to the slightest, the diastolic murmurs with the presystolic accentuation, to the moderate, and the pure presystolic murmurs, to the most severe grades of stenosis. Certainly this explanation is partially correct, since it is plain that with a moderate stenosis the current giving rise to the murmur will be maximal at the beginning of diastole and at its end (during the auricular contraction); whereas, with a very marked stenosis, it requires an exceptionally active contraction of the auricle to force a sufficiently rapid current into the ventricle in order to give rise to a murmur.

Sometimes in one and the same case a pure diastolic murmur is heard at the base of the heart, whereas at the apex most of what is heard is the presystolic portion (p. 267, Law 4).

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auriculoventricular orifices. With a moderate degree of auriculoventricular insufficiency the systolic murmur begins exactly at the commencement of systole, with the first tone, because the insufficiency deprives the ventricle of a proper closure time, and because the blood, overcoming the low auricular pressure, regurgitates, even at the very beginning of the ventricular contraction. It is another thing with aortic and pulmonary systolic murmurs. Here the murmur appears first at the beginning of the expulsion time—*i. e.*, after the closure time is completed, the intraventricular pressure overcomes the aortic pressure. This causes a delay of the systolic murmur as contrasted with the first tone; and though slight, a skilled ear can frequently appreciate and make use of it in diagnosing systolic murmurs of the great vessels from those of the auriculoventricular orifices. The distinction may be designated symbolically thus:

Mitral and tricuspid insufficiency.

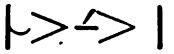
Aortic and pulmonary stenosis.

Only very close attention will detect this distinction. The closure time between the systolic tone and the murmur is so short (0.07 to 0.14 second, according to Martius,[1]) that the interval cannot be detected as a distinct pause, but merely gives an impression of a somewhat less close connection between the murmur and the tone.

We need not fear confusion with a prediastolic murmur, because the latter is separated from the systolic tone by a longer pause, which is very plain; and because the succeeding diastolic tone follows so closely that it seems a part of the murmur.

Combinations of Murmurs from Multiple Valvular Lesions.—The same valve is frequently both narrowed and insufficient; hence we may meet with the following combinations of murmurs and tones:

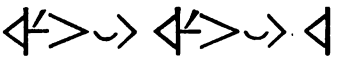
Mitral insufficiency and stenosis
Tricuspid insufficiency and stenosis } ftáfta ftáfta

Aortic insufficiency and stenosis
Pulmonary insufficiency and stenosis } taftáf taftáf 

When several valves are affected at the same time, the murmurs, if loud enough, may be transmitted over the entire heart—*e. g.*, with a combination of mitral and aortic insufficiency. In case the aortic murmur is transmitted to the apex, we may hear the following:

táftaf táftaf

If there is a mitral stenosis in addition, we shall hear at the apex this modification:



ftáftaf ftáftaf

Method of Localizing Murmurs in Multiple Valvular Lesions; Maximum and Minimum Points.—If several valves are diseased, and if one of them is doubly so—that is, with a stenosis and insufficiency—the murmurs will be transmitted in all directions, and the diagnostic

[1] *Zeit. f. klin. Med.*, vol. xiii., 1888.

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picture will become so complicated that it may be very difficult to determine where each murmur really arises and where each one is to be heard merely by transmission. If, for example, a systolic murmur is heard at two orifices—*e. g.*, at the mitral and the aortic—it is very difficult to say whether the murmur at the aorta depends upon an aortic stenosis or is merely the transmitted mitral murmur. It is very easy to make a mistake in either direction.

The best way to solve such a problem is to first represent upon a chest chart, as in Fig. 104, the two murmurs, the tones and the cardiac dulness, and then to decide, after very careful auscultation, whether any difference in the acoustic quality of the two murmurs can be detected.

Should the two murmurs exhibit a slight difference in quality—for

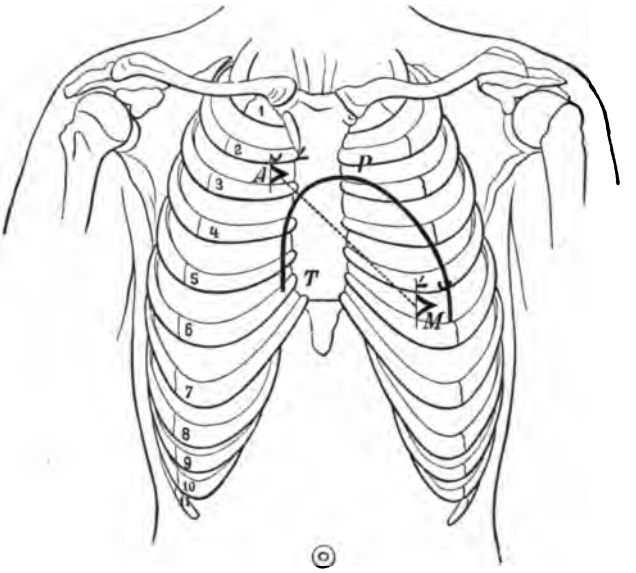

FIG. 104.

example, one rough, the other smooth; one musical or whistling, the other blowing—it is probable that each murmur has a separate origin. But one must be somewhat cautious in drawing this conclusion, as the quality as well as the intensity of a murmur may, of course, be slightly altered by transmission.

If no distinction in quality can be distinguished, a careful auscultation will usually determine that the murmur is heard more distinctly or intensely at one point than at another. Then the following possibilities will exist:

1. If the murmur is equally intense at both orifices, it is probable that a separate murmur arises at each spot, because transmission generally deprives a murmur of some of its intensity. Very loud murmurs might, however, be heard over the entire heart with equal intensity.

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2. If the murmur is heard at the two orifices with unequal intensity —*e. g.*, over the aortic much less distinctly than over the mitral—the weaker murmur would generally be transmitted. This is not necessarily so, however, as a faint murmur might arise from the aortic, and a very loud murmur from the mitral. To clear up the difficulty, we should auscultate from the one orifice to the other along the line *A M*, Fig. 105, I. If the murmur *A* is merely transmitted from *M*, its intensity will steadily increase from *A* to *M* (represented diagrammatically by a wedge-shaped figure, whose breadth at each spot is a measure of the intensity of the murmur). But if, on the contrary, in auscultating from *A* to *M*, we find that the intensity of the murmur at first diminishes, reaches a minimum somewhere between *A* and *M*, then increases again

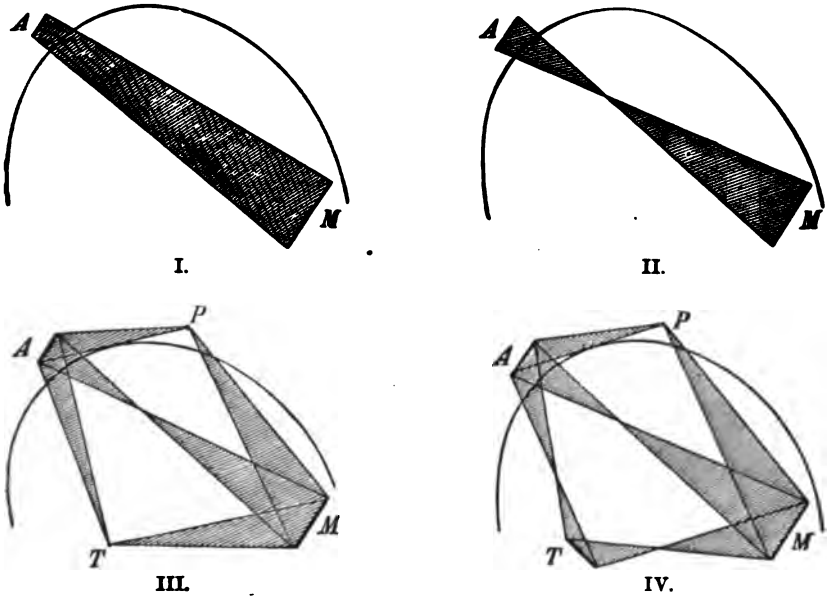

Fig 105.—Diagram of the relations of transmission of cardiac murmurs: Maximum and minimum points.

up to a second maximum at *M* (Fig. 105, II.), so that there are two maximal points, then, even though the murmur is weaker at *A* than at *M*, we can be sure that there are two different murmurs.

Supposing, as is so often the case, that the systolic murmur can be heard not only at *A* and *M*, but also (Fig. 105, III. and IV.) at *T* and *P* (tricuspid and pulmonary areas), the question arises whether at these four points we have to do with transmitted or original murmurs. Here we must again carefully heed the resonating quality and then auscultate along the lines *A P* and *A T*, *M T* and *M P*. The relations of intensity expressed in Fig. 105, III. show that the murmurs *P* and *T* are merely transmitted from *A* and *M*, whereas those in Fig. 105, IV. show that separate murmurs arise at *A*, *M* and *T*, but that the murmur at *P* is only transmitted.

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In this way the beginner learns to orient himself upon the points of origin of murmurs in complicated valvular lesions. The important point is merely to proceed systematically. If systolic and diastolic murmurs are both present, first analyze the diastolic, not heeding the systolic, and later proceed in the same way with the systolic. With diastolic murmurs the problem is often simplified by remembering that modified diastolic murmurs—*i. e.*, presystolic or presystolic accented murmurs—can arise only at the auriculoventricular valves. Another almost constant law is that aortic murmurs, especially the systolic variety, are strongly transmitted to the neck vessels.

We hardly need mention that no importance should be attached to slight differences of intensity, because they may arise from causes we do not yet comprehend. The diagnosis of a combination of an aortic and a mitral murmur which occupy the same phase of heart action requires especial attention. We not infrequently find two plainly marked maximal points with an intermediate minimum, as in Fig. 105, II., due to one single murmur; because murmurs arising from the left heart are best transmitted to the surface wherever the left heart is most closely approximated to the thoracic wall—viz., in the region of the aorta and at the apex—whereas at other places the superimposed right ventricle makes the appreciation more difficult. If a murmur is musical and of the same quality and pitch both at *A* and *M*, this explanation is pretty safe. The resonating quality in any case should therefore be closely studied. The above-mentioned difficulties render it practically impossible for auscultation alone to differentiate between the single and double origin of a murmur; hence, all the other relations should be carefully heeded.

Other Methods of Examination.—Variations in the size of the heart and changes in the circulation are quite as important in determining a valvular lesion as is the existence of murmurs.

So-called Accidental Heart Murmurs.—Many cardiac murmurs depend upon relative or functional insufficiencies of the valves, and are called *relative* or *functional* murmurs. They generally depend upon a dilatation of the valvular ring (p. 263 *et seq.*). To all intents and purposes we can consider them under the same heading as organic murmurs, and whatever we have said thus far under the latter head will apply to these functional murmurs.

Accidental murmurs, on the contrary, are entirely independent of the valvular mechanism, and, although acoustically they cannot be distinguished from the murmurs of true valvular lesions, at autopsy the valve is found to be perfectly normal.

They are frequently called *inorganic* or *functional* murmurs. "Functional" seems to the writer as incorrect a term as "inorganic," because real valvular murmurs may depend upon purely functional disturbance, and some accidental murmurs have an anatomic origin. For example, anemia is certainly an anatomic disease. Accidental murmurs have also been described as "anemic." This is inaccurate, because all accidental murmurs do not depend upon anemia, nor are all anemic murmurs accidental (relative insufficiency occasioned by anemia, p. 277). The only way to escape this confusion is to distinguish *valvular murmurs sharply from accidental murmurs*, and to separate *relative insufficiency murmurs from accidental murmurs* and to classify them with the true organic murmurs.

Although the real origin of accidental murmurs still remains unproved, the writer will attempt to state his own views upon the subject in what follows:

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Accidental murmurs are, with few exceptions, systolic in time. They are not at all uncommon in health, and are then heard for the most part either at the apex or over the pulmonary artery. Autopsies furnish negative results, so that we have no pathologic help in regard to their origin. Acoustically, they are not essentially different from valvular murmurs; therefore we assume that they, too, are current murmurs arising in conformity with the laws applied to valvular defects. In the normal heart the experimental conditions (p. 261 *et seq.*) essential to valvular murmurs are surely present, and it is certainly more astonishing that ordinarily the blood flows through the heart without murmur than that murmurs may arise in perfectly sound individuals. Consider the irregularly formed inner surface of the ventricle, the chances for friction associated with this, and the alteration in cross-section in the connecting portion between the ventricle and aorta or pulmonary artery. Indeed, it is almost more logical to attempt to explain how it is possible for tones, and not murmurs, to arise over the normal heart. Perhaps it is because the normal blood-current is not rapid enough to give rise to murmurs. Accidental murmurs would then depend upon an increased rapidity of the blood. The latter, in so far as it concerns systole, depends apparently upon the rapidity with which the ventricle contracts and upon the mass of blood to be emptied—*i. e.*, upon the amount of diastolic filling of the heart. Stolnikow[1] has demonstrated experimentally that the rapidity of the blood-current may vary a great deal. In one experiment he found that the maximum was sixteen and one-half times greater than the minimum current.

How completely the ventricle is filled during diastole appears to be of especial importance. For v. Frey and Krehl[2] found in their experiments that slowing the heart action, and thereby increasing the diastolic filling of the heart (vagus irritation or suffocation), only slightly prolongs the duration of the ventricular contraction. As a direct consequence, the pulse is slowed and the current rapidity is increased. But, so far as the writer knows, no direct experiments show that the current rapidity with low arterial pressure—*i. e.*, with slight resistance to systole—or with altered innervation of the heart can be considerably accelerated without altering the frequency of the beat or the diastolic filling. Nevertheless, this does seem probable.

Accidental murmurs may therefore arise if the diastolic filling increases while the expulsion time remains constant, or if the ventricle contracts more rapidly while the diastolic filling remains constant. The latter possibility seems possible, because accidental murmurs, like many valvular murmurs, sometimes arise when the heart action has been excited or made rapid. But even so, this does not explain why they are ordinarily to be heard over the pulmonic or over the left ventricle, and very rarely over the aortic or over the right ventricle, unless it is on account of the varying anatomic configuration of the right and left ventricles and of the conus arteriosus.

They rarely appear during diastole, because the diastolic current, on

[1] *Arch. f. Anat. u. Physiol.*, Physiologic Part, 1886. [2] *Ibid.*, 1890, No. 47.

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account of the longer duration of diastole and its weaker power, is much slower than the systolic. Besides, the diastolic current does not encounter any such alteration in the cross-section of the heart as does the systolic current in its entrance into the aorta or pulmonary artery.[1] Experience with mitral stenosis shows that the conditions in the interior of the heart are not especially favorable for the formation of diastolic murmurs, for frequently the diastolic murmur can be heard only during the presystolic accentuation of the auriculoventricular current (p. 269). Again, no other valvular lesion is so apt to run its course without a murmur as mitral stenosis (p. 264).

Accidental murmurs appear most often when the ventricle contracts quickly; therefore we should expect them most frequently in strong people with good blood-pressure, and yet the reverse of this is true. To this objection the writer can only reply that rapid contractions of the ventricle are in no way to be identified with high blood-pressure. Since the blood-pressure furnishes a resistance to the cardiac contraction, it is, on the contrary, probable that the ventricle would contract more rapidly with low pressure than with high. No special researches, so far as the writer knows, have been made upon this point. To claim that because high blood-pressure increases the friction it favors murmur formation is the same as saying that the pressure, in so far as it does not influence the rapidity of current, has little or no effect upon murmur formation. (See the experiments of Weber, Heynsius, Nolet, and Thamm, pp. 261–263.)

The question now comes up: Are we justified in believing that general weakness, fever, and anemia favor this explanation of the origin of murmurs? For under these conditions accidental murmurs are certainly very common. In so far as general weakness is combined with low blood-pressure, its effect has been already discussed. In fever the acceleration of the pulse and the decreased tension of the arteries, expressed by the sphygmogram, would make the supposition of a rapid expulsion of the blood from the heart probable. Rüedi's experiments, the so-called "tachogram" of the fever pulse, also add some weight. In regard to anemia, we must distinguish between the acute anemia of hemorrhage and the chronic anemias or oligochromemias; in the former the low blood-pressure, in the latter the diminished cohesion of the blood, probably occasions an increased flow from the heart by lessening the resistance. Cohnheim proved that artificial hydremia accelerates the blood-current, thus explaining the "venous hum" of chlorotics (see p. 285 *et seq.*). In chlorosis and other oligochromemias the diminished cohesion of the blood may also favor murmur formation (according to p. 262, Law 4).

The hypothesis advanced by many authors, that accidental murmurs depend upon an abnormal facility of vibration of the valves and of the arterial walls, seems to the author absolutely inexplicable. Whoever appreciates the fundamental difference between the much prolonged acoustic character of a murmur and the stroke-like character of a heart

[1] The difference of cross-section between the veins leading to the heart and the auricle is much less than that between the distended ventricle and its efferent arteries.

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tone will probably not be satisfied with the supposition that the tension of even a relaxed and irregular vibrating membrane can produce such a blowing murmur.

Special roughness of the walls through which the blood-current passes (p. 262, Law 2), independent of valvular disturbances, does probably play a certain part in the origin of accidental murmurs, as in atheroma of the aorta and of the endocardium. This explanation evidently cannot apply to accidental murmurs in general, however, because accidental murmurs are only rarely heard loudest over the aortic orifice; because the pulmonary artery is almost never atheromatous; and because cases presenting accidental murmurs during life show no atheroma post mortem. In certain exceptional cases, so-called accidental murmurs are nothing more than vesicular breathing (p. 221).

So much for systolic accidental murmurs. The conditions in the heart itself are very unfavorable for producing diastolic accidental murmurs; but in rare cases a diastolic murmur with its maximum over the aorta, and so easily confused with an aortic insufficiency murmur, does appear without being dependent upon any valvular lesion. In anemic individuals such a murmur is to be regarded as the diastolic accented portion of the venous hum transmitted from the jugular veins to the adjoining portions of the innominate veins and the vena cava superior to the heart (p. 285). By auscultating from the aorta upward to the jugular vein, one can generally convince one's self that the diastolic murmur, heard perhaps loudest over the aorta, gradually merges above into the rhythmic diastolic accentuation of a continuous venous hum. The severest cases of oligochromemias (with 15 to 25 per cent. hemoglobin) sometimes furnish an accidental diastolic murmur audible all over the precordia and not connected with a venous hum. There is very little more known about diastolic accidental murmurs, and in general, when diastolic, a murmur is nearly always valvular.

The most practical points for distinguishing accidental from valvular murmurs may now be profitably reviewed. If nothing points to a valvular lesion, a systolic murmur may be rightly considered accidental. By nothing the writer means no preceding etiologic factor, such as inflammatory rheumatism, no abnormality of the pulse or of the circulation, no abnormality of the tone intensity, and no demonstrable dilatation of any cardiac chamber. Anemia, fever, atheroma, or a venous hum favors the diagnosis of an accidental murmur. Still, not every murmur heard over an anemic individual's heart is necessarily accidental; for relative insufficiencies are very often caused by anemic dilatation of the heart, and can be distinguished from anatomic valvular lesions only by the fact that they diminish or disappear with the improvement of the anemia. They are limited to the mitral and tricuspid, and are governed by the same diagnostic rules as the anatomic varieties. (See the Special Diagnosis of Mitral and Tricuspid Insufficiencies.) Location of the maximum point of a doubtful systolic murmur over the pulmonic valve argues in favor of its being

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accidental. The accidental murmurs of atheroma, in accordance with the anatomic seat of the atheroma, are preferably localized at the aortic orifice, as well as frequently at the apex. Though signifying no valvular lesion, they are really of serious import, because they depend upon anatomic changes which may later occasion fatal disturbances.

Diastolic accidental murmurs are exceedingly rare, are almost always associated with a venous hum, very often with a systolic murmur, and are confined to serious anemia (oligochromemia).

It is questionable whether accidental murmurs can really be distinguished by any acoustic peculiarity from valvular murmurs. In general they are not as loud as the valvular (see p. 266); nevertheless, we do hear very loud accidental murmurs, and, conversely, faint valvular murmurs, even in serious lesions. Similarly, the particular quality of a murmur, its blowing, scraping, or musical nature, is not distinctive, although the latter two characteristics generally depend upon some abnormal configurations of the cardiac cavities or orifices and rarely appear with purely accidental murmurs. The prediastolic character of a systolic murmur argues with certainty against its being accidental, and in favor of a mild insufficiency of the auriculoventricular valve. It expresses merely a yielding of that valve during the maximum of systolic ventricular pressure. A prediastolic murmur would never be accidental (see p. 270).

Influence of Breathing Upon Endocardial Murmurs.—Whether valvular or accidental, endocardial murmurs are influenced by the phases of respiration in numerous ways. On the one hand, the extent to which the lung overlaps the heart is variable, depending upon the different phases of breathing; on the other hand, the breathing favors or hinders the current of blood through the heart.

So far as the overlapping of the lung is concerned, inspiration always weakens a murmur, because the heart is then more perfectly covered by the lung; expiration, on the contrary, intensifies it. The effect of breathing upon the blood-current is more complicated (see p. 116 *et seq.*). The entrance of blood to, and the exit from, the right heart is always favored by inspiration. In the left heart it depends upon the rapidity of breathing. During rapid breathing the entrance to, and the exit from, the left heart, in consequence of the increased capacity of the pulmonary vessels and of the retention of more blood in the lungs, is hindered by inspiration. With slow, deep respiration, however, this influence can be noted only during the first half of inspiration, because the dilatation of the pulmonary vessels diminishes the resistance in the pulmonary channels, and so must favor the passage of blood through to the left heart. Expiration works exactly in the opposite way. It retards the blood-current through the right heart somewhat, because it diminishes the negativity of the intrathoracic pressure. Yet this influence is insignificant so long as expiration is merely passive. For the left heart the effect of expiration depends upon whether it is quick or slow. With slow breathing the first part of expiration aids the flow by compressing the pulmonary vessels, while the second part hinders the filling of the left heart by increasing the resistance in the pulmonary circulation. With quick breathing, on the contrary, only the first effect of expiration is felt, and the filling of the left heart is improved. This influence is sometimes very plainly observed in valvular lesions, since any increased current through a cardiac orifice necessarily intensifies a murmur arising there. Very important points for differentiating right- and left-sided valvular lesions will sometimes be furnished by carefully heeding these facts about the influence of quick breathing. It may be further mentioned that the Valsalva experiment (see p. 280) directly diminishes the endocardial mur-

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murs of the right heart by inhibiting the venous current. It also diminishes the murmurs of the left only after an initial but transitory increase. This brief intensification is due to the pressure suddenly exerted upon the pulmonary vessels.

Paracardial Murmurs.

Under this title are included all murmurs synchronous with the heart action which depend upon changes outside of the heart chamber—*i. e.*, in the pericardium or its immediate neighborhood. They include (1) the pericardial friction rub; (2) the pleuropericardial rub; (3) the precordial emphysematous murmur; (4) the pericardial splashing.

Pericardial Rub.—Analogous to the pleural, the pericardial rub arises from the rubbing of the two pericardial surfaces when roughened by the deposition of inflammatory fibrinous or connective tissue, by tubercles, by tumors, or by abnormal dryness (in cholera). Pericardial rubs present exactly the same acoustic varieties as pleural rubs—*e. g.*, they are sometimes finely creaking, sometimes crackling, sometimes scratching. Fine pericardial friction rubs, caused by an exceptionally slight roughness of the pericardium, are sometimes confused with endocardial murmurs. To make a diagnosis in such cases (as well as conversely in exceptionally rough endocardial murmurs), it is very essential to pay close attention to the phases of cardiac action. Endocardial murmurs conform most accurately to the phases of cardiac action, coinciding with either the systolic or diastolic tone; pericardial murmurs, on the contrary, occur halfway between the tones, overstep the boundary between systole and diastole, or even quickly change phases, which endocardial murmurs never do. Sometimes pericardial murmurs are to be heard as a continuous scratching, which is merely intensified during the phases of cardiac activity. All this is perfectly comprehensible when we consider that the point of time during which a pericardial murmur is to be heard depends much less upon the phase of the heart action than upon the chance position of a roughness, which very frequently alters its form and extent. In explaining why the systolic part of the pericardial murmur does not exactly tally with the beginning of systole, Geigel noted that the greatest systolic excursion of the surface of the heart and, of course, of its pericardium does not occur during the closure, but during the expulsion time of systole, and, the writer believes, even at the termination of the latter period.

We make use of the sign ∧∧∧ to describe and picture symbolically the pericardial murmurs. The height of the teeth expresses the intensity of the murmur at a given moment. The following diagrams exhibit different types of pericardial murmurs (see also p. 268):

Pericardial rub in the middle of systole and in the middle of diastole.

Pericardial rub systolic in time but also prolonged into diastole.

Continuous scraping pericardial rub, intensified in the middle of systole and in the middle of diastole.

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Though pericardial murmurs may arise from the entire pericardium, the rubbing of the anterior surface of the heart is all that is usually heard, and more particularly of that portion uncovered by the lung or overlapped merely by a thin pulmonary covering. These murmurs are therefore heard most distinctly over the area of superficial cardiac dulness and over the sternum, and they may frequently be felt there.

Pericardial rubs depend in most cases upon inflammatory deposits. They may come quickly, change their character quickly, and disappear quickly. Their disappearance may depend (*a*) upon the retrogression of the inflammation, (*b*) upon the formation of adhesions, or (*c*) upon the collection of a fluid exudate, which separates the pericardial layers from each other. A pericardial rub may, however, persist despite the presence of an effusion, for the fluid may be mostly collected in the lateral parts of the pericardium and against the great vessels, and leave enough of the rough visceral layer exposed anteriorly to still grate upon the parietal pericardium. Again, even with large exudations, friction rubs may arise from the inferior pericardium if the weight of the heart has displaced the fluid and brought the two roughened layers in contact inferiorly.

The most trustworthy signs to distinguish pericardial rubs from endocardial murmurs are: the acoustic peculiarity of the former, its decided variability, and its lack of correspondence to the cardiac phases. Pressure of the stethoscope intensifies a rub but does not affect a murmur. Bending forward from a sitting posture in bed will accentuate a pericardial friction rub, whereas a more decided change of position is required to affect an endocardial murmur. Pericardial murmurs can be influenced by the breathing in as many ways as endocardial. Valsalva's experiment weakens or abolishes a valvular murmur, while it often intensifies a pericardial rub. This experiment is practised in the following way: After a deep inspiration the patient attempts an expiration with a closed glottis, and at the same time exerts strong abdominal pressure. This maneuver raises the intrathoracic pressure and prevents the entrance of the blood into the heart from the great veins. The test is not suitable for very sick people. Faint pericardial murmurs are not transmitted as well as are endocardial of the same intensity, because the latter are not only favored by the blood-current, but arise from each of the heart chambers bounding the lesion. Loud pericardial murmurs can, however, be heard over the entire precordia. Finally, all the other signs of pericarditis, on the one hand, and of endocarditis on the other, should in doubtful cases be utilized to clear up the diagnosis.

Pleuropericardial Rub (Extrapericardial; Pseudopericardial).—Pericardial may be confused with pleural rubs when the latter arise near the heart, because friction of pleura costalis or pulmonalis, on the one hand, and pleura pericardiaca on the other, may be brought out by movements of the heart as well as of the lung. They are called pleuropericardial, extrapericardial, or pseudopericardial rubs. One of the most essential points in differentiation is that the maximum intensity of true pericardial rubs is in the region of superficial cardiac dulness and over the sternum, whereas the extrapericardial are best heard outside this area; again, the latter possess distinct cardiac and respiratory phases, while the former are less influenced by the respiration than by the heart action. If the deposit is quite circumscribed, holding the breath in extreme inspiration or expiration will decrease or abolish pleuropericardial rubs, but not true pericardial rubs, with any degree of ease. If this diminution or disappearance occurs at the end of inspiration, the roughness will be situated upon the pleura pericardiaca, and so separated by the inflated lung edge from the corresponding roughness upon the pleura costalis (Fig. 106, *a*); whereas, if at the end of expiration, the roughness will be upon the

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pleura pericardiaca and pleura pulmonalis, which are then no longer in contact (Fig. 106, *b*). True pericardial rubs are, on the contrary, usually intensified by holding the breath in extreme inspiration, especially if abdominal pressure is exerted (Valsalva's experiment, p. 280); but this is, of course, better appreciated over the superficial cardiac dulness, where the distended lung edge does not overlap the heart.

Precordial Emphysematous Murmur.—If from the rupture of alveoli, air escapes along the interstitial pulmonary tissue to the hilus, and from there to the connective tissue of the anterior mediastinum, the superficial cardiac dulness will be diminished, the heart tones weakened, and resonating, crepitating, or metallic noises simulating râles may appear over the heart. They will be synchronous with the cardiac action, not with the respiration, and thus can be distinguished from the râles of interstitial emphysema (p. 240). From the cardiac râles which may be heard over consolidations and cavities in the neighborhood of the heart, they can be differentiated by paying attention to the relations of the cardiac dulness, of the heart tones, and of the respiratory murmur, by the demonstration of signs of a

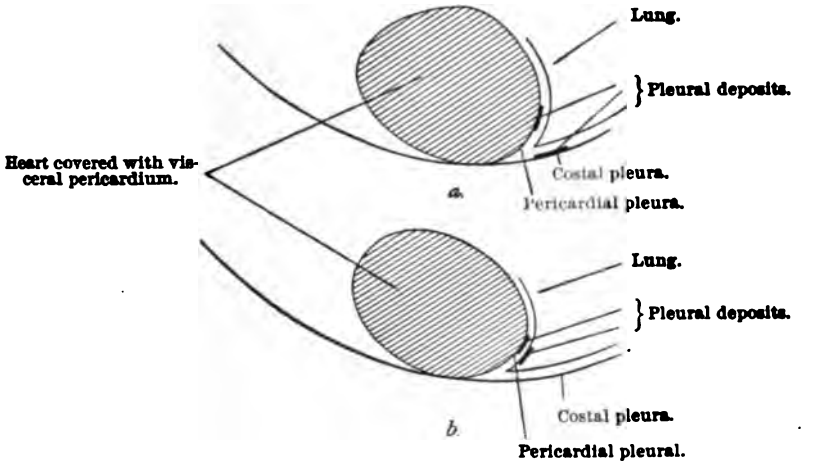

FIG. 106.—Two possibilities in pleuropericardial rubs. Diagrammatic horizontal section of the chest.

pulmonary emphysema or of an emphysema of the skin, and by the consideration of the accompanying appearances and the history.

Pericardial Splashing.—If the pericardium contains both air and fluid, a characteristic splashing noise arises synchronously with and in consequence of the heart action. It will resemble what we hear by shaking a patient with pneumothorax, and sometimes will be metallic. The heart tones will then be diminished or, by the resonance, sometimes increased (pp. 250 and 257), and in the latter event will present a metallic character. The cardiac dulness disappears in the recumbent posture, whereas in the sitting position the fluid pushes the deeper portion of the heart forward and occasions dulness. The heart action may produce similar splashing when there is a distended stomach, large cavities, or a pyopneumothorax, so that we must note the spots where the murmur is most intensely heard, the severe signs of pericarditis or pericardial perforation, the relations of the cardiac dulness, the exact conditions of the lungs, and the modifications, if any, produced by emptying and filling the stomach.

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AUSCULTATION OF THE VESSELS.

Both tones and murmurs can be heard over the vessels as well as over the heart. (See pp. 260 and 261 *et seq.* and p. 245 *et seq.* in regard to the definition of tones and murmurs and the discussion of their origin.) Some of them are transmitted from the heart. The stethoscope is always used to auscultate the vessels, and care should be taken to avoid any pressure.

AUSCULTATION OF THE ARTERIES.

The *carotid* is auscultated at the angle of the jaw or at the inner edge of the sternocleidomastoid; the *subclavian*, above the clavicle, between it and the sternocleidomastoid, or below the clavicle, in the so-called "Mohrenheim's groove," between the pectoralis major and the deltoid; the *brachial*, at the inner edge of the biceps, or at the bend of the elbow with a slightly bowed arm; the *radial*, at the place where one ordinarily feels the pulse; the *crural*, just below Poupart's ligament.

NORMAL CONDITIONS.

Two tones are heard normally over the carotid and subclavian—a systolic, from the systolic tension of the vessel-walls, and a diastolic, transmitted from the aortic valves. Over the crural and over the abdominal aorta we normally hear either nothing at all or a systolic tone. The small arteries are normally toneless.

A so-called "pressure murmur" can be produced by applying a stethoscope with some force upon the larger and even upon the smaller arteries, such as the brachial. This is frequently a very loud, vibrating, systolic murmur, and is due to the artificial stenosis of the artery. (See Auscultation of the Heart, p. 261.) If strong enough pressure is applied to occlude the lumen of the artery, a systolic "pressure tone" is produced. Pressure tones and pressure murmurs are perfectly physiologic, so that, to bring out pathologic tones or murmurs, with the exception of Duroziez's double murmur, we should never employ pressure of the stethoscope in auscultating the vessels. A systolic murmur is sometimes heard over the skull of children from the third month to the sixth year, perhaps best over the vertex; it probably arises in the internal carotid—exactly how we do not know—is normal and without diagnostic importance. [Fisher called attention to it in rickets.—ED.]

PATHOLOGIC CONDITIONS.

The diastolic and especially the systolic murmurs of aortic lesions are readily transmitted to the vessels of the neck. When the second aortic tone disappears in aortic insufficiency, ordinarily but one systolic tone can be heard over the neck vessels.

A systolic tone may be appreciated even over toneless arteries with a pulsus celer of fever or of aortic insufficiency; and with the latter a tone may be appreciated over very small arteries—*e. g.*, the radial, dorsalis pedis.

A crural double tone is occasionally audible in aortic insufficiency with an exquisite pulsus celer; then both the systolic tension and the diastolic relaxation of the arteries give rise to a plain tone. This same phenomenon has been noted, though very rarely, in chlorosis, pregnancy, and chronic lead-poisoning.

The so-called Duroziez's double murmur is more common in aortic insufficiency than the crural double tone. If the pressure of the stethoscope over the crural or brachial artery is gradually increased, the following series of sounds can be heard: without pressure the single or double arterial tone; with somewhat stronger pressure the normal systolic pressure murmur; with a farther very carefully adjusted amount of pressure the systolic murmur, followed by a second, generally much

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fainter, diastolic murmur; finally, with still more pressure, a single or double tone again. This second diastolic murmur is caused by the diastolic reflux of blood through the artificial stenosis of the crural. Aortic aneurism will sometimes furnish this double murmur if there should be a diastolic reflux of blood into the sac. It may also be found with any pulsus celer—*e. g.*, in chlorosis and exophthalmic goiter. The writer has heard it over the left lobe of the liver in a case of inflammatory liver pulse (see p. 300) by applying strong pressure with the stethoscope. A good deal of patience is required for the demonstration of the Duroziez phenomenon, because it is a question of applying just the proper amount of pressure.

A systolic murmur heard only over one subclavian artery, when the arm is hanging down and when no pressure is exerted by the stethoscope, suggests chronic disease of the apex of the corresponding lung. Pleural adhesions to the subclavian vessel sheath probably twist the artery, and so cause the murmur. Inspiration applies the thorax more closely against the stethoscope, so that we must be careful to avoid any pressure. Sometimes, however, a subclavian murmur can be heard on both sides, more rarely on one side, in perfectly healthy people, and in many persons such a murmur may also be caused artificially by certain positions of the arms which compress the artery against the clavicle or against the subclavian and pectoralis minor muscles.

Systolic murmurs heard over the arteries, especially the carotids, without stethoscopic pressure, possess some diagnostic interest. They may arise in anemia in the same way as accidental heart murmurs (p. 277), and if not transmitted from the heart they are often of the greatest importance in verifying the accidental character of a heart murmur in the same way as the venous hum (p. 285). The increase of the systolic blood-stream in the pulsus celer of aortic insufficiency, exophthalmic goiter, and chlorosis can cause murmurs over the arteries, independent of any transmission.

Localized arteriosclerosis will furnish a systolic murmur over an artery. A case of this kind was of considerable interest to the author. An old man presented a loud systolic murmur over the left carotid for months, and the diagnosis of an arteriosclerosis of that carotid was confirmed later by the origin of a left-sided cerebral thrombosis. Slight pressure of the stethoscope—not enough to cause a murmur in a normal artery—will sometimes bring out such an arteriosclerotic murmur. Litten recently emphasized their diagnostic importance, and described them, as phenomena of palpation, under the name "spritzen." They may be heard over the abdominal aorta as well as over the carotids.

Systolic and diastolic murmurs are frequently heard over *the enlarged thyroid* of exophthalmic goiter. The systolic are doubtless arterial, dependent upon a pulsus celer. It has not been determined whether the diastolic are arterial, and, like the second part of the Duroziez double murmur, a consequence of pulsus celer; or whether they are the diastolic portion of venous murmurs (p. 286), isolated and strengthened because the veins are compressed or closed by the arteries during systole.

Anyone who takes the trouble to auscultate the vessels frequently will hear sounds which have never been described and many which have not been thoroughly explained. The writer mentions as an example the occurrence of three sounds in the carotid in cases of aortic insufficiency, which together give a rhythm completely analogous to the gallop rhythm of the heart, although such a rhythm is not present in that organ. It is probably produced by a combination of the transmitted first sound of the heart with two local sounds occurring somewhat later in the artery. In aortic insufficiency the writer has also repeatedly heard a presystolic murmur—*i. e.*, presystolic in reference to the local arterial sounds—which is probably produced by the marked increase in the velocity of the current (pulsus celer) which precedes the distention of the artery.

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AUSCULTATION OF THE VEINS.

TONES OVER THE VEINS.

The blood flows through the veins normally without tones and without murmurs. The so-called venous hum is only very rarely heard in healthy individuals. The reflux blood-wave of the regurgitating venous pulse in the greater veins (especially in the jugular vein, and in the bulb) may cause a systolic tone, due to the relaxation of the valves and of the walls of the vein. This systolic bulb-valve tone can be differentiated from the synchronous systolic carotid tone only by observing that it slightly precedes the latter, forming a sort of preface (see p. 151).

MURMURS OVER THE VEINS; VENOUS HUM.

Most venous murmurs are continuous, because the current in the veins varies so little in rapidity. The most important is the so-called "venous hum," "nun's murmur," or "bruit du diable." It is heard over the jugular vein very frequently in anemic and chlorotic individuals, but rarely in health. It exhibits a characteristic continuous sound, sometimes blowing, sometimes humming, and sometimes musically whistling, with a rhythmic accentuation corresponding to systole, to diastole, and to the phase of respiration. It is heard most plainly on the right side, over the carotid, in the angle between the sternum and the clavicular portions of the sternocleidomastoid. The patient should stand erect and hold his head straight, because the recumbent posture diminishes the intensity of the murmur and sometimes causes it to disappear. Turning the head to the opposite side ordinarily increases the murmur. Pressure of the stethoscope should be avoided, for if marked pressure is exerted the murmur practically always disappears, and then the carotid tone or an artificial murmur of the stenosed carotid is all that is heard. If faint, we can sometimes distinguish only the accentuated systolic, diastolic, and inspiratory portions of the venous hum. Such an interrupted murmur may be confused with an arterial or with a respiratory murmur. Pressing very lightly with the stethoscope or turning the patient's head toward the opposite side will, however, intensify the hum and transform the interrupted murmur into a continuous roar, and so clear up the diagnosis. The diastolic accentuation of a venous hum may be transmitted to the cardiac region and simulate a diastolic accidental murmur, but auscultating from the heart to the jugular will generally distinguish the one from the other (p. 277).

To explain the venous hum we must start with the fact that it occurs, although not exclusively yet much the most commonly, in anemic individuals; and then we must turn to the general physical laws applying to the origin of murmurs in flowing currents (p. 261 *et seq.*). The two factors which are all important in producing current murmurs are: (1) the presence of abnormal narrowings or widenings in the tube, and, (2) the current rapidity. To explain the venous hum in anemic individuals, the hypothesis has been advanced that the jugular veins collapse in consequence of a diminished mass of blood, whereas the

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bulbus remains distended in virtue of its attachment to the cervical fascia. An abnormally pronounced change of lumen thus results between the vein and the bulb, and so produces the murmur. This explanation is certainly incorrect, because, in the first place, in those anemic individuals in whom the venous hum is most common (chlorotics), the supposition of a diminished blood-mass is quite erroneous, and because, in the second place, it is easy to see that in chlorosis the jugular veins are generally well filled, and indeed often abnormally so. Since we must give up the hypothesis of a change of lumen at the auscultation spot of the jugular vein as an explanation of the murmur, we must turn our attention to the second of the above-named factors—viz., the current rapidity. Have we any proof that anemic blood flows with increased rapidity? Cohnheim's experiment in submitting the mesentery of an artificially hydremic animal to direct observation proved that hydremic blood actually flows more quickly than normal blood, and probably on account of its diminished cohesion or viscosity.[1] Though anemics usually exhibit a diminution of the blood-coloring matter, their blood is not necessarily hydremic. Nevertheless, specific-gravity estimations of their blood do show that it is very frequently hydremic; and, besides, it is perfectly conceivable that the cohesion of anemic blood is diminished even without actual hydremia, and so the friction between the layer of blood against the vessel-wall and the circulating current would be diminished, and hence result in an increase of the current rapidity. An increased current rapidity would naturally offset to some extent the disadvantage of a deficient hemoglobin. The explanation that the venous hum in anemic individuals depends upon an increased rapidity of the current is certainly the most probable. Of course, this theory assumes a normal change in lumen between the jugular vein and the bulbus. The venous hum which is quite rarely observed in perfectly healthy individuals may reasonably be supposed to depend upon individual anatomic relations or conditions of distention of the vein sufficient to narrow its lumen and produce a murmur with normal current rapidity. The current rapidity of the blood may perhaps also vary considerably within normal limits.

We explain the accentuation of the hum in the standing position by the influence of gravity upon the column of venous blood; it exercises suction, thereby narrows the vein, and so hastens the jugular blood-stream. The murmur is more plainly heard upon the right side, because the right jugular vein is almost a direct linear continuation of the vena innominata dextra; whereas the left jugular empties into the innominata sinistra at an oblique angle, and so there is less obstacle to the current on the right side. By turning the head to the opposite side, the upper part of the vein is compressed by the sternocleidomastoid and omohyoid; hence this movement accentuates the venous hum. The accentuation due to the stethoscopic pressure needs no further explanation.

The rhythmic accentuation of the hum requires explanation. The

[1] *Allg. Path.*, 1882, vol. i., p. 441.

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inspiratory intensification is easily explained by the increased rapidity of the venous blood during inspiration. The systolic and diastolic accentuation are more complicated. The curve of the physiologic venous pulse (Fig. 68) seems to show that the venous current is hastened during ventricular systole; slowed, on the contrary, during diastole, judging from the small secondary oscillations in the ascending limb of the curve. Hence, the systolic accentuation of the venous hum may be explained by the systolic increase of rapidity of the blood-current, whereas the diastolic accentuation must depend upon another cause. The conditions of change of lumen which the vein suffers coincident with the ascending limb of the venous pulse—*i. e.*, diastole—at the place where the wave crest passes over into the wave trough may favor the origin of a murmur by helping the vibrations of the vessel-wall with the accompanying whirling.

Murmurs similar in character and origin to the venous hum may also be heard over the crural vein and over a vascular goiter. In the latter they may be favored by irregularity of the blood-vessels and tortuosity of the veins.

The writer attributes considerable diagnostic significance to the presence of a venous hum. If not absolutely pathognomonic, it at least suggests the presence of anemia. The demonstration of a venous hum confirms the diagnosis of an accidental heart murmur. A venous hum is also found in exophthalmic goiter, and is located most frequently over the goiter. Here, too, it probably depends upon an increased rapidity of blood-current.

AUSCULTATION OF THE ABDOMEN.

Excepting for the sounds heard over the pregnant uterus (fetal heart tones, uterine and placental bruits, and cord murmurs), described in obstetric text-books, auscultation of the abdomen is generally barren of results. (Compare the section upon Auscultation of the Vessels (p. 282 *et seq.*) in regard to the examination of the abdominal aorta.) Friction murmurs synchronous with respiration, caused by peritoneal exudations over the liver or spleen (ordinary palpable as well), have a certain importance (perihepatitic and perisplenic friction murmurs). Gerhardt claims that most cases of cholelithiasis furnish such friction sounds in the gall-bladder region. Similar friction murmurs arise over other parts of the abdomen between roughened surfaces of the peritoneum, but they are generally better appreciated by palpation than by auscultation, because they are mostly brought out by manipulation. In normal cases intestinal movements proceed so quietly that only very faint intestinal murmurs are to be heard; but in pathologic increase of peristalsis the intestinal movements can sometimes be heard at a distance, the so-called "borborygmi." Further, the coincident presence of gas and fluid in the abdominal cavity may, in moving a patient with perforative peritonitis, cause succussion murmurs in the abdomen, frequently metallic in character and similar to the *succussio Hippocratis* (p. 240). They may be sometimes heard by means of the stethoscope, sometimes at a distance. They possess a certain interest but no great diagnostic significance, since in the very diseases whose differentiation from perforative peritonitis is desired—for example, in ileus and in non-perforative peritonitis—there is ordinarily an accumulation of air and fluid in the stomach and distended intestines capable of giving rise to the same sort of a succussion murmur. The so-called "splashing noise" will be mentioned under Palpation of the Abdomen. In the diagnosis of intestinal stenosis (from tumors) the writer has several times

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been aided by the presence of a sizzling or whistling stenotic murmur, appreciable sometimes at a distance even by the patient himself, sometimes by means of a stethoscope, and sometimes by palpation. This is due to peristalsis, gas and fluid being forced through a stenosis in the intestines. A peristaltic wave or a colicky pain will point to the best time for examination. (See Auscultatory Percussion of the Abdomen, p. 185 *et seq.*)

Auscultation of the esophagus. (See the section, Examination of the Esophagus.)

PALPATION OF THE LUNGS AND PLEURA.

(In regard to the inspection of these parts, see the section, Relations of Respiration, p. 77 *et seq.*)

As has been already mentioned in several places (Râles, Rubs, Succussion Murmurs), palpation of the lungs and of the pleura serves partly to confirm symptoms recognized by auscultation, partly to furnish independent results. Examining for fluctuation, changes of resistance of the thorax, abnormal pulsations and pectoral (tactile) fremitus belong to the special province of palpation.

DETERMINATION OF FLUCTUATION AND CHANGES OF RESISTANCE IN THE THORAX.

Superficial purulent affections of the thorax or a *purulent pleurisy* which has broken through the chest-wall directly under the skin (a so-called *empyema necessitatis*) fluctuate. No real fluctuation can ever be appreciated over a serous or non-perforating purulent pleurisy, because of the tension of the intercostal soft parts.

But a kind of fluctuation or thrill (vibratory fluctuation) can be obtained over an effusion by vigorously percussing the posterior thorax, and at the same time with the other hand palpating the anterior corresponding half of the thorax (bimanual palpation percussion). The appreciation of this phenomenon requires a very nice sense of touch. The writer has noticed it in exceptional cases in sero- and pyopneumothorax, where the free mobility of the fluid permits a strong vibration wave. Here the phenomenon has some diagnostic importance, because the fluid concealed beneath the lung in a pneumothorax is often demonstrable only with considerable strength of percussion (see p. 207). The succussion murmur felt in sero- and pyopneumothorax is an accentuation of the same phenomenon.

Palpation over pleuritic exudations and the different kinds of lung infiltrations ordinarily appreciates an increase of resistance, which frequently the finger as plexor also appreciates during percussion.

ABNORMAL PULSATIONS IN THE REGION OF THE LUNGS AND PLEURA.

Pulsation over the precordia will be described later (p. 299 *et seq.*). Where pulsating tumors have pushed the lungs aside and lie against the thorax, inspection or palpation, one or both, may appreciate such pulsation over the lungs. In marked mitral lesions, especially insufficiency, one can palpate here and there through the thoracic wall diffuse pulsations of the lungs. This can be appreciated better with the ear or the single-barrel stethoscope against the chest than with the hand. The phenomenon must be distinguished from a mere mechanical shaking

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of the thorax diffusely transmitted from the heart. A pulmonary pulse may also be detected in insufficiency of the pulmonary valves (pulsus celer of the pulmonary artery). Pleuritic exudations may pulsate in the intercostal spaces (pulsating pleurisy) by transmitting the heart movements through the fluid to the thoracic spaces. This phenomenon is very rare, because the tension of the intercostal soft parts is too great unless they and the pleura itself become softened and decomposed from the inflammation, and unless the intrapleuritic tension diminishes to equal that of the atmosphere. In short, empyemas, but only very few serous exudations, pulsate.

TESTING THE VOCAL (TACTILE) FREMITUS.

By vocal (tactile) fremitus is meant the purring vibration appreciated by the hand placed upon the thorax of a person speaking or singing. It arises from the transmission of the glottis vibrations through the air column of the trachea and bronchi to the thoracic wall. Physiologically, the louder and deeper the voice, the stronger the fremitus. The fremitus frequently cannot be appreciated in women and children with high voices, in very fat people, and in patients too sick to speak aloud. The fremitus is strongest at the upper posterior parts of the thorax, over the great bronchi. From there downward and outward it gradually diminishes.

Differences in the fremitus over different places of the thorax are appreciated by comparative palpation while the patient speaks the same words aloud; for example, "one, two, three," or "ninety-nine." The ulnar edge of the hand, which is generally endowed with a very keen sense of touch, appreciates the fremitus most accurately. The naked ear applied to the thorax will serve the double purpose of auscultation and palpation. Naturally, we should only compare symmetric parts, and we should remember that the fremitus is normally somewhat stronger upon the right than upon the left side, on account of the greater breadth and the more direct branching of the right bronchus.

Similar rules apply to the increase and decrease of the fremitus as to the voice itself and to the physiologic laryngotracheal breathing murmur. The fremitus is increased wherever bronchophony appears (p. 241 *et seq.*)—*i. e.*, over infiltrations and other consolidations of the pulmonary parenchyma so long as the bronchi remain patent—over cavities and over dilated bronchi. It is diminished if the bronchi are plugged, or if gases, fluid, or solid tissues intervene between the pulmonary surface and the chest-wall (pneumothorax, pleural exudates, tumors). Increased fremitus corresponds, as a rule, to bronchial breathing and to bronchophony; diminished fremitus, to diminished breathing and lack of bronchophony. Yet frequently in one and the same case there are factors working at variance with each other for the production of bronchial breathing, bronchophony, and increased fremitus. Such factors do not always affect them equally. So, although these three signs are of the same diagnostic import, we must determine each one separately. For example, in pleuritic effusions the exudation decreases, while the pulmonary compression increases bronchophony, bronchial breathing, and fremitus; but the effect upon each of these signs is not the same,

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for frequently over a moderate-sized pleural exudation we get bronchial breathing, bronchophony, but diminished fremitus.

Fremitus is always increased over pulmonary infiltrations except when the bronchus leading to the infiltrated part is occluded by secretion, by a foreign body, or by a compressing tumor. It is then absent or decidedly diminished, although usually not permanently, as the secretion, which is the usual source of the plugging, may be coughed up.

The rule that the fremitus is diminished over fluid exudations and over pneumothorax also has exceptions, because if the exudation is small the compressed lung increases the fremitus more than the exudation diminishes it. This is rare; but increased fremitus as well as bronchial breathing is common at the upper border of the pleuritic exudate, where the fluid layer is thin and wedge-shaped (see Fig. 94, I.), whereas toward the base it is plainly weakened (see Fig. 97, I.). Finally adhesions, if membranous or string-like, can transmit the fremitus to the surface through an exudation or through a pneumothoracic cavity.

One of the helpful methods of determining the level of the fluid in a pleuritic exudation is to map out accurately the line of demarcation between this increased and decreased fremitus. In tapping a chest we may sometimes avoid circumscribed adhesions within the area of a pleuritic effusion or of a pneumothorax by testing the fremitus.

Changes in the thoracic wall also influence the fremitus. Thickening of the wall, edema, and the like, diminish it. Over differently curved portions it varies under otherwise equal conditions, so that the relations of fremitus are not trustworthy in the scoliotic or deformed chest. Changes of elasticity of the thorax decidedly influence the fremitus, so that it may be diminished over contracted portions lined internally with thickened pleura, even without any exudation.

INSPECTION AND PALPATION OF THE HEART REGION (PRECORDIA).

We shall discuss these two methods of examination together, because they are so intimately related. Marked bulgings of the heart and of the pericardium have already been described in the section upon the Shape of the Thorax (p. 34).

HEART BEAT AND APEX BEAT.

The heart beat is the visible and palpable vibration of the heart against the thorax; the heart apex beat, more simply apex beat, the portion confined to the neighborhood of the apex. Most diagnostic points are especially concerned with the apex beat. To locate the heart beat correctly, place the flat hand upon the precordia horizontally and close to the left parasternal line, with the fingers reaching to the left axillary line. The finger-tips may, at the same time, be utilized for

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more accurate localization of the apex beat. If the examiner stands in front of the patient he uses his right hand; if he stands behind the patient, the left hand. In women with large breasts the entire left mamma must be drawn up to the right. *Sp.* designates the apex beat in our diagrams.

HEART BEAT UNDER NORMAL CONDITIONS.

A vibration synchronous with the heart action, and for the most part systolic in time, can be felt over the precordia of healthy individuals. This vibration is called the heart beat, and with it a slight systolic depression to the right and above the apex can sometimes be felt. The actual apex beat is limited to a circumscribed area of this vibration (one or two neighboring intercostal spaces). It may be visible or palpable, one or both, and corresponds accurately enough to the apex of the heart—*i. e.*, to that point of the deep cardiac dulness which lies farthest to the left. If the apex of the heart is considerably overlapped by lung, the apex beat lies a little within this point. In adults it lies normally in the fifth intercostal space, somewhat inside of the mammillary or midclavicular line. In children it may be situated a space higher; in the aged, a space lower. Even in adult life its position depends a good deal upon the form of the thorax; variations of about one intercostal space may be within physiologic limits. In small children the apex beat may lie physiologically somewhat beyond the midclavicular line. The extent of the apex beat varies; ordinarily it covers an area about 2 cm. square. In conformity with the results of topographic percussion, the heart apex, during median respiration, is generally covered only by a thin layer of lung, which cannot interfere with the appreciation of the apex beat. Still, the heart apex may normally lie directly against the thoracic wall and so render the apex beat more distinct. Under pathologic conditions, the enlarged right ventricle, more or less spread out, may share in the production of the apex beat, although normally, as is well known, the heart apex is produced by the left ventricle alone.

The intensity of the apex beat varies a great deal even under physiologic conditions, depending upon the amount of overlapping lung and upon the thickness and resistance of the thoracic wall. These variations and the fact that the apex may lie behind a rib instead of at an intercostal space make it clear why quite healthy persons sometimes show no apex beat. Normal breathing does not alter the position or the intensity of the apex beat, but deep respiration generally hides it. Inspiration may depress it, due to the dropping of the diaphragm and of the heart with inspiration; expiration elevates it again.

In accordance with the passive alteration of the position of the heart described under Topographic Percussion (p. 179 *et seq.*), the apex beat can change its position, moving to the left in left-sided positions, to the right in right-sided positions, or in the latter being completely obliterated by the overlying left lung. Bending the trunk forward in the

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erect posture, and so pushing the left lung edge somewhat aside, frequently brings out the apex beat more plainly. This device is to be recommended in attempting to localize the left heart boundary when the apex beat is rather indefinite. Of course, any lateral movement of the trunk should be avoided. Similarly, deep expiration will sometimes bring out the apex beat, because the lung is thus more completely retracted. The Valsalva experiment, combining expiration with abdominal pressure, is not applicable, because it hinders the flow of venous blood to the heart, and so diminishes its size.

Stimulation to cardiac activity, either psychic stimulation or bodily exertion, intensifies and diffuses the apex beat.

According to physiologists, the essential cause of the heart beat is a projection of the heart apex and the neighboring portions of the anterior ventricular wall against the thoracic wall. Such projection is caused by the systolic change in the heart's shape. Martius' experiments demonstrated that the entire phenomenon of the heart beat occurs within the systolic "closure time" when no blood has yet left the ventricle, but still remains there under higher tension. These experiments refuted all the other theories (recoil theory, theory of the stretching of the vessels). Under pathologic conditions certain other factors besides the change of form of the anterior ventricular wall must have some influence in the causation of circumscribed or diffuse vibrations over the precordia (see p. 299 *et seq.*).

PATHOLOGIC DISLOCATION OF THE HEART BEAT.

The heart beat may be displaced by the enlargement of the heart or by its dislocation.

Displacements of the Heart Beat in Gross Cardiac Changes.—The left ventricle normally gives rise to the heart beat; dilatation of its cavity in particular will therefore displace the apex beat to the left, sometimes even out to the left axillary line. Dilatation of the right ventricle may also cause a marked displacement of the apex beat to the left, because, on the one hand, the right ventricle has some share in the formation of the apex beat, and, on the other hand, a dilated right ventricle will push the entire heart to the left (see p. 183). Therefore we have no right to diagnose a dilatation as limited to the left ventricle, because the apex beat is displaced to the left, without paying special attention to all the other pathologic signs. If such a displacement is very marked, and if at the same time there is no increase of cardiac dulness to the right, we are justified in assuming a preponderating dilatation of the left ventricle. The heart apex rests closely against the surface of the diaphragm, running obliquely downward and to the left, so that in dilatation of the left ventricle an apex beat pushed to the left will also be situated lower than normally. Dilatation of the right ventricle, on the contrary, merely dislocates the apex horizontally to the left, because an enlargement of the right ventricle supported upon the diaphragm tends to raise the heart apex (see Fig. 74). This elevation must naturally follow the

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dome of the diaphragm, under the influence of the air pressure from the abdomen. It is nevertheless doubtful if this distinction always applies, because with marked dilatation of its chamber the right ventricle forms the apex itself, which must then drop downward in the direction of the heart's axis.

Simple hypertrophy of the cardiac muscle without dilatation is never sufficiently marked to occasion any noticeable dislocation of the apex beat. (See p. 182 for exceptions to this statement.)

Only the most marked degrees of cardiac atrophy would be accompanied by or associated with a dislocation of the apex inward.

Displacements of the Heart Beat from Dislocation of the Heart.—(See the section upon Topographic Percussion, p. 188 *et seq.*)

In *situs inversus* the apex beat lies symmetrically placed upon the opposite side. In *deformities of the thorax* the apex beat may be displaced in any direction. In *emphysema* it lies lower than normally, although often the heart is so thoroughly covered by the lung that it cannot be felt at all. In *unilateral retraction* of the lung the apex beat may be drawn toward the affected side and generally upward, the latter on account of the high position of the diaphragm. *Pleuritic exudations and pneumothorax* cause a purely lateral dislocation of the apex beat (see p. 189). If the dislocation is excessive, there may be added a pendulum movement. If a left-sided effusion or pneumothorax extend around in front of the heart, the apex beat may disappear. Right-sided effusions will sometimes crowd the apex beat even to the left axillary line, and frequently to an abnormal height, in consequence of a rotation or pendulum movement, the left side of the diaphragm, of course, accompanying the apex. *Retraction of the left lung* and consequent dislocation of the mediastinum would accomplish a similar result. *Increase of the intra-abdominal pressure* from *meteorism, ascites, tumors*, etc., will force the apex upward, and frequently, in consequence of a pendulum movement, somewhat to the left (see p. 189 *et seq.*).

INTENSIFICATION AND DIFFUSION OF THE HEART BEAT.

We appreciate the intensity of the apex beat both by inspection and palpation. If the palpating finger is raised quite vigorously the apex beat is characterized as forcible or powerful. Under such circumstances it is often diffused, shaking the entire precordia, although the actual apex beat can almost always be determined by localizing a circumscribed area of more decided elevation. Such a strong movement signifies nothing but an intensification of the apex beat of no very exact import, because the strength of the beat varies so decidedly, even under physiologic conditions.

The apex beat is increased pathologically (and then ordinarily also diffused) by any *stimulated condition of heart action* (*bodily exertion, nervous palpitation, exophthalmic goiter, chronic tobacco-poisoning, fever*). Cardiac dilatation, even without any pronounced hypertrophy or increased car-

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diac activity, not only displaces and diffuses the apex beat, but also intensifies it. This seems anomalous; and so does the violent heart beat with weakened pulse which is so often observed in uncompensated heart lesions and in the so-called overexertion of the heart. But Martius' cardiographic experiments proved that the heart beat occurs during the closure time of systole, and that it is entirely independent of the power with which the ventricle empties its contents. The larger the heart, the greater the change of its form at the closure time, and consequently the more marked the apex beat, quite independent of the heart's power. In conditions of cardiac weakness the closure time is prolonged at the expense of the expulsion time, the heart only moderately diminishes during diastole, the heart-wall during the expulsion time moves from the thoracic wall only a little and quite slowly, and so the change of form during closure time is emphasized, and the heart beat seems especially strong. Conversely, it is plain that when the heart's power improves in such a case the heart beat will become weaker, because the expulsion of the blood begins earlier, is more complete, the heart becomes smaller more quickly during systole, and therefore recedes farther from the thoracic wall.

An accentuation and diffusion of the heart beat frequently signifies merely a more extensive uncovering of the heart (pulmonary retraction, high position of the diaphragm, upward dislocation of the heart, (see p. 181)).

As has been said, an abnormally powerful apex beat alone does not always signify cardiac hypertrophy. But one form of increased apex beat, F. Müller's[1] so-called "heaving beat," admits of no doubt; it always implies cardiac hypertrophy. In this, although the heart action need not be violent, and is frequently not especially diffused, yet the heart apex lifts the palpating finger with pressure and with unconquerable force. "Slow heaving beat" is a better term, as the prolongation of the beat is so noticeable. Very frequently the heart action is also slowed. These cases are characterized, according to Müller, by an exceptionally slow elevation of the cardiographic curve and by an increased systolic intracardial pressure, as well as by an increased resistance to systole. The slowness of the heaving depends upon the prolongation of the closure time, whereas the intensification of the heaving is the direct palpatory expression of increased intracardial pressure. The high intracardial pressure—*i. e.*, the resistance to the ventricular contraction—may depend upon a high arterial pressure or upon some opposition to ventricular emptying between the heart and the arteries—*e. g.*, an aortic stenosis. In any case it necessitates a cardiac hypertrophy to overcome the resistance, so that any permanently slow heaving may be regarded as a safe sign of cardiac hypertrophy (primary hypertrophy). More importance should be attributed to the slowness of the heaving than to its force and extent, in order to prevent confusion with the increased beat in cardiac insufficiency, just mentioned above. In the recognition of cardiac hypertrophy a slow heaving heart beat has about the same diagnostic significance as a continual high-tension pulse.

It is not as yet certain whether other forms of left ventricular hypertrophy which depend upon increased diastolic ventricular filling (the so-called secondary hypertrophy (see p. 316, 2)) may eventually lead to the slow heaving apex beat described above, or to a mere accentuation of the apex beat. Increased resistance, which prolongs the closure time, does not seem to exist in the secondary hypertrophy depending upon dilatation. The effect must, however, be similar, because (according to Pascal's law of the hydraulic press) so long as the arterial pressure remains equal, the total ventricular loading increases in proportion to the

[1] *Berlin. klin. Woch.*, 1895, No. 35, p. 757.

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increase of the inner surface of the ventricle. Future exact palpatory and cardiographic examinations must settle the question whether this kind of "overloading" can produce the slowly heaving apex beat, for evidently "overloading" is not identical with increased arterial resistance.[1]

F. Müller's "vibrating apex beat" differs both from the simple increased beat and from the slow heaving beat. Its impulse is more rapid and sudden, depending, according to Müller's experiments, upon a change in the form of cardiac stimulation. It is practically confined to subjective cardiac palpitation, especially the nervous form.

As soon as the major part of the heart is uncovered, the apex beat is no longer localized; but the entire precordia seems to vibrate with a wave-like movement, an undulation in which the different parts taken by the movement of the ventricles, auricles, and great vessels can often be distinguished (see p. 299 *et seq.*).

Compare p. 258 *et seq.* for the relation of the so-called gallop rhythm to the diastolic recoil of the ventricular walls.

WEAKENING OF THE HEART BEAT.

The heart beat may be weakened or may entirely disappear. Either effect may be produced by an *emphysematous lung* overlapping the heart; by *pericardial* or *left pleural effusions;* by *pneumothorax;* by *tumors* or *collections of air in the anterior mediastinum;* by *marked panniculus adiposus;* by *edema* or *emphysema of the chest-wall* (see Fig. 14).

Its disappearance on account of a pericardial effusion is diagnostically the most important of these causes. Before any conclusions can be drawn as to the presence of such an exudation, the character of the apex beat in health should have been observed, because it may not have been typical before the patient's illness. Pericardial effusions do not always hide the apex beat. The writer once saw a case in which, despite a very large pericardial exudation, the apex beat persisted, because the apex of the heart was adherent to the parietal layer of the pericardium.

[One of us observed in Dr. Evans' ward at the New York City Hospital a pneumonia patient in whom the apex beat was very plainly visible, and slightly heaving to palpation in the anterior axillary line. The autopsy revealed a "pericarditis with localized double empyema of the pericardium. The middle and lower portions of the pericardium were adherent to the heart, the upper and right portion over the right auricle was dilated and filled with several ounces of thick greenish pus; on the left side of the heart and directly over the left ventricle another sac, somewhat smaller, contained several ounces of similar pus."—ED.]

Further, as the persistence of the pericardial friction proves, a considerable pericardial effusion may collect and distend the lateral portions of the cavity before it conceals the apex beat (see p. 280).

A weakening or disappearance of the heart beat is in exceptional cases observed as a consequence of diminished *cardiac activity;* but these are mostly conditions of excessive cardiac weakness (agony, collapse). The heart beat diminishes when the cardiac power becomes so

[1] O. Frank (*Zeits. f. Biol.*, vol. xxxii.) and Moritz (*D. Arch. f. klin. Med.*, vol. lxvi.) call the loading of the ventricle caused by increased filling "loading" ("Belastung"); and that caused by arterial resistance or tension, "overloading." They show that the two factors have an entirely different importance in the heart activity. The original work must be consulted for this distinction.

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weak that the alteration in shape of the heart during closure time essential to the production of the heart beat fails. But before this point is reached the heart beat will be vigorous or even increased in spite of the diminishing cardiac power.

The diminished tension of the radial pulse is generally a far safer guide to the condition of the cardiac power than a weak heart beat, especially if we are not familiar with the patient's normal heart beat.

ABNORMAL POSITION OF THE APEX BEAT IN RELATION TO THE SUPERFICIAL CARDIAC DULNESS.

The apex beat may lie inside of the cardiac dulness, or, perhaps, more clearly expressed, the superficial cardiac dulness may extend to the left beyond the apex beat. This peculiarity may occur in a case of pericarditis, where the exudation is collected in the lateral portions of the sac and the apex beat still persists. It is not, however, a pathognomonic sign of such a condition, for it may sometimes be observed in cardiac enlargements—*e. g.*, in mitral insufficiency. In this lesion there is no true closure time, for the left ventricle begins to empty itself at the very onset of systole, and the heart-wall is not pushed as soon as normally against the chest by the systolic intraventricular pressure—in fact, not until the left heart has already contracted to some extent, and has therefore drawn the apex upward toward the base. The diastolic size of the heart, of course, determines the limits of the cardiac dulness, and so the elevated apex beat lies inside the dulness. Again, the same sign might occur if an enlarged left ventricle had compressed the lung edge enough to make it atelectatic; the apex beat would, of course, be further to the left than normal, but part of the increased dulness to the left of the apex would be formed by the compressed lung.

SYSTOLIC RETRACTION AT THE APEX.

This is sometimes observed instead of an apex beat. It can be shown to be systolic by auscultating the heart.

If noted in a healthy individual, as it sometimes is, the sign cannot be perfectly explained. The writer's theory, however, is as follows: The change of form of the heart normally causes a blow of the apex against the chest-wall; at the same time the sections of the heart lying above and within the apex retreat toward the interior of the thorax. Under physiologic conditions this would produce a palpable and visible systolic retraction of the thoracic section lying inside of the apex beat (see p. 290). If, then, for some reason or other the apex beat is absent, we should speak of a systolic retraction of the heart apex; but in reality the apex would not be systolically retracted, but, rather, a part of the anterior cardiac wall lying above and inside the apex.

A systolic retraction at the apex has been observed in many cases which have revealed later at autopsy an adhesion of the parietal and visceral pericardium or, in a few cases, an adhesion of the pericardium to the left parietal pleura. An explanation of the sign is difficult. Perhaps the cicatricial adhesions tie the heart down, so that it does not assume its normal shape and position during systole, but some other which pulls the apex backward; or perhaps some of the bands attached at the base of the heart tend to drag the apex away from the chest at each contraction of the base. The action of external atmospheric pressure is then sufficient to explain the sign of retraction at the apex, without assuming that the apex of the

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heart itself is directly attached to the thoracic wall by means of the pericardium. A systolic retraction near the apex is sometimes observed in normal cases; further, many cases of adherent pericardium show no trace of a systolic retraction during life, so we must acknowledge that this sign, although often suggestive, is by no means absolute nor pathognomonic of adherent pericardium.

DOUBLE HEART BEAT; CARDIA BIGEMINUS; HEMISYSTOLE; SYSTOLIA ALTERNANS; PSEUDOHEMISYSTOLE.

Two heart beats may under certain conditions follow one another very quickly, and pairs or groups of two heart beats be separated from other pairs by a longer pause (double stroke of the heart). There results one of three distinct forms of cardiac activity, distinguished by the titles: *cardia bigeminus*, *hemisystole*, and *systolia alternans*.[1] These three merge into each other without any sharp boundary (Unverricht[2]), and are, so to speak, genetically related. Thus far they have been observed only in heart disease, especially in mitral lesions combined with tricuspid insufficiency.

With *cardia bigeminus* a peripheral arterial pulse corresponds both to the first and to the second of the double heart beat. The former is commonly stronger than the latter, thus producing a sort of pulsus bigeminus alternans (Fig. 44). If there should be a venous pulse, each beat of the heart will also produce a distinct venous pulse. There is a regular alternation of stronger and weaker heart action; the first heart beat has more strength, probably because the preceding longer pause has enabled a more complete diastolic filling of the heart.

If the second contraction of the left heart becomes progressively weaker until it finally disappears, perhaps from some obstacle or from a nervous influence, and if at the same time both contractions of the right heart remain distinct, *hemisystole* results. In this condition, then, the right heart beats twice for each single beat of the left heart. It might be possible to diagnose such a condition in a tricuspid insufficiency, where a double heart beat was localized most plainly over the right ventricle, where the radial pulse corresponded only to the first beat, but where a positive venous pulse in the neck corresponded to both beats.

Systolia alternans arises from hemisystole when the first of the two beats of the right heart becomes weaker and finally disappears. In a tricuspid insufficiency with a regurgitating venous pulse this peculiar condition might be determined. The radial pulse unaccompanied by a venous pulse would correspond to the first beat of the heart (left), and the venous pulse unaccompanied by a radial pulse to the second beat of the heart (right). Here contraction alternates between the right and left sides of the heart.

The following scheme explains clearly the transition from one to the other:

		First heart beat.	Second heart beat.
Cardia bigeminus	Right heart.	Effective.	Effective.
	Left heart.	Effective.	Effective.
Hemisystole	Right heart.	Effective.	Effective.
	Left heart.	Effective.	Ineffectual.
Systolia alternans	Right heart.	Ineffectual.	Effective.
	Left heart.	Effective.	Ineffectual.

It is self-evident that valvular murmurs which could be localized would be of great assistance in distinguishing these three forms of heart beat, which Unverricht has so cleverly pictured as modifications, one from the other.

Most authors, however, agree with Riegel[3] and not with Unverricht. The former considers that the bigeminus is the only type of double heart beat which has

[1] For these terms the expressions, twin beat, half-beat, and altered beat of the heart may be employed.

[2] *Berlin. klin. Woch.*, 1890, No. 26.

[3] Riegel and Lachmann, *D. Arch. f. klin. Med.*, vol. xxvii., p. 393; Riegel, *Volkmann's Vorträge*, No. 227. Again, Riegel, *Zur Lehre von der Herzirregularität und Incongruenz in der Thätigkeit beider Herzhälften*, Wiesbaden, Bergmann, 1891.

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been conclusively demonstrated in man; and that the hemisystole, first described by v. Leyden,[1] and the systolia alternans assumed by Unverricht, in reality, only belong to the bigeminus.

That this is true, so far as hemisystole is concerned, is proved, according to these authors, by the fact that where an arterial pulse is not palpable for the second heart beat, it can still be appreciated in the sphygmogram; and, according to Riegel, the venous pulse suffers less than the arterial. Here the so-called hemisystole should be regarded as a bigeminus in which the second heart stroke is so weak that it is noted only in the venous pulse and in the sphygmogram, but cannot be felt at the wrist —in other words, a "*pseudohemisystole.*" Although physiologists have for a long time demonstrated hemisystole upon the exposed mammal heart, thus far there has been too little clinical observation to determine whether it does actually appear in man. Unverricht drew his conclusions[2] about systolia alternans, not from the alternation of the positive venous pulse with the radial pulse, but from less secure criteria—viz., the different location of heart tones, murmurs, and the beats in the two heart actions. The existence of systolia alternans must therefore be left undecided.

A double heart beat may quickly change into normal heart activity. We do not know yet how to explain the cause of such a peculiarity, nor its prognostic significance.

INEFFECTUAL (FUTILE) HEART CONTRACTIONS.

Quincke and Hochhaus[3] designated by this expression peculiar cardiac contractions which occur in heart disease, and which are characterized by the insertion of a beat between normal contractions, sometimes periodically, sometimes quite irregularly, and especially by the disproportion between the increased intensity of the heart beat and of the first tone, on the one hand, and the weakness of the corresponding arterial pulse on the other.

They are probably incomplete abortive cardiac contractions, with which the heart beat may be abnormally vigorous, while the arterial pulse is weak. (See Martius' explanation, p. 293.) In other words, we might consider that an identical cause produces both these transitory futile contractions and the more permanent conditions which were observed and described by Martius in the compensatory disturbances accompanied by an increased cardiac impulse. But Quincke and Hochhaus contend that this view is untenable, because the first sound in the ineffectual heart contractions is intensified and peculiarly changed in quality (drum-like). They consider it is a question not so much of a quantitative—*i. e.*, a diminution of the heart's action—as of a qualitative change, which they liken to a cardiac "spastic gait." The contractions are strong enough, but so spasmodic and so poorly coordinated that they do not accomplish the normal amount of pumping (perhaps because all parts of the ventricle do not contract simultaneously).

Further peculiarities of these futile contractions are: the diastolic tone is weakened and premature; the abortive contraction itself seems premature—*i. e.*, it follows an apparently shorter and precedes a longer diastole; the systolic and diastolic murmurs accompanying such ineffectual contractions are weakened or disappear; and the cardiogram may reveal changes which it is impossible to detail here. Not infrequently patients complain of these contractions as being painful or indefinitely unpleasant, or they may describe the sensation as that of a jolt or a blow.

HEART BLOCKING AND AURICULOVENTRICULAR ALLORRHYTHMIA.

Under this term Gaskell, for physiology, and later W. His, Jr., clinically, described a peculiarity which consists in the failure upon the part of the ventricle[4]

[1] v. Leyden, *Virchow's Archiv*, vol. xliv., 1868.
[2] *Loc. cit.*
[3] *D. Arch. f. klin. Med.*, 1894, vol. liii., p. 414.
[4] *Ibid.*, 1899, vol. lxiv., p. 316.

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to follow every contraction of the auricle. In other words, the auricle contracted oftener than the ventricle. Stannius' experiments proved that ventricular contraction is normally excited from the auricular wall by some nervous or muscular connection, so the name "heart blocking" was selected to signify some obstruction in this connection. W. His, Jr., demonstrated a more frequent auricular contraction, in a case of Stokes-Adam's disease (arteriosclerosis with brachycardia and epilepsy), by comparing the rapid venous pulse with the slow radial pulse and heart beat. The radial pulse corresponded accurately to the heart beat and heart tones, while the venous pulse, very much more rapid, corresponded to the rhythm of tones over the auricle, but without any palpable vibration of the heart region.

This phenomenon might be confused with hemisystole or pseudohemisystole (p. 296 *et seq.*). In the latter a double heart beat occurs, and one or two heart tones correspond with each venous pulse and heart beat. But in the "heart blocking" the venous pulse has no corresponding beat and tones. The ineffectual heart contraction only hurries the radial pulse, and possesses both a vigorous heart beat and strong tones. From experimental observations "heart blocking" is supposed to depend upon affection (1) of the ventricular muscle, (2) of the vagus, and (3) of the connection between the auricle and ventricle.

Auriculoventricular Allorrhythmia.—Though this has been recognized in physiology, it has not been studied as yet clinically. The auricle and ventricle here beat entirely independently of each other, not as in heart blocking.

THE CARDIOGRAM.

The cardiograph is an apparatus which conducts the movements of the heart apex beat, through hollow tubes, so as to represent them graphically upon a revolving drum. The resulting curves, so-called cardiograms, have already solved a series of very important questions

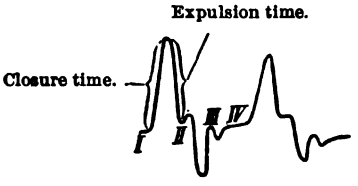

FIG. 107.—Martius' normal cardiogram: *I.*, Beginning of systole; *II.*, closure of semilunar valves; *III.* and *IV.*, recoil waves of stasis which arise in the aorta.

in heart physiology, and give promise of important conclusions for heart pathology.

Fig. 107 represents one of Martius' typical cardiograms. This author, to whose experiments [1] the writer refers the reader, explains the bearing of the apparently manifold and confused curves seen upon the drum under physiologic conditions.

[1] "Graphische Untersuchungen über die Herzbewegungen," *Zeits. f. klin. Med.*, vol. xiii., parts 3 and 4, 1888, p. 327; *Deutsch. med. Woch.*, 1888, Nos. 15 and 18; *Zeits. f. klin. Med.*, vol. xv., parts 5 and 6, 1889, and vol. xix., parts 1 and 2, 1891.

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OTHER PULSATIONS IN THE PRECORDIA AND ITS NEIGHBORHOOD.

The great vessels—aorta and pulmonary artery—are normally so thoroughly covered by the sternum and the lung that their pulsations escape appreciation. Marked dilatation of these vessels or a retraction of the normally overlapping lung makes their pulsations visible and palpable. That of the pulmonary artery appears in the second left space; that of the aorta, in the second right (Fig. 74). *Aneurisms* or the *diffused aortic dilatation* caused by aortic insufficiency (Fig. 92) are the more usual causes of the aortic pulsation, while mitral lesions are generally responsible for the pulmonic pulsation. In the latter the dilated left auricle and left ventricle push the lung aside and expose the vigorous hypertrophied right ventricle (see Fig. 90). If the lung is retracted by adhesions these pulsations may become evident even with a normal-sized heart and great vessels. If the heart is much enlarged, slight pulsations of the anterior ventricular wall are readily mistaken for pulsations of the great vessel trunks. In the former case the area over which the heart stroke is palpable would be larger and the pulsation felt earlier—*i. e.*, corresponding to the closure time of systole—while in the latter instance the pulsation would coincide with the expulsion time. This point is frequently serviceable for distinguishing a markedly dilated right ventricle from an aneurism. In case of an increased area of cardiac dulness upward, the powerful heaving character of a pulsation alone should be sufficient to show that the increased dulness is due to a widening or uncovering of the arterial trunks, and not to a dilatation of the auricle.

The systolic pulsation of an aortic aneurism may be diffused over a considerable area. This area will usually be situated near the second right intercostal space. It is frequently visible as an expansile pulsating tumor; but is, perhaps, better appreciated by the palpating hand. If, as is ordinarily the case, an aneurism proceeds from the ascending or first portion of the arch, the pulsation will generally be visible in the jugulum, and can be very plainly felt there as a pulsating tumor by crooking the finger behind the top of the sternum.

The left bronchus, which is straddled by the aorta, will be depressed by each pulsation of a large aneurism, and will in turn transmit this depression to the trachea, and so to the larynx. By grasping the cricoid cartilage between the thumb and forefinger of the right hand while facing the patient, the examiner can feel at the larynx this transmitted pulsation as a rhythmic systolic downward tug, the so-called **tracheal tug (Oliver-Cardarelli phenomenon)**. A faint tug is sometimes accentuated if the patient bends his head back and so increases the tension of the trachea. This procedure may even make the tugging visible. The sign should not be confused with the ordinary carotid pulsation, nor with the pulsation of the arteries of the thyroid gland, neither of which is strictly limited to a downward movement. The same sign has been plainly demonstrated in a *pulsating mediastinal sarcoma* and in *dilatation of the arch of the aorta*, so that it can no longer be considered absolutely pathognomonic of aneurism.

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A short, sharp blow, corresponding to the *closure of the semilunar valves*, may be felt at the base of the heart whenever the aorta and pulmonary artery, uncovered by the lung, are exposed and situated directly behind the chest-wall. It is sometimes heard even without such uncovering. It is practically never visible. Its brevity, which can be well appreciated by palpation, corresponds accurately to that of the auscultation sound of the second tone. It is to be felt most distinctly over the pulmonary artery in mitral lesions which are associated with an accentuated pulmonic second sound.

A *diffuse, feeble, systolic auricular vibration* distinct from other pulsations of the heart may sometimes be felt at the base of the heart. It comes out especially plainly when a marked auricular dilatation from mitral or tricuspid insufficiency has pushed the lung away from the base of the heart. We feel, then, not auricular contraction, but auricular diastolic filling, which in the valvular lesions mentioned above is intensified by the stasis or regurgitation at the mitral valve.

A *systolic valve shock*, the palpatory equivalent of the systolic valvular tone, and correspondingly brief, should not be confused with this auricular vibration. The former is felt in the region of the auriculoventricular valves to the right and left of the sternum, especially when marked cardiac dilatation pushes the lung aside. The ear against the stethoscope readily appreciates the difference between it and the actual heart beat; it is not really heaving, and is much shorter and sharper than the latter.

An *epigastric pulsation*, a visible, palpable, systolic shaking or lifting of the epigastrium, appears in emphysema. The diaphragm is low, the heart lies in more intimate contact with the epigastrium, the right ventricle is hypertrophied and may be dilated; hence the heart beat is transmitted directly through the diaphragm to the epigastrium. A normal heart if excited sometimes causes an epigastric pulsation.

Pulsations of the abdominal aorta in a thin patient with retracted abdominal walls and partially filled intestines often resemble an epigastric pulsation, but a careful palpation of the abdominal aorta from below upward will generally distinguish them.

Although we discussed *pulsation of the liver* in the section upon the Venous Pulse (see p. 150 *et seq.*), it should also be noted that an arterial pulse is sometimes felt over the liver (see p. 152). This occurs most frequently in aortic regurgitation as a result of the pulsus celer, and also in inflammatory conditions of the liver. The writer has observed such an arterial liver pulse in a case of infectious cholangitis.

In aortic regurgitation, as a result of the pulsus celer, the spleen may also pulsate. The *splenic pulse* is always arterial, since the splenic vein empties into the portal vein, which never pulsates, as it is cut off from the heart by the intervening liver.

The reader is also referred to what has been said about pulmonary pulsation and pulsating pleurisy (p. 288 *et seq.*).

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PALPATION OF CARDIAC MURMURS.

(Thrill; Fremissement.)

Some heart murmurs are as plainly palpable as the heart tones. They are most readily appreciated by a light touch of the finger, or of the flat hand or its ulnar edge.

Pericardial friction rubs are naturally very distinctly palpable (see p. 280 *et seq.*).

Neither accidental nor valvular endocardial murmurs can usually be appreciated by palpation unless they are very loud and vigorous. In *mitral stenosis*, however, a distinct presystolic thrill is frequently felt at the apex even when no murmur can be heard. This peculiarity is explained by the difference in the number of vibrations appreciated by the sense of hearing and that of touch. (See Auscultation with Reference to the Localization of Murmurs, etc., p. 260.)

INSPECTION AND PALPATION OF THE ABDOMEN.

(SEE the section upon the Examination of the Stomach and the Intestines, pp. 353 and 415.)

Inspection and palpation, discussed here together, are more reliable in the examination of the abdomen than percussion.

INSPECTION OF THE ABDOMEN.

This should be attempted first with the patient in the dorsal decubitus; afterward, if necessary, in the erect, and then in other positions. We should first notice any increase or decrease of the general abdominal volume, the significance of which must be controlled by percussion and palpation.

Obesity is one of the most frequent causes of abdominal enlargement, for the skin of the abdomen is a favorite place for the deposition of fat. The abdomen is then uniformly arched, when lying or standing; or the bulging may be somewhat lobulated, owing to the arrangement of fat; the navel is depressed, because the connective tissue thereabout is so rigid that fat cannot be deposited. An attempt to grasp the panniculus adiposus between the fingers will solve any doubt. Percussion elicits a dulness, the intensity of which depends upon the thickness of the fat-layer.

Edema of the abdominal wall presents the same retracted navel and may possibly be confused with a fat belly; but the almost constant existence of the edema elsewhere, its preponderance in the lateral and inferior portions as contrasted with the median deposition of fat, the pitting of the skin, and other clinical relations (p. 49) will solve any doubt.

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Meteorism (tympanites) causes abdominal distention. It lessens or obliterates the umbilical depression unless the wall is thickened by edema or fat. The contour of the stomach or of the intestinal coils can frequently be seen through thin abdominal walls, especially when peristalsis is active or when the organs are dilated, particularly if the dilatation is dependent upon a stenotic obstruction in the digestive tract (*pyloric stenosis, ileus, intestinal tumors*). The intestinal tumor in ileus is at first plainly visible; but later the distended coils, particularly those in the neighborhood of the obstruction, become paralyzed and immobile and occupy most of the abdominal surface. By noting through the abdominal wall the contour of an especially distended and completely immobile coil of intestine, the position of the obstruction can sometimes be determined. In very marked meteorism the caliber difference between the large and the small intestine cannot be appreciated. The paralytic immobility of the visible intestinal contour and the absence of intestinal murmurs to auscultation are important signs for the diagnosis of *acute diffuse peritonitis.*

Free **accumulation of air in the peritoneal cavity** from perforation of the stomach or intestine is characterized by a uniform distention of the abdomen without visible stomach or intestinal contour.

A collection of free fluid also produces an apparently uniform distention of the abdomen. In the dorsal decubitus the fluid, when under no great tension, is mostly accumulated in the lateral portions, so that the abdomen seems proportionately broad. In case the fluid reaches so high, the navel (as contrasted with its position in meteorism) protrudes slightly. Percussion of an abdomen containing free fluid elicits a dulness of the dependent portions (see p. 216). This dulness shifts with change of position. Still more essential to the diagnosis of free fluid is the so-called *fluctuation wave,* which can sometimes, when the patient is moved, be appreciated, even by inspection, as a characteristic flopping. If the tension of the abdominal contents is increased, this flopping is not visible, but frequently the peculiar way in which a patient's movement makes the abdomen fall to one side like a heavy body shows the presence of fluid, in contrast to mere meteorism.

The presence of **dilated veins in the abdominal wall** (p. 55 *et seq.*) aids in diagnosing the cause of the free fluid in the abdominal cavity. If they are limited to the sides of the abdominal wall (Fig. 17), and if an examination shows that they conduct the blood from below upward, they present the type of collateral circulation observed most plainly in *thrombosis* of the *vena cava inferior.* In this case a part of the blood which should proceed through the vena cava inferior is deflected, and flows directly from the lower extremities, through the veins of the anterior abdominal wall, into the vena cava superior. Any fluid effusion in the abdominal cavity might compress the vena cava inferior enough to cause some obstruction; hence, this sign of a collateral circulation is of slight value unless the venous dilatation is very marked. Then it would point to an actual thrombosis of the inferior vena cava. On the contrary (p. 56 *et seq.*), dilated veins

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which occupy more the middle of the abdominal wall, radiating from the umbilicus in the form of a so-called "caput Medusæ," and whose blood-current plainly flows in every direction from the umbilicus, would point to an anatomic *obstruction of the portal circulation* (*cirrhosis of the liver, thrombosis of the portal vein*). It must be acknowledged that this radiating arrangement is not a striking factor in every case of portal obstruction, but its median position and the direction of the blood-current away from the navel is constant enough to be sufficiently diagnostic (see Fig. 18). One might think that the high pressure of any effusion in the peritoneal cavity would cause a certain degree of portal stasis from its compressing action, and therefore produce a similar collateral circulation. Yet experience shows that this type of collateral circulation is practically characteristic of *cirrhosis of the liver* or of *portal thrombosis*, because the small anastomotic branches lying between the portal and the peritoneal cavity veins, and essential to the formation of this collateral circulation, are just as much compressed by ascites as the portal vein is itself (see p. 56 *et seq.*). It is a different thing with the portal stasis in a limited sense, which arises from a portal obstruction due to hepatic cirrhosis, or due to thrombosis of the portal trunk near its entrance to the liver. The collateral circulation is then undertaken by the veins of the ligamentum teres and of the serous covering of the liver.

Large ovarian tumors or *other abdominal cysts* are differentiated from effusions of free fluid by inspection, palpation, and percussion (p. 215 *et seq.*). With them the greatest degree of prominence and resistance is in the median line of the abdomen, not in the more dependent portions. But all other methods of examination must frequently be employed for a certain differentiation.

Enteroptosis presents to inspection a peculiarly characteristic and practical clinical picture. This condition depends upon a relaxation of the abdominal walls, of the mesentery, and of the parietal peritoneum; it is more frequently observed in the female sex, and is due either to a marked loss of the subperitoneal, submesenteric, and subcutaneous fat, and to an atrophy of the abdominal muscles, or, following pregnancy, to an impaired tension of the stretched abdominal walls and peritoneum. In the latter case there is almost always associated a loss of fat of the abdominal walls and of the abdominal contents, a certain atrophy, and frequently even a spreading of the recti muscles. As a consequence the abdominal organs are abnormally mobile; the liver and the kidneys in the erect posture are situated lower than normal; the right kidney especially gradually draws down a mesentery for itself and becomes a movable, or even a genuine floating, kidney. Without sufficient support the stomach and transverse colon may drop and sink deep into the abdomen. The weight of its contents causing passive distention, frequently dilates the stomach. When the physiologic support of the abdominal wall fails, the intestines become distended with gas. The skin of the abdomen becomes lean, withered, and frequently wrinkled; in patients who have born children it is covered with striæ. The walls

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are so thin that the contour of the stomach and intestines, especially in the dorsal decubitus, can be made out very plainly. In the erect posture a veritable paunch is noticed, and through the gaping breach between the recti abdominales a considerable portion of the abdominal contents projects like a hernia.

Empty intestines in **inanition**—*e. g.*, from starvation or esophageal stenosis—produce a very decided retraction of the abdomen. A similar appearance in tubercular meningitis, the so-called "scaphoid belly," is due to a contraction of the intestinal muscles, and perhaps of the abdominal muscles as well. Inspection sometimes reveals local prominences (cysts, tumors, enlargement of the abdominal organs) and their mobility with respiration.

PALPATION OF THE ABDOMEN.

METHOD OF PALPATION.

The first requisite is a thorough relaxation of the patient's abdominal walls. Therefore, if his condition permits, place the patient in the dorsal decubitus, take away everything from under his head except a very thin pillow, and get him to breathe quietly, regularly, and without exerting abdominal pressure. Sometimes flexing the knees slightly will help to relax the abdominal walls. Considerable tension of the belly, if due to meteorism, can frequently be relieved and the examination be facilitated by first emptying the colon of feces and gas with the aid of a copious water enema. Great tension and extreme sensitiveness of the abdomen may necessitate the employment of anesthesia. It should, therefore, never be neglected in any serious and doubtful case if there is a question of an operation, as the absolutely relaxed walls under anesthesia enable the examiner to make a more thorough examination.[1]

The physician himself can prevent a reflex spasm of the abdominal muscles by his method of palpation. If his hands are cold, they should be warmed before he touches the patient's belly. He should not palpate with the finger-tips, but always with the flat hand, with gradually increasing pressure. All hurried movements annoy the patient, render the examination difficult, and are therefore to be avoided. The more superficial should precede deeper palpation, because the former is less disagreeable to the patient. The expiratory retraction of the abdominal walls enables one to press the hand gradually into the depths. At first the hand should be placed upon the spot to be examined, and left there quietly during both inspiration and expiration. The way the parts under the hand move during the respiratory excursion should be noted; and only after this procedure should tactile movements be made use of to feel the entire abdominal surface. In palpating painful affections it is a good rule to begin with the painless parts, thus gaining

[1] According to the writer's view, an ether examination should be limited to the determination of some condition which might require a decided change in therapy. He emphasizes this because such examinations and exploratory laparotomies, the former sometimes not less dangerous than the latter to the patient's well-being, are often abused.

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the patient's confidence and distracting his attention. This is especially to be recommended in examining children. The examiner's hand should rest as comfortably as possible, otherwise its sensitiveness is impaired. Bimanual palpation frequently furnishes very useful results; one hand placed upon the lateral or anterior surface of the abdomen presses the parts against the other hand in the flanks, in the vagina, or in the rectum.

In all difficult cases it is of decided advantage to palpate in different positions of the body—*e. g.*, in the dorsal decubitus, the right and left lateral, the erect, and the knee-chest posture.

Rubbing the skin of the abdomen with oil or, even better, with powdered chalk frequently facilitates an examination. The palpating fingers slip over the skin more readily, and so their sensitiveness is increased. This device makes palpation less disagreeable and less painful to the patient, and is therefore to be recommended.

Jerky or interrupted palpation ("*dipping*") is often advantageous for certain purposes. The palpating hand is placed flat upon the part of the abdomen to be examined; then quite powerful but short strokes are employed with the flat hand or sometimes with the fingers. This method may demonstrate the *collection of fluid* in the abdomen, or the presence of *deeply placed solid masses* covered by fluid which ordinary palpation does not reach. In favorable cases the deeply placed solid parts seem to strike the palpating hand, because the blow excites such a great wave movement in the fluid. The same method of palpation is employed in obstetric examinations, the so-called "ballottement" of the child's head. "Dipping" can be recommended to demonstrate *enlargement of the liver or of the spleen, deep-lying tumors or a distended gall-bladder,* and for palpating an abdomen tensely distended with fluid where ordinary palpation as well as percussion leaves one in doubt.

An important rule in palpating the abdomen is to proceed very cautiously, since roughness can very easily do harm, especially in acute inflammatory affections.

GENERAL RESULTS OF ABDOMINAL PALPATION.

Abdominal palpation should appreciate the general relations of resistance in the belly (both of the walls and of the abdominal contents), the palpable boundaries of the organs, their sensitiveness to pressure, and any local resistances and tumors. Palpation then quickly confirms, completes, and corrects the results of inspection in regard to the presence of fat-accumulation, of edema in the abdominal wall, of fluid within the abdominal cavity, or of meteorism. The "fluctuation wave" is the best method of demonstrating fluid. One hand strikes a short, sharp blow upon one side, over a dependent portion where the fluid is suspected, and if fluid is really present the other hand, upon a diametrically opposite spot of the abdomen, will then feel a plain wave transmission. *Fluctuation,* in the ordinary sense of the word—*i. e.*, the transmission of slow pressure movements through the abdomen—cannot be relied

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upon to demonstrate fluid in the belly, because the normal intestines filled with air fluctuate in that sense. The distinct transmission of short blows is, on the contrary, quite characteristic of the presence of fluid rather than of gas, because the phenomenon requires a considerable mass and there is the delay of inertia in the moved substance.

Enteroptosis presents to palpation an abnormal thinness and flabbiness of the abdominal walls, and hyperesthesia. The organs may all be palpated and bounded just as if there was no covering. Tense intestinal coils and a stomach distended with gas are often much more readily outlined by palpation than by percussion, on account of their firm resistance, like an air cushion. Contracted intestinal loops feel like tubes, varying in size from the little finger to the thumb. They are generally movable enough to be rolled under the finger. The "haustra" of the large intestines can sometimes be plainly felt. In palpating abdominal organs this respiratory mobility is always important. (See Special Palpatory Relations of Certain Abdominal Affections.)

In palpating *tumors* or *pathologic resistances* we should carefully heed their position, their form, their size, the possibility of outlining their boundaries, their consistence (solid, hard, compact, elastic, fluctuating),[1] the character of their surface (smooth, nodular), and their sensitiveness to pressure. We should then determine from what organ the tumor arises; whether it is covered by the stomach or by the intestines, etc. Artificial distention of the stomach or colon will often clear up this part of the examination. The stomach can be distended either by means of an effervescing powder or by inflation through a stomach tube. (See Gastric Examination.) The colon is most conveniently inflated by means of an ordinary Davidson syringe.[2] Inflation will conceal from inspection, percussion, and palpation tumors which are situated behind the stomach or colon or in their posterior wall.

In determining *sensitiveness to pressure* very gentle palpation is first employed. If this produces no pain, more force is used. We should then determine whether there is sensitiveness to quiet and regular pressure or only to a sudden blow, for in many cases the sensitiveness is produced only by a blow, not by mere pressure. This is frequently the case in peritonitis. After we have demonstrated the existence of a sensitiveness to pressure, we should always attempt to differentiate at what depth it is situated. By pinching up the skin or the entire abdominal wall in a fold we can readily prove whether it is merely a sensitiveness of the abdominal wall and of the skin or whether it arises from the abdominal cavity. If we can determine a hyperalgesia of the unaltered skin, even to light touch or to pin prick, over the painful area in any doubtful painful affection of the abdominal

[1] There is a special sort of fluctuation, the so-called "hydatid thrill," which is sometimes appreciated over echinococcus cysts by striking a blow upon one spot of the tumor. The thrill proceeds from the impact of the daughter bladders.

[2] It is frequently necessary beforehand to cleanse and empty the rectum thoroughly with a copious enema of water.

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contents, very probably the pain in question is not alone peripheral (organic pain), but is either increased or exclusively caused by central stimulation ; in other words, the patient's suffering depends upon more than an anatomic change limited to the abdominal contents. Cutaneous hyperalgesia plays an important rôle in the diagnosis of pseudoperityphlitis. This is a painful condition of the iliocecal region which often arises psychically, especially since public attention has been more and more attracted to the vermiform appendix. (See Examination of the Nervous System ; Hyperalgesic Zones of the Skin in Diseases of Deep-lying Organs, p. 774.) Such psychical iliocecal pain is frequently responsible for an unnecessary operation.

SOURCES OF ERROR IN PALPATING THE ABDOMEN.

The abdominal walls frequently resist palpation with a vigorous reflex tension which no device can control, even under perfectly physiologic conditions. The individual parts of the abdominal wall, especially the muscular bellies and tendinous bands of the recti, vary

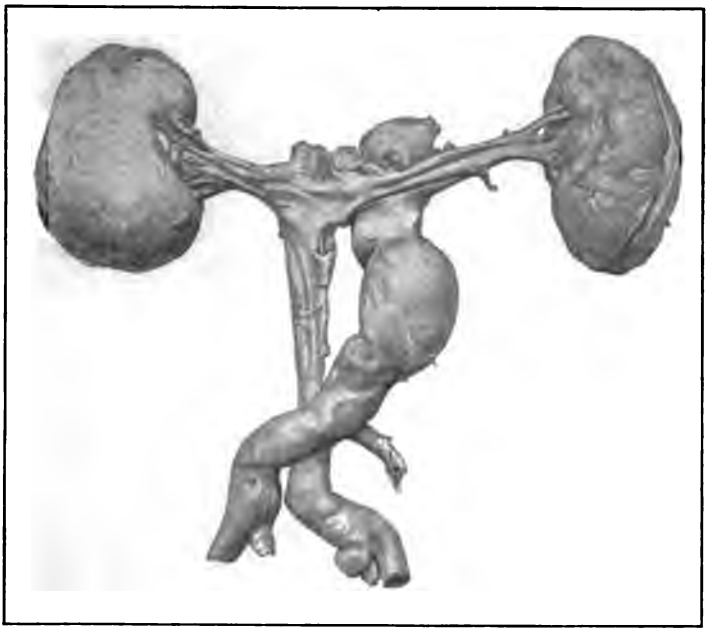

FIG. 108.—Abdominal aneurism. (Specimen from an autopsy at the New York City Hospital.)

considerably both in their tension and in their resistance, so that they sometimes produce the impression of circumscribed tumors or, at least, of pathologic resistances. This is even more striking when the section of the abdominal muscles lying directly over the affected parts becomes reflexly contracted and more tense. Abdominal pressure will facilitate the recognition of such tense muscle bellies—*e. g.*, coughing

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will make their contour still plainer. Examination during coughing or during increased abdominal pressure is an especially useful means of differentiating whether tumors or resistances are situated beneath or within the abdominal wall. Tension of the abdominal muscles from coughing will cause the resistance to disappear if beneath the abdominal wall. If situated in the wall itself, a tumor or resistance either becomes more distinct or, at least, remains just as plain during coughing. The lobules of fat of the panniculus adiposus may simulate a nodular growth within the abdomen. The same methods may be used to prevent this source of error. Contracted intestinal coils are often plainly felt through thin abdominal walls, as if they were cords (see p. 306). The string-like contracted transverse colon running across the abdomen is often mistaken for a tubercular degenerated and contracted omentum. The characteristic mobility of the transverse colon, which we can ordinarily roll under the palpating finger, the palpable recognition of the haustra, and finally the results gained by the distention of the colon from the rectum (p. 306), will generally prevent a mistake.

Fecal tumors frequently cause an error in diagnosis. These are collection of feces in the colon, sometimes in the form of large compact masses, sometimes in that of smaller tumors arranged in a row, like a string of beads. Constipation usually accompanies such phenomena; but fecal tumors are sometimes observed even in patients whose bowels move daily. They present a peculiar, somewhat doughy consistence; they can frequently be crumbled into smaller pieces; they are often arranged characteristically, like a string of beads; their position in the course of the colon and the sigmoid flexure is suggestive. The coincidence of constipation, the lack of serious symptoms, and the disappearance of the masses after purgatives or enemata should prevent an error in diagnosis. Epigastric or aortic pulsations may sometimes simulate an aneurism or a tumor (p. 300). If such a pulsation is transmitted to a tumor, the latter might be taken for a pulsating tumor. Remembering this should be sufficient to prevent the mistake.

SPECIAL RESULTS OF PALPATION IN CERTAIN AFFECTIONS OF THE ABDOMEN AND ITS ORGANS.

Inflammatory exudations are appreciated by the examiner as imperfectly or perfectly circumscribed tumor-like resistances. They are almost always passive and immovable with respiration. *Perityphlitic exudations* are inflammatory exudations characterized by their peculiar location about the cecum. In spite of the presence of pus—*i. e.*, appendicular perforation and abscess—they may exhibit a very firm consistence. A distinct palpatory differentiation between the resistance of purulent and of non-purulent appendicitis has often been attempted, but the differences are only those of degree.[1] The firm resistance which has been claimed to be characteristic of the so-called stercoral form of typhlitis does not, as was supposed, depend upon feces, but is caused by the inflammatory exudate and phlegmonous infiltration of the tissues. A firm tumor (as contrasted with a diffused resistance) is very likely due to the formation of thick adhesions and localized infiltration and exudation. Such forms are also frequently perforative, despite their gener-

[1] See the writer's paper upon "The Pathology and Treatment of Typhlitis," *der Verhandlungen des XIII. Cong. f. inn. Med. in München*, 1895, Bergmann, Wiesbaden.

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ally favorable outcome. The long-continued persistence of such a tumor, despite the lack of all serious appearances, is one of the most trustworthy signs of an abscess which for the time being remains quiescent. Wherever, despite the most serious symptoms, the resistance remains merely diffuse a grave state of affairs ordinarily exists, and, frequently enough, primary diffuse perforative peritonitis. Roux considers as an especially characteristic sign of pus formation the doughy edematous infiltration of the cecal-wall, appreciable to palpation (like thick blotting paper). Even large perityphlitic abscesses do not furnish any fluctuation until very late in their course. The abdomen itself, on account of its air contents, frequently fluctuates to a certain extent. A perityphlitic abscess lies in the depths and is surrounded by yielding walls; hence fluctuation is absent or indistinct. If the walls should be firm, the resistance from the infiltration would prevent the appreciation of fluctuation. Not infrequently we can palpate a sort of swashing, gurgling, or splashing over a perforated perityphlitic abscess, because it contains gas and fluid. But intestinal coils which contain air and fluid will furnish the same sign, so that it is not diagnostic of a gas-containing abscess unless it is very striking over an area where the presence of a large, firm, and superficial tumor makes the supposition of a normal intestinal coil improbable. A cylindric, rope-like body, corresponding in its form and its position to an enlarged appendix—*i. e.*, inside and below the cecum, at the edge of the true pelvis—will, especially if it is sensitive to pressure, support the diagnosis of a previous attack of appendicitis, and sometimes will furnish a strong argument for an interval operation. A contracted intestinal coil might cause confusion.

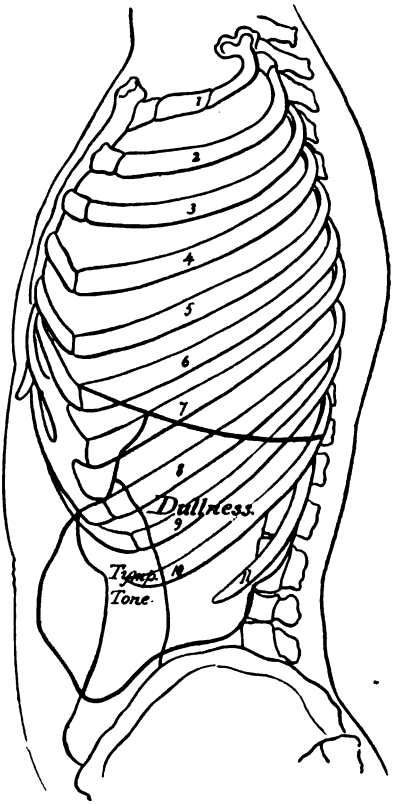

FIG. 109.—Sarcoma of the kidney, with the descending colon pushed to one side and forward.

In the anatomico-pathologic sense of the word *a tumor* is appreciated as a firm, often hard, and lumpy mass. Tumors of the intestine (those of the colon are much the commonest) are best characterized by the resulting intestinal stenosis and by their mobility, which latter, unless adhesions occur, is frequently very pronounced, much more so than with any other abdominal organ. Tumors of the stomach are generally recognized with ease, both by their position and by the accompanying gastric symptoms. They are most frequently situated at the pylorus. Despite a very widespread opinion, they should not be searched for much to the right of the median line, because, as a result of the loop form of the stomach, the pylorus reaches only slightly to the right (see Fig. 74). Moreover, tumors of the stomach frequently lead to gastric dilatation, and therefore to alterations of its position. Hence they may be found very low in the abdomen and quite exceptionally even in the pelvis. The best way to determine the relations of a *gastric tumor* is to examine the stomach both when empty and when distended with gas. (See Examination of the Stomach, p. 354.) Gastric tumors are generally but slightly movable with respiration; but there are countless exceptions.

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Renal tumors, which grow from the loins, can scarcely be pushed forward by the posterior palpating hand. Besides, while growing forward, they still remain covered by the hepatic or splenic flexures of the colon. This covering can be plainly demonstrated to percussion, palpation, and inspection (Fig. 109), by inflating the colon with gas (inserting an ordinary Davidson syringe in the rectum). If we inflate by jerks, inspection and palpation will probably show the anterior position of the colon most distinctly. Renal tumors sometimes, but not necessarily, move with respiration. *Hydronephrosis* gives rise to a renal tumor which is characterized by an elastic consistence, and sometimes also by a change in size coinciding with the alterations in the amount of urine.

Tumors of the liver and of the *spleen* are characterized by their position and by their relation to organ boundaries, which we can definitely palpate and percuss, and also by their marked mobility with respiration.

Tumors of the *mesenteric* and *retroperitoneal glands* are characterized chiefly by their multiplicity; by the rounded contour of the individual tumors; by their deep position underneath the intestines, recognized by percussion as well as by palpation; and by their etiology, for they are generally metastatic. *Tuberculous retroperitoneal and mesenteric glands* present similar conditions. *Tuberculous tumors of the peritoneum* are palpated as nodular or irregularly-defined resistances. The *tubercular infiltrated* and *retracted omentum* presents a very characteristic picture. It can be felt as a knobbed cord between the xiphoid process and the navel, running horizontally and superficially. It may sometimes closely simulate an enlarged and uneven liver. *Tumors of the bladder* and *tumors growing out from the pelvis* are characterized by their position, which can be more accurately established by means of a rectal or vaginal examination.

Diffuse enlargements or *low position of the liver* are recognized quite easily by the shape of the resistance; the determination of the sharp hepatic edge; and the respiratory mobility. The diminution in the size of the liver in *acute yellow atrophy* and in *cirrhosis* can safely be diagnosed by palpation and percussion only where the previous position of the liver edge has been known, The normal liver is, however, not always palpable, depending upon the thickness of the abdominal covering and upon the extent of the respiratory excursions. In palpating the liver border it is often very important to recognize the normal notch at the insertion of the ligamentum teres. *Enlargement of the gall-bladder* from biliary stasis, gall-stones or empyema may be characteristically localized by palpating just to the right of this notch, where we can frequently very plainly follow the sharp edge of the liver above the rounded or gourd-shaped tumor, the gall-bladder. Occasionally we can feel one very large or countless small gall-stones enclosed in the enlarged gall-bladder of a gall-stone affection. Any hard places are usually caused by an inflammatory thickening of the wall or by carcinomatous infiltration, which so frequently follows old cases of cholelithiasis. "*Dipping*"—*i. e.*, jerky palpation—over the enlarged gall-bladder will sometimes bring out a crepitating or grating of the gall-stones upon each other. If we cannot feel the enlarged gall-bladder in cholelithiasis, we frequently can feel a peculiar tongue-like projection of the liver drawn out by the enlargement of the gall-bladder and situated directly above it (Riedel) (Fig. 110). A similar or even larger projection can be determined over the corset liver (Fig. 111). Alterations in the liver consistence—*e. g.*, the characteristic induration of cirrhosis—can be easily recognized at the thin sharp liver edge, provided the patient has thin and relaxed abdominal coverings. The large lumps or nodules of the lobulated syphilitic or carcinomatous liver are still easier to feel.

It is generally easy to palpate a *dislocated* or *movable kidney*, especially if the dislocation is marked (*true floating kidney*). It is commoner upon the right side. The best method of palpation in difficult cases is with the left hand behind the loin to push against the right hand palpating the organ from in front with the abdominal walls as relaxed as possible. A movable kidney can then be recognized as a bean-shaped, vertically placed body between the two palpating hands. At times it is somewhat sensitive to pressure. At the height of inspiration it may be partially grasped between the two hands. The deep location, the verification of

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the upper bluntly rounded end of the kidney, the lack of any sharp edge which could correspond to the liver border, should prevent confusion with a corset liver. Sometimes the hilus and the pulsating renal artery on the concave side of the

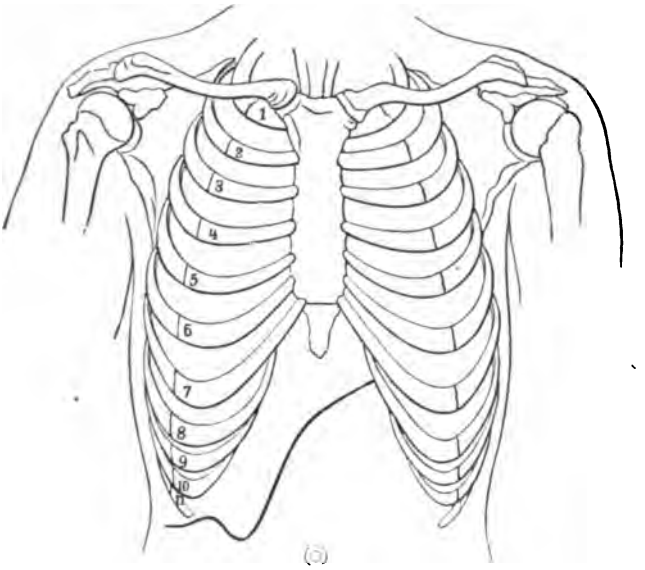

FIG. 110.—Riedel's projection of the liver in cholelithiasis.

kidney can be felt. With this condition the abdominal walls are frequently much relaxed, so that it is sometimes possible to feel a dislocated kidney better in the standing or sitting, than in the recumbent, posture. If the walls are very relaxed,

FIG. 111.—Corset liver (Frerichs).

the lower end of even a normally placed kidney may be felt. A disregard of this fact has led to the diagnosis of entirely too many "wandering kidneys."

Diffuse enlargement of the spleen, acute splenic swelling, passive congestion of

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the spleen, the spleen of leukemia, of *pseudoleukemia,* and of *intermittent fever* are all very easily palpated and determined by their position, shape, and mobility with respiration. The very large splenic tumor of leukemia and malaria often shows one or more horizontal notches on the anterior edge (Fig. 112), and its anterior surface is smooth and reaches some distance below the navel. Acute splenic swelling in *typhoid fever* and *other infectious diseases* can sometimes be demonstrated only at the height of inspiration when its inferior edge is felt just below the costal margin. This is of far greater diagnostic import than the results of splenic percussion, which, as we have seen, are frequently untrustworthy. In adults a spleen of normal size can only very exceptionally be palpated beneath the rib margin even with deep inspiration, and then the abdominal walls are generally very lax or the organs are dislocated (floating spleen, enteroptosis). In young children, however, this is frequently possible, and therefore with them one must be rather cautious in diagnosing splenic enlargement. To recognize a moderate grade of splenic enlargement by palpation we should examine both in

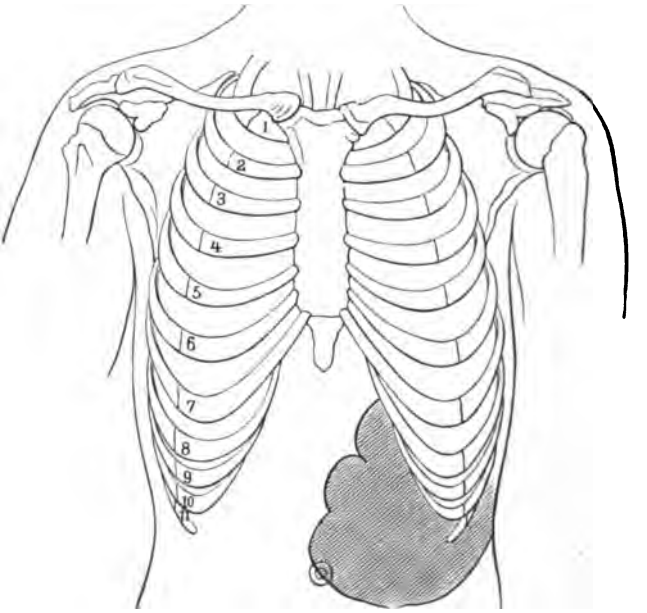

FIG. 112.—Splenic tumor of leukemia.

the dorsal decubitus and in the right semilateral position (diagonal position). Sometimes one and sometimes the other position furnishes better results. In palpating the spleen, stand, if possible, upon the right side of the sick-bed, place the palpating right hand as flat as possible upon the left hypochondrium, with the fingers at the costal margin, and with each expiration gradually exert slightly more pressure and attempt to feel the edge of the spleen during deep inspiration. Especially in typhoid fever a splenic enlargement can be felt only at the end of deep inspiration. In very sick cases the best method is from time to time to encourage one or two deep inspirations, because frequently repeated respiratory efforts tire the patient and his breathing becomes very superficial. Trying to feel the spleen with the fingers hooked in under the rib margin from above ordinarily produces such vigorous reflex abdominal contraction that nothing else can be felt. Correct splenic palpation requires much practice. It is a good preparation for abdominal palpation.

Large left-sided pleural effusions sometimes, though exceptionally, dislocate

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the spleen sufficiently for it to be palpable. Ferber explains this peculiarity (his opinion is supported by postmortem researches) in this way: Such effusions push the spleen so that the posterosuperior end reaches forward, the inferior end, backward; hence the long axis of the spleen assumes at first a vertical position, and finally a direction from in front and above backward and downward. In other cases the upper edge of the spleen is turned so toward the interior by the diaphragm, bowing convexly downward, that the splenic surface lies no longer vertically, but horizontally. Both results are thoroughly unfavorable for palpation, and, besides, the respiratory mobility is lost, because the diaphragm on that side is almost stationary. What we often mistake for the spleen in these cases is the bulging of the depressed diaphragm running parallel to the costal border (see Fig. 97, *I.*, 206).

(Compare the section on Examination of the Stomach in regard to the palpability of the spleen in certain cases of gastric dilatation.)

A *distended bladder* is easy enough to recognize after it has once been felt. It may be confused with the pregnant uterus, with other enlargements of the uterus, with ovarian tumors, and with inflammatory exudations. To differentiate we must utilize other methods of examination—*e. g.*, by vagina, and especially examination after catheterization.

A *peritoneal friction rub* may be appreciable to palpation over the different organs or over tumors of the abdomen. The friction can be felt as a rough grating with the respiratory and palpatory moving of the parts. It may also be appreciated by auscultation (see p. 286). These friction murmurs can be produced by any uneven surface (tumor surface), but in most of the cases they are caused by inflammatory deposition of fibrin. Splenic enlargement (in leukemia) frequently leads to perisplenic, and cholelithiasis to perihepatic, friction murmurs.

Peristaltic intestinal noises can also be appreciated by palpation. (See Section on Auscultation of the Abdomen, p. 286.)

"Dipping" over the abdomen will elicit a *splashing* wherever both fluid and gas are present in the digestive tract or in the peritoneal cavity. We can feel splashing over the stomach and over the region of the small intestine normally. Over the large intestine, splashing signifies that a diarrhea is either already present or impending. Splashing over the sigmoid region frequently depends upon the overlying small intestine, and cannot be trusted for diagnosis. Over the cecal region, splashing may generally be considered pathologic, and might suggest the possibility of typhoid fever. Concerning the splashing and swashing in perityphlitis, see page 309. Concerning the splashing of the stomach, see Stomach Examination (p. 354). Shaking noises of the abdomen (p. 286), which appear when patients move, can ordinarily be felt as well as heard.

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DIAGNOSIS OF INDIVIDUAL VALVULAR LESIONS, OF AORTIC ANEURISMS, AND OF PERICARDITIS.

To appreciate this section the reader should first study the sections upon Cardiac Percussion and Auscultation, and upon Palpation and Inspection of the Cardiac Region.

FOUNDATIONS OF THE PATHOLOGIC PHYSIOLOGY OF VALVULAR LESIONS.

EFFECT OF VALVULAR LESIONS UPON THE CIRCULATION; MECHANICS OF COMPENSATION; LAWS GOVERNING THE ALTERATIONS OF SIZE OF THE INDIVIDUAL HEART CHAMBERS IN VALVULAR LESIONS.

To understand the symptom-complex of any valvular lesion we must appreciate: the fundamental factors of physical diagnosis (outlined in the preceding sections), the effect of each individual valvular defect upon the circulation, and the anatomic changes and the modifications of the functions of the individual heart chambers which are produced by the wonderful efforts of compensation. In the following introduction we give, to avoid repetition, the fundamental factors of the pathologic physiology of valvular lesions:

Any valvular lesion, whether a stenosis or an insufficiency, from the moment of its origin leads to certain alterations in the distribution of pressure upon each side of the affected valve. If the body and the heart itself did not possess a series of powerful compensatory aids to improve this relation of altered pressure, then every serious lesion at its very inception would not only cause serious general disturbances of circulation, but very soon prove fatal. Experience shows that neither is the case. Without compensation the blood in every valvular lesion would be collected behind the diseased valve, and peripherally the blood-mass and arterial pressure would gradually diminish more and more. The heart's reserve power prevents to a certain extent such a dangerous condition; the sections of the heart lying behind the injured valve work harder, diminish the blood-stasis, furnish enough blood to the peripheral arteries, and so prevent a dangerous fall of the arterial pressure. The reserve power is utilized in stenosis to overcome the obstacle; whereas in insufficiency it must force more blood forward during the succeeding phase through the injured valve. Rosenbach and Cohnheim produced artificial valvular lesions in animals, and proved that immediately after the onset of a lesion an increase of cardiac work prevented any serious consequences to the circulation. To effect this increased work permanently, anatomic changes in the heart are bound to ensue within a short time. These consist in *hypertrophies* and *dilatations* of the different chambers.

Under the head of *Compensation* we include the entire complex,

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increased filling and increased work of certain heart chambers with their resulting dilatation and hypertrophy—*i. e.*, the sum of all those functional and anatomic changes which, in spite of serious lesions of the valvular apparatus, correct the fault up to a certain point, render life possible, and also warrant a condition of circulation subjectively endurable to the patient. Compensation, however, does not make the circulation normal, because the pressure behind the valve must be kept abnormally high, so that despite the resistance the essentials for the circulation may be preserved. What compensation accomplishes is only to keep the pressure in the systemic arteries, capillaries, and veins within the physiologic limits, so that the current rapidity and volume of the circulation is approximately normal, and so that in consequence the most essential bodily functions can be performed without the patient feeling very sick. To fulfil all these requirements, the compensatory changes must naturally prevent the increase of pressure from working backward behind the next valve. Therefore, in mitral lesions the most perfect compensation will not change the increase of blood-pressure and volume within the pulmonary circulation. Such patients suffer from dyspnea in case of any unusual demands upon the respiration (see p. 86).

This conception of compensation is only a relative one. In this connection another point should be mentioned. In stenoses of the auriculoventricular valves and in all insufficiencies[1] compensation does not prevent a stasis in the chambers situated behind the obstacle. The general circulation in these valvular lesions therefore suffers from a poverty of blood, because the mass of blood which permanently distends these congested chambers is of no avail to the circulation. We do not yet know whether in these cases the circulation adapts itself to such a condition by narrowing the remaining vascular system, or whether, in order to make up for the deficit, the blood-mass gradually increases. Unless the latter is the case, the deficit in the circulation would act something like a venesection and be essentially equalized by a narrowing of that part of the vascular system which is not dammed.

Dilatations and hypertrophies of separate heart chambers follow very simple physiologic laws, and from them the cardiac condition in any valvular lesion can be determined.

These laws are as follows:

1. Any heart chamber which suffers an increase of pressure only during systole hypertrophies—*i. e.*, corresponding to the greater amount of work, its muscle increases in thickness, without an increase in the size of the cavity (primary hypertrophy).[2]

[1] The stenoses of the arterial orifices behave differently, because they are the only valvular lesions where, during the stage of compensation, there exists no increase of diastolic pressure above the obstacle; in other words, the pressure during systole is increased, but their is no damming behind.

[2] The pathologists designate this kind of hypertrophy of the cardiac wall in which the included cavity is not enlarged as *simple hypertrophy*, in contrast to the so-called *eccentric hypertrophy*, in which wall thickness and cavity both increase (see the following note). It does not seem easy to explain why an enlargement of the cardiac cavity does not always result as a consequence of the growth of the muscular elements, but since an enlargement of a heart cavity would increase the amount of work, it is clear how very injurious to the progress of compensation such an enlargement would be. As a matter of fact it can be proved mathematically, as well as demonstrated experimentally, that the cavity must increase in size coincident with the increase in size of its walls. For

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2. Every heart chamber which suffers an increase of pressure during diastole—*i. e.*, which is filled over normal—becomes enlarged (*primary dilatation, dilatation from increased diastole, compensatory dilatation*). In order that the circulation shall be well preserved, it is necessary for the resulting dilated chamber to contract completely or very nearly so. Therefore it must accomplish more work, because, as is well known, the work done during systole equals the product of the systolic volume by the pressure to be overcome. As a result of this increase of cardiac work incumbent upon such a heart chamber, its walls must eventually *hypertrophy* (*secondary hypertrophy*). This hypertrophy is apparent either in an increase of thickness of the walls or (perhaps more probably) in a preservation of their normal thickness despite the dilatation.[1]

3. Where the conditions for *primary hypertrophy* and *primary dilatation* occur coincidentally, hypertrophy and dilatation of the heart chamber in question may take place entirely independently of each other.

4. In addition to the *primary dilatation* mentioned above (Law 2), a so-called *secondary dilatation* may arise. The latter, for the same reasons as under Law 2, occurs if a heart chamber without any primary increase in its blood-supply is not able to contract fully, and so during diastole suffers an increased pressure, because the blood entering finds a certain amount of blood left there from the preceding diastole. This type of dilatation depending upon an incomplete systole is called a *secondary* or *paralytic dilatation*. It has no compensatory significance, as contrasted with the type of dilatation described under Law 2, which is (as we shall see from examples) of decided value to the circulation, and therefore fittingly called a *compensatory dilatation*.

Dilatations due to incomplete systole are most frequently met with in the so-called compensatory disturbances of heart lesions (see below). Here, despite the variation in their causation, they all possess this common attribute, they all depend upon cardiac weakness, with the result that systole becomes weaker and the systemic arteries are less completely filled than during compensation. By the appearance of secondary or paralytic dilatations these disturbances of compensation can alter the primary picture of compensated valvular lesions in manifold ways. Such secondary alterations may disappear when the heart has regained its complete power, or they may remain permanent

example, suppose we place one of the writer's glutoid capsules (*Deutsch. med. Woch.*, 1897, No. 1), filled with some oily substance, in a 2 per cent. solution of muriatic acid and let it stand at an incubator temperature. The capsule-wall will gradually swell, and, since there can be no increase in the internal contents, the surface will become indented, the wall sinking in to fill up the space gained. Now, how can we explain the different behavior in the growth of the heart-wall so that there results a pure simple hypertrophy without any dilatation? Perhaps it is because a growth of muscular fibers in the layers of the myocardium surrounding the cavity is made mechanically impossible by the maximum systolic compression. This compression persists as long as a ventricle for which the requirements for a pure hypertrophy are present—*i. e.*, increased resistance to the systole—is able by means of the reserve power at its disposal to contract itself completely.

[1] What the writer has called *secondary hypertrophy* is the same condition pathologists designate as *eccentric hypertrophy*.

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despite the cure of the compensatory disturbance—*e. g.*, the secondary dilatation of the hypertrophied right ventricle in mitral insufficiency. The persistence of such secondary alterations may perhaps be explained by the fact that, if a heart chamber has once been enlarged by a paralytic dilatation, its recovery is rendered difficult, because the opposition to systolic contraction increases in proportion to the increase in its contents (since the work of a heart chamber in contracting is equal to the product of its systolic volume by the opposed pressure). If this opposition to systole caused by the dilatation reaches such a grade that the secondary hypertrophy of the cardiac muscle is no longer able to equalize the dilatation, but is limited to propelling the normal volume of blood from the diastolic position, then the heart chamber in question will finally to a certain extent become decidedly dilated. In this way a paralytic dilatation will persist anatomically fixed notwithstanding the fact that the compensatory disturbance has receded.

Even without the previous appearance of a distinct disturbance in compensation, similar dilatations sometimes affect those chambers of the heart which, according to Law 1, should be purely hypertrophied, because they have been subjected not to greater filling, but merely to increased systolic pressure. The most frequent instance is again the dilatation of the right ventricle in mitral insufficiency, which may occur even without any disturbance in compensation. Such dilatations are in a measure related to the paralytic dilatations after a compensatory disturbance which are described above as anatomically fixed. The fact that such a disturbance in compensation does not precede can probably be explained by assuming that the dilatation in these cases begins quite gradually, not from an acute loss of cardiac power, but in consequence of a slow increase in the obstacle, which has gone too far to permit the persistence of a complete systole, and by further assuming that this dilatation is rendered comparatively harmless by the hypertrophy which develops step by step and becomes at the same time fixed.

Another conception of such primary dilatation of certain chambers of the heart which are simply under higher pressure during the systole and have not been subjected to greater filling (as in aortic and pulmonic stenosis) is that it is a compensatory phenomenon. The ventricle subjected to an excessive systolic pressure reaches a greater degree of efficiency when its musculature is distended, just as a skeletal muscle gains in power with an increase in the distance between its two extremities. Nevertheless, the writer would not venture to claim that in this respect the heart acts in an analogous manner to the skeletal muscles, nor that this explanation is a better one than the preceding.

After we have reviewed these latter observations we find that we must modify Law 1, for an increased resistance to the systole of a heart chamber can produce a pure hypertrophy of its wall only so long as this resistance does not overstep a certain degree, but as soon as this point is passed incomplete systole, dilatation, and finally secondary hypertrophy of the dilated heart chamber occur. Perhaps this will explain the peculiarity that in some cases of chronic nephritis we find a pure hypertrophy, and in other cases a hypertrophy with dilatation of the heart.

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It has often been questioned whether such heart chambers, enlarged by paralytic dilatation and later anatomically fixed in their dilatation, ever contract again completely in spite of their increased contents, or whether they permanently produce incomplete systoles. An objection to the former of these two possibilities is that if no stasis occurs, provided a circulation is permanently possible, the other heart chambers will be more extensively dilated during diastole in order to receive the excess of the propelled blood, and so they will be obliged to accomplish correspondingly more work during systole, and for this purpose they must naturally be permanently dilated and at the same time hypertrophied. This would finally result in an entirely purposeless enlargement of and in excessive work for the entire heart, and besides in a purposeless increase in the circulation (excessive compensation). It is not possible to definitely and absolutely decide whether such conditions of the circulation can really occur. They are quite opposed to the teleologic conception of the process of compensation. If so, these permanent excessive burdens of the heart which increase with each disturbance in compensation will furnish an explanation for the fact that practically every heart lesion leads, sooner or later, to a definite cardiac insufficiency, although the causal valvular lesion does not grow.

To decide whether such a heart chamber with fixed paralytic dilatation always contracts incompletely depends very closely upon another question: Does the systole of the heart, under physiologic conditions, always contract completely, or is it sometimes also incomplete? This latter question must, it seems to the writer, according to our present knowledge, be answered in the following way: The ventricle contracts completely or almost completely if the obstacles opposed to its systole are normal. It should act in the same way so long as the obstacles to its emptying are only slightly increased. Aside from the teleologic significance which the latter condition possesses for the preservation of the circulation even under somewhat more difficult conditions, the writer sees a clinical demonstration in proof of it in the fundamental Law No. 2—*i. e.*, with a mere systolic increase of work a ventricle will ordinarily merely hypertrophy without any dilatation. As soon as the slightest increase in opposition prevents the ventricle from contracting completely, it must always dilate—*e. g.*, a dilatation of the right ventricle must therefore always occur in mitral insufficiency. With more pronounced opposition to its emptying, the heart seems to react with a diminished systole (concurred in by Marey,[1] Dreser,[2] Tigerstedt and Johansson,[3] and O. Frank[4]). The question resolves itself, then, into whether we are to consider this latter phenomenon as a physiologic reaction or as a consequence of a pathologic condition—a paralysis of the heart. Personally, in agreement with O. Frank and Moritz,[5] the writer considers it a physiologic reaction. His reasons are: the skeletal muscles behave quite analogously; the phenomenon is frequently of great teleologic significance for assisting the heart and the arteries with high blood-pressure; and it has been experimentally demonstrated that such a heart, as soon as the opposition has been removed, recovers itself immediately and contracts again completely. If we believe that incomplete systoles occur physiologically, it seems perfectly conceivable and entirely within physiologic facts that a cardiac chamber enlarged by paralytic dilatation permanently contracts incompletely.

A certain number of autopsies upon patients with valvular lesions seem to argue against the above-mentioned laws. We must remember, however, that the relations of size of the chambers in the heart of the cadaver differ essentially from those in the living heart, because the phase in which each cardiac chamber is paralyzed (systolic, diastolic immobility) is of especial significance. The degree of rigor mortis of the heart is very rarely heeded at autopsy, although it would be a valu-

[1] *La circulation du Sang.*, 1881.
[2] *Arch. f. exp. Path. u. Pharm.*, vol. xxiv.
[3] *Skandinav. Arch. f. Physiol.*, vol. i., 1889.
[4] *Zeit. f. Biol.*, vol. xxxii.
[5] *D. Arch. f. klin. Med.*, vol. lxvi.

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able study to determine the time of onset of the heart-muscle rigor and its influence upon the size of the heart chamber. Moreover, we have no right to compare the relations of the heart of valvular disease at autopsy with the relations of the compensated valvular lesion, because disturbances of compensation usually affect patients with valvular disease some little time before their death, and so the relations of the size of the heart may be very different. We will mention this again in the special description of individual valvular lesions.

In reality, therefore, in order to determine the relations of size of the individual heart chambers during good compensation we should utilize only autopsies upon patients with valvular lesions who have died suddenly from some intercurrent trouble, and not those upon patients whose death is directly dependent upon their heart lesion. Even in the former the autopsy finding is not actually and necessarily the same as during life.

It is rather doubtful whether the Röntgen rays will furnish us more trustworthy results about these relationships.

Compensatory anatomic alterations of the heart are usually permanent, because the valvular lesion itself rarely improves, and because life depends upon the maintenance of compensation. Compensatory changes are more apt to increase in the course of years, because most valvular lesions are progressive. Where the valvular lesion itself is capable of retrogression, the compensatory changes, too, may retrograde to a slight extent or even completely disappear. These are extremely rare cases. Dilatations and hypertrophies proceed in accordance with the necessity which occurs, and this necessity can be perfectly explained by studying the above-mentioned laws.

COMPENSATORY DISTURBANCES.

A valvular lesion, thanks to the so-called compensatory changes, the hypertrophy and dilatation of individual heart chambers, may for years produce no marked symptoms, but sooner or later the general circulation will be affected more or less decidedly, because compensatory contrivances fail. We then speak of "disturbances in compensation." The causes of these disturbances may be, on the one hand, an increase in the opposition to the circulation produced by the valvular lesion, or the appearance of new circulatory obstacles (arteriosclerosis, nephritis); or, on the other hand, an injury to the heart muscle, which recent investigations have taught us not infrequently depends upon inflammatory alterations of the heart muscle, and which in valvular lesions is frequently produced quite gradually. The same process goes on quite similarly in other circulatory obstructions which do not depend upon an injured valve. For a long time they also may remain compensated by the hypertrophy of certain heart chambers, until finally disturbances in compensation ensue. All cases of compensatory disturbance depend upon a disturbance in the favorable relation between the circulatory obstacles and the cardiac power, either an absolute diminution of cardiac power or a relative diminution of the latter in proportion to an increasing obstacle. This condition is

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exhibited by a diminution of the systoles of all or of individual heart chambers. The heart begins to work with an increasing amount of residual blood in all or in a certain one of its chambers. We will mention under Individual Valvular Lesions how the relation of the size of the heart is affected by secondary or paralytic dilatations. General stasis ensues in consequence of diminished systoles. If the weakened condition affects the right heart more than the left, the stasis will be localized more in the systemic than in the pulmonary circulation, and *vice versâ*. Thus, the clinical picture will vary. Compensatory disturbances have, however, considerable uniformity despite the most varied obstacles to the circulation, and they are equally significant whether the diminution of heart power proceeds from the left or from the right heart. Only in rare individual cases do these disturbances vary decidedly. This uniformity probably depends upon the fact that the coronary arteries are injured as much in paralysis of the right as in paralysis of the left heart, so that the entire heart finally shares in the paralysis. This usually uniform clinical picture of compensatory disturbance may be described as follows: The filling of the systemic capillaries diminishes. The distention of the veins and the venous pressure increase. The circulation is slowed by diminution in the blood-supply. Cyanosis and dyspnea ensue. Edema and other dropsical accumulations are added. The urinary excretion is diminished and the urine frequently contains albumin. The causes of these appearances are discussed in the sections on edema, dyspnea, cyanosis, amount of urea, and albuminuria. The pulse frequently varies; it is very often accelerated, as if the heart attempted to overcome to some extent its fault of incomplete contraction by great frequency of contraction. The heart action is frequently very irregular and produces the impression of overstimulation. Very pronounced irregularity has been designated quite properly delirium cordis. Neither great rapidity nor irregularity of the pulse necessarily accompanies compensatory disturbances. Like the other symptoms, one or the other of them may be lacking or very much less pronounced. The single constant factor in compensatory disturbances is the diminished cardiac power and the insufficient systole. Quite an important diagnostic sign of disturbed compensation in a valvular lesion with an audible murmur is that this murmur becomes fainter and perhaps inaudible on account of the diminution in the current rapidity within the heart. On the other hand, a dilatation of the ventricle caused by the disturbed compensation may produce a murmur where one was not audible before, or bring one out very much more distinctly, due to a relative insufficiency of the auriculoventricular valve (p. 264 *et seq.*), which is superimposed upon the original lesion. This subject has already been discussed under Cardiac Murmurs. From evident reasons, the heart tones are frequently weaker in disturbed compensation. In patients with disturbed compensation we sometimes find a relatively tense pulse—*i. e.*, high arterial pressure—which would seem to argue against our explanation that compensatory disturbances depend essentially upon insufficient cardiac power. As a matter of fact, the

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difficulty vanishes when we remember that the left ventricular work does not depend merely upon the arterial pressure, but is measured by the product of the arterial pressure by the volume of blood emptied during systole, and that with marked arterial resistance even a very weak systole may produce high arterial pressure. The writer calls such conditions "high-pressure stases," in contrast with the more frequent "low-pressure stases," where arterial pressure is diminished by the disturbed compensation. "High-pressure stases" occur in circulatory disturbances associated with arteriosclerosis or chronic nephritis.

INDIVIDUAL VALVULAR LESIONS.

The alterations in size of the different heart chambers (dilatations and hypertrophies) which occur in individual valvular lesions will be explained perfectly by a thorough comprehension of the laws which have been discussed in the preceding pages concerning the origin of compensation and compensatory disturbances. For the sake of brevity, only the number of the law which explains the hypertrophy or dilatation will be mentioned in the text (see p. 315 *et seq.*). The reader will find that his comprehension of these laws will be facilitated by studying the hydraulic diagram given at the beginning of each description. He will find represented there the alterations in pressure which the valvular lesion produces upon each heart chamber. The explanation of the signs employed in these diagrams is given with Fig. 113. The reader should consult pages 244 to 274 in order to understand the diagrams representing the results of physical examination in each valvular lesion, and he should recall (p. 164 *et seq.*) that the blue represents the superficial, the red the deep cardiac, dulness. A cross ($\times$) is used to designate the point upon the skeleton for the percussed boundaries. The shaded wedges represent the murmurs, and their thickness the intensity of the murmur. Near the blunt end of the wedge the sign $>$ represents a decrescendo murmur; the sign $<$ represents a crescendo murmur. The tones at the apex of the heart and over the tricuspid valve are represented by the signs $\perp \smile$; while over the base of the heart (over the great vessels) the tones are represented by the signs $\smile \perp$. The phase of the murmur is determined by its position, as compared with the tones. Accentuation of the tone is represented by an accentuation of the sign.

VALVULAR LESIONS OF THE LEFT HEART.

The valvular lesions of the left heart are much the most frequent. (Congenital heart disease is not included.) In any given case therefore the diagnosis will be facilitated by the rule that wherever a valvular lesion appears after birth, as a result of joint rheumatism or arteriosclerosis, we should think first of a left-sided lesion. On the contrary, in congenital cardiac disease we think first of right-sided valvular lesions.

MITRAL INSUFFICIENCY.

The essential fault in *mitral insufficiency* (Figs. 113 and 114) consists in a systolic regurgitation of blood into the left auricle. This regur-

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gitation depends upon the imperfect closure of the mitral valve and gives rise to a systolic murmur. It is the most frequent of all valvular lesions. The regurgitation increases the pressure in the auricle enough to dilate its walls. The increased pressure in the auricle is transmitted back through the entire pulmonary circulation. The pulmonary semilunar valves, however, are shut during diastole, so that the right ventricle does not suffer from the raised pressure at this stage. But the increased pulmonary pressure acting to augment the work of the right ventricle during systole necessitates its hypertrophy (primary hypertrophy) (Law 1). Dilatation of the left auricle is responsible for its own secondary hypertrophy (Law 2). But there is another especially important element. The left ventricle receives more blood during its diastole than normally because the left auricle is overfilled; hence it

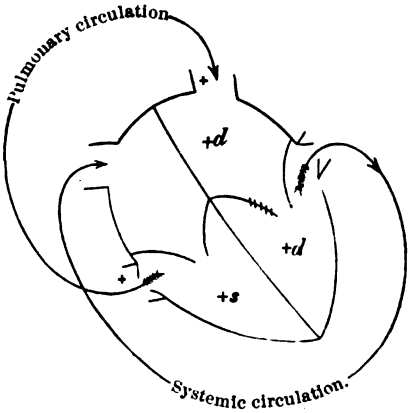

FIG. 113.—Hydraulic diagram of mitral insufficiency: +, Increased pressure; + *s*, increased systolic pressure; + *d*, increased diastolic pressure; + *ds*, increased systolic and diastolic pressure in the heart chambers within which the signs appear. Hence, a heart chamber marked with + *s* hypertrophies primarily; one with + *d* dilates primarily; and one with + *ds* both dilates and hypertrophies primarily.

becomes primarily dilated (Law 2) because the pressure within it is increased during diastole. This dilatation leads in turn (Law 2) to a *secondary hypertrophy* of the left ventricle. The reason for the left ventricular dilatation is plainly compensatory; because despite the fact that a part of its blood is regurgitated into the auricle, its increased capacity enables the arterial system to be completely filled.[1]

In a well-compensated mitral insufficiency the systemic circulation is practically normal. The pulse is not at all small, as has often been stated, and the only striking disturbance which persists during compensation even of a severe case of mitral insufficiency is a more or less pronounced *dyspnea*, which ensues because the lungs are overfilled with blood.

[1] Theoretically, a very slight deficit in the arterial filling must necessarily ensue, because an extra amount of blood is required for the increased filling of the pulmonary circulation, of the left auricle, and of the left ventricle.

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With this the series of compensatory changes in mitral insufficiency is complete. Clinically (Fig. 114) the lesion is recognized by a systolic murmur, generally heard most clearly and loudly at the apex of the heart, but under certain circumstances (see p. 269 *et seq.*) with its maximum at the base. This murmur may even overpower or obliterate the systolic mitral tone. Sometimes we can feel it as a thrill as well as hear it. The primary dilatation of the left ventricle and left auricle can be demonstrated by percussion (frequently by palpation). Simple or pure hypertrophy of the right ventricle cannot ordinarily be appreciated by percussion (p. 182); but the increased pressure in the pulmonary circulation which produces this hypertrophy of the right ventricle is generally evident from an accentuation of the second pul-

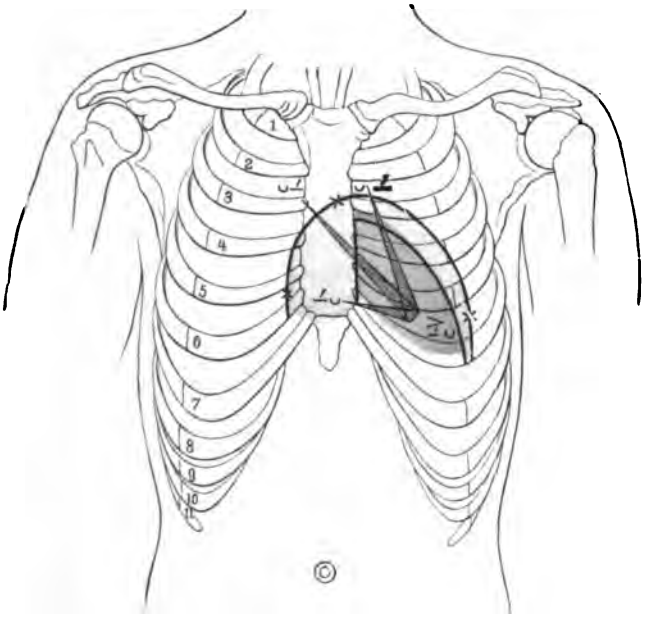

FIG. 114.—Diagnostic diagram of mitral insufficiency. (The signs are described upon p. 321.)

monic tone (p. 251), sometimes from the increased pulsation, or shock, of the pulmonary valve closure, visible or palpable in the region of the pulmonary artery (p. 300). The diagnostic significance of the pulmonary second accentuation is, however, frequently overestimated. Not infrequently it fails to appear, and again there are many cases where the compensatory power of the right ventricle is not sufficient to produce it—*e. g.*, cases of a mild degree of insufficiency, where the increased pressure is slight; and finally cases where, before the appearance of the mitral insufficiency, the pulmonic second was physiologically weaker than the aortic second. In contradistinction to the diastolic murmur of aortic insufficiency (see p. 330), it is worth remembering that the murmur of mitral insufficiency is heard with less intensity

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when the patient is standing than when lying down. This is very likely due to the action of gravity. In fact, a faint mitral insufficiency murmur can frequently be heard only in the recumbent posture.

The above-mentioned signs are ordinarily noted in a well-compensated case of mitral insufficiency. Sooner or later, however, disturbances of compensation are bound to appear. They ordinarily begin in this way: The right ventricle can no longer completely contract, and therefore dilates (paralytic dilatation, Law 4); as a consequence the accentuation of the second pulmonic disappears, the left auricle and the left ventricle in turn receive less blood, and the filling of the systemic arteries and capillaries diminishes. This last danger signal is still more marked when from the same cause the left ventricle begins to weaken. All the other appearances of disturbed compensation described above can be added to the lack of arterial filling until, under the influence of rest or cardiac tonics, compensation is again established. The heart power then increases. The right ventricle again empties itself completely and returns to its condition of pure or simple hypertrophy. But ordinarily during the course of re-establishing compensation the right ventricular dilatation becomes anatomically fixed by the development of a secondary hypertrophy—*i. e.*, by the addition of layers of new muscular tissue (p. 317). This is permanent and can be demonstrated by percussion. (See p. 330 in reference to the completeness of the contraction of this permanently dilated right ventricle.) With the re-establishmeut of compensation the severe circulatory disturbances completely disappear.

The right ventricle may become dilated and hypertrophied in a similar way even without compensation being disturbed, depending upon the conditions described upon p. 317.

The dilatation of the right ventricle in mitral insufficiency is in reality (see p. 316) a secondary one—*i. e.*, a result of incomplete systole—and should be sharply distinguished from the left ventricular dilatation. The latter has a compensatory significance.

(See p. 132 in regard to the characteristics of the pulse in mitral insufficiency; p. 288, in regard to the presence of a pulmonary pulse; p. 176, in regard to the depth of the lung borders from pulmonary stasis; p. 86, in regard to the character of the respiration; p. 253, in regard to the disappearance of the mitral tone, or both tones to the left heart; p. 270, in regard to the difference between a mitral and an aortic systolic murmur; p. 270, in regard to the occurrence of a prediastolic murmur, and p. 256, in regard to a splitting or doubling of the second tone.)

MITRAL STENOSIS.

In mitral insufficiency the obstacle to the circulation is effective during systole; but in mitral stenosis, a comparatively frequent valvular lesion, the obstacle acts during diastole of the left ventricle. To understand what follows we should remember that the ventricular diastole can be subdivided into two intervals in relation to the auricle. During

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the first part of the ventricular diastole the auricle is passive; the blood, under the influence of a moderate pressure from its course through the pulmonary circulation, flows into the left ventricle without the aid of the auricle. In mitral stenosis, however, an obstacle prevents this emptying of the auricle; the left auricular pressure increases, and, the blood collecting, distends the auricular walls, which are ordinarily relaxed at this period. The first result of mitral stenosis upon the heart (Law 2) is therefore the dilatation of the left auricle. During the second part of ventricular diastole the auricular systole comes into action, and on account of the stasis behind the stenosed mitral valve it has to propel an increased amount of blood, and in addition overcome the obstacle at the mitral valve. As a result (Laws 1 and 2) a hypertrophy of the dilated left auricle supervenes. We might imagine that (without help from the right ventricle) the increased power of the left auricle could effect the compensation. But experience has shown us

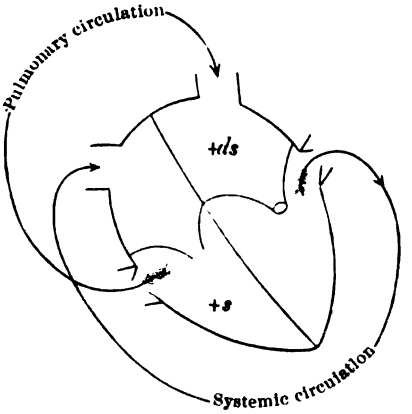

FIG. 115.—Hydraulic diagram of mitral stenosis. (See Fig. 113 for the explanation of the signs.)

that this is not the case, because hypertrophy of the right ventricle belongs as much to mitral stenosis as to mitral insufficiency, so that, despite the increased work of the left auricle, there remains an increased task for the right ventricle. The explanation is probably that the left auricle does not grow to be strong enough both to expel completely its increased content and to overcome the mechanical obstacle of the narrowed mitral valve.[1] This is not to be wondered at when we consider the slight muscular power of the auricle; as a matter of fact, autopsies show no very striking hypertrophies of the auricle. Assuming, then, that in mitral stenosis the left auricle cannot completely empty itself, and so is continually laboring with residual blood, it happens, naturally, that during auricular diastole the blood from the lungs does not flow into an empty, but into an already partially filled, left auricle.

[1] Perhaps the same laws in regard to the completeness and incompleteness of contraction apply here as to the ventricle (see p. 318).

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This is of equal significance to an increased resistance to the pulmonary circulation present during auricular diastole or ventricular systole. There are no valves at the outlet of the pulmonary veins; hence it is easy enough to understand that the contraction of the left auricle must drive back a part of the blood stagnating in the auricular chamber into the lungs and part through the narrowed mitral valve.[1] The increased resistance in the pulmonary circulation persists until the ventricular systole occurs, and can be overcome only by the hypertrophy (primary) of the right ventricle (Law 1). Thus, the hypertrophy of the right ventricle in mitral stenosis enables compensation to be effected by producing a continuous diastolic flow through the stenosed mitral valve. It presupposes an elastic tension of the pulmonary vessels, so that the

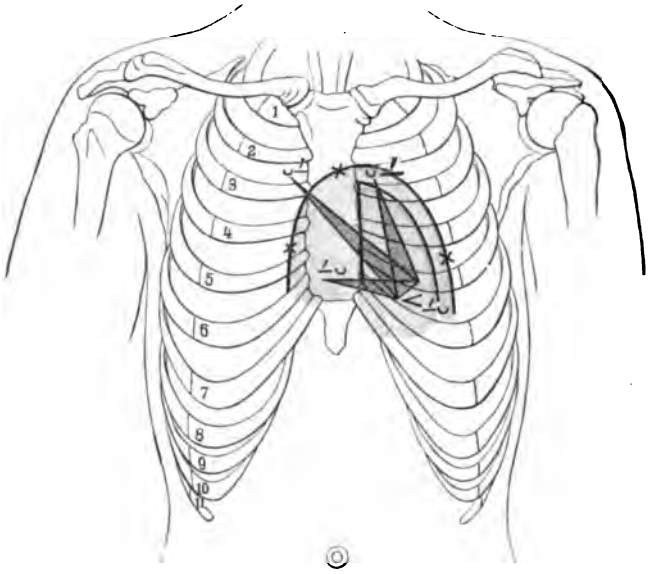

FIG. 116.—Diagnostic diagram of mitral stenosis. (See p. 321 for the explanation of the signs.)

systolic power of the right ventricle is transmitted through the lung into the left auricle. Despite its complexity, this supposition alone seems to explain that the systole of the right ventricle aids in producing compensation, although the obstacle is diastolic.[2] The conditions are much simpler in mitral insufficiency; for here, as a direct result of the regurgitation, both ventricles are working directly at variance, making it perfectly clear why the right ventricle must do more work.

[1] The fact that a considerable portion of the blood is driven backward instead of forward by the auricular contraction also explains the absence of dilatation of the left ventricle, in spite of the increase in the pulse volume of the left auricle.

[2] This passive elastic function, in which the propelling force is traced back to the right ventricle, is in reality a normal function of the left auricle and the pulmonary vessels, since during the first part of the ventricular diastole the blood flows into the left ventricle mainly on account of the excess of pressure which is left over from the lungs.

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There is no question of a primary dilatation of the right ventricle in mitral stenosis any more than in mitral insufficiency, because in either case, so long as the pulmonary semilunar valves close perfectly, there is no increase of right ventricular pressure except during systole (Law 1). Stenosis of the mitral valve evidently furnishes no reason for any change in the left ventricle. In a well-compensated mitral stenosis the left ventricle does not show concentric atrophy, as is sometimes contended, because our conception of good compensation is not compatible with the supposition, necessarily included in such a contention, that the diminished left ventricle forces decidedly less blood than normally into the systemic arteries.[1]

We not infrequently see at the autopsy table a case of mitral stenosis with a large right and a small left ventricle. The reason is either that the lesion could not be compensated, on account of the extreme degree of the obstruction (essential obstruction[2]), or that the patient died during a disturbed compensation, in which the incomplete systole of the right ventricle occasioned a paralytic dilatation of its own chamber, and consequently an imperfect filling of the left ventricular cavity.

This paralytic dilatation of the right ventricle may remain fixed even after the disturbed compensation has been adjusted, and an enlarged and hypertrophied right ventricle may result permanently, just as in mitral insufficiency. Even without actual disturbance of compensation the same result may, under some circumstances, be accomplished by the causes mentioned upon p. 317. Whether such a dilated and hypertrophied right ventricle is then completely or incompletely contracted is not certain (see p. 318). This must have some effect upon the other heart cavities, for the left auricle and the left ventricle would then contain much more blood than normally and the left ventricle would also be dilated. In this way we might explain cases of pure mitral stenosis which show a dilatation of the right, as well as of the left, ventricle. But such a supposition cannot be accepted without hesitation, because it supposes that the diseased mitral stenotic heart would finally perform a greater systolic effort than the healthy heart. And this is not only *à priori* improbable, but it is not in any way supported by the character of the pulse. So in mitral stenosis, where we meet with a dilatation of the left ventricle, we assume that either a simple paralytic dilatation from a final paralysis of the left heart, or, with a coincident hypertrophy of the left ventricle, a complication with mitral insufficiency exists. This may escape anatomic demonstration and, if there is no systolic murmur, even clinical confirmation.

Mitral stenosis will produce a dilatation of the left auricle (see pp. 184, 185), which can be demonstrated by percussion; but the hypertrophy

[1] The deficit which the systemic circulation suffers in consequence of the collection of blood in the left auricle and in the lungs is of little importance to the left ventricle during the stage of compensation, because the vessels of the greater circulation possess a capacity of adapting themselves to varying degrees of fulness. Perhaps the deficit is cared for by an increase of the blood-mass (see p. 315).

[2] The reader is referred to the author's paper: "Herzmittel und Vasomotorenmittel" (Cardiac and Vasomotor Remedies), *Cong. f. inn. Med.* at Berlin, 1901.

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of the right ventricle, unless associated with dilatation, ordinarily escapes such clinical demonstration. In later stages mitral stenosis may be associated with an appreciable increase of the heart dulness to the right as well as to the left. The enlargement to the left may in exceptional cases (see above) be due to a true dilatation of the left ventricle; but ordinarily it is the dilatation of the right ventricle which pushes the heart and the apex beat to the left (see pp. 182, 291 *et seq.*).

In a case of mitral stenosis we generally hear a diastolic murmur with a presystolic accentuation or merely a presystolic murmur at the apex; frequently we can also feel it as a presystolic thrill. (In regard to the mode of production of this murmur and its significance see p. 269 *et seq.*; in regard to the details of its localization see p. 266; in regard to the presystolic mitral murmur of aortic insufficiency see p. 331). The second pulmonic tone is generally accentuated in mitral stenosis in consequence of the increased pressure in the pulmonary circulation and the hypertrophy of the right ventricle. The value of this sign for the diagnosis of mitral stenosis has, however, often been overestimated (see p. 251). If compensation is disturbed the accentuation may disappear. The normal second pulmonic tone is often (if not generally) weaker than the normal second aortic tone, so that a pathologic accentuation of the former is frequently not apparent to the examiner. And, finally, with a mild degree of mitral stenosis the increase of pressure in the pulmonary circulation may be insufficient to produce a plain accentuation of the second pulmonic.

We frequently hear a splitting or doubling of the second tone (see p. 255 *et seq.*), and occasionally a presystolic or diastolic tone due to an imperfectly opened mitral valve (see p. 257 *et seq.*).

During quiet cardiac action sometimes the only auscultatory sign of mitral stenosis is a triple rhythm, the third tone of which is furnished by the presystolic tone. Besides, it is well to remember that mitral stenosis is more liable than any of the other valvular lesions to run its course without giving rise to a murmur (see p. 264).

(In regard to the character of the pulse of mitral stenosis see p. 132; in regard to the character of the respiration see pp. 109 and 315; in regard to the presence of a pulmonary pulse see p. 288; in regard to the deep position of the lung borders on account of pulmonary stasis see p. 177 *et seq.*).

AORTIC INSUFFICIENCY.

Aortic insufficiency—imperfect closure of the aortic valve (Figs. 117 and 118)—is, next to mitral insufficiency, the most common valvular lesion. Its mechanism is the one most readily comprehended by the beginner. The fault is that during diastole (Fig. 117) the blood rushes back into the left ventricle through the imperfectly closed semilunar valves, and so gives rise to the characteristic diastolic murmur (see below). Without compensation the mechanical effect upon the circulation would be that the aorta, deprived of part of its blood during diastole, could not fill the arteries and keep up the pressure. Compensa-

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tion prevents such a result in the following way: The regurgitating blood enters the diastolic relaxed left ventricle. This chamber is at the same

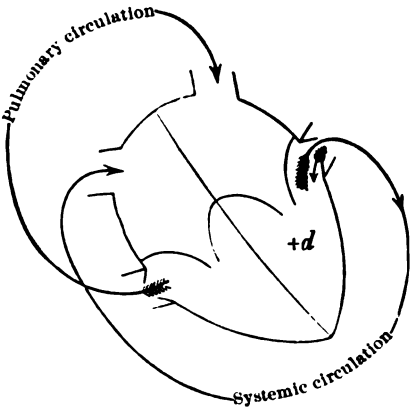

FIG. 117.—Hydraulic diagram of aortic insufficiency. (Compare Fig. 118 for explanation.)

time receiving blood through the mitral valve. Its walls have therefore to endure an increased pressure during diastole, and hence become dilated (p. 316 *et seq.*, Law 2). In virtue of its reserve power the left ventricle still contracts itself completely, and naturally sends an in-

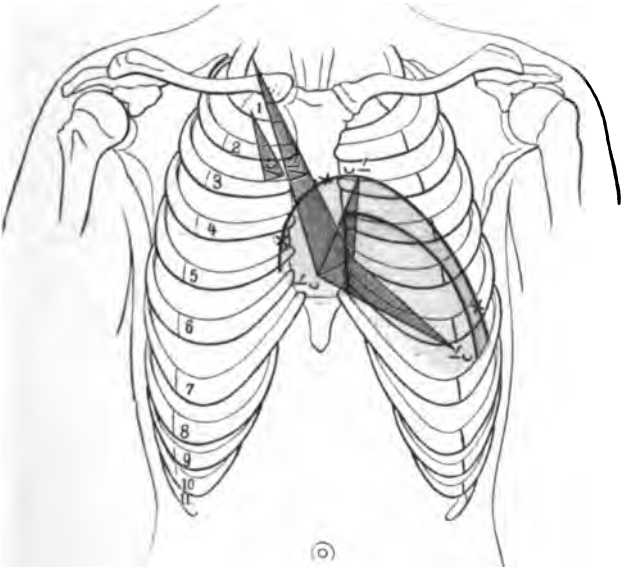

FIG. 118.—Diagnostic diagram of aortic insufficiency. (Compare p. 321 for an explanation of the signs.)

creased volume of blood into the aorta. But it cannot continually perform this extra work without hypertrophy (Law 2). As soon as hyper-

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trophy takes place the valvular lesion is compensated, and for the time being harmless. Then at every systole the aorta receives more blood than normally, and the loss through regurgitation is not significant. So one sees in this lesion, as in mitral insufficiency, that the dilatation of the left ventricle is primary, and that it is essential to the accomplishment of compensation. Therefore primary dilatation and secondary hypertrophy of the left ventricle are an essential result of aortic insufficiency (Fig. 118). Frequently a diffuse dilatation of the aorta, which depends upon its being stretched by the increased power of the systole, goes hand in hand with the dilatation of the left ventricle. This aortic dilatation may become evident in the appearance of an exceptional pulsation and an increased dulness in the upper intercostal spaces to the right of the sternum (Fig. 92). It is liable to be confused with a real sacculated aortic aneurism (see p. 345). Thus far the right ventricle takes no part in compensating an aortic insufficiency. But as soon as disturbance in compensation arises, as soon as the left ventricle is unable to perform its increased work perfectly, so that during systole it is incompletely emptied, then the residual blood within its cavity prevents a complete emptying of the left auricle, and, just as in mitral stenosis, this reacts upon the pulmonary circulation and the right ventricle. If such a paralysis of the left ventricle occasions a considerable dilatation of its walls, a relative mitral insufficiency will also appear and, in the well-known way, react upon the pulmonary circulation and the right ventricle. This reactionary effect upon the pulmonary circulation, which, according to our discussion above, does not depend entirely upon the occurrence of a relative mitral insufficiency, but which can sometimes be caused by the residual blood in the left ventricle, explains how failure of the left ventricular power in aortic insufficiency finally produces the ordinary picture of disturbance of compensation, with cyanosis, dyspnea, edema, etc., just as in the mitral lesions. The disturbance of compensation may be repaired; and then, depending upon the individual peculiarities of the case, the right ventricle either regains its normal size, or else, if a relative mitral insufficiency (see p. 317 *et seq.*) persists on account of the hypertrophied left ventricle, may remain permanently hypertrophied, or both hypertrophied and dilated. Such a permanent dilatation of the right ventricle, supposing it contracted itself completely, would produce secondarily a further enlargement of the left auricle and ventricle (excessive compensation?) (see p. 318).

The most important of the physical signs of this valvular lesion is (Fig. 118) a diastolic murmur audible at the aortic area and over the sternum (see p. 266, 2). The murmur is sometimes plainly transmitted into the carotids, and is usually more distinctly heard in the standing than in the recumbent posture, because gravity favors the regurgitation. A weak murmur of aortic insufficiency (contrasted with that of a mitral insufficiency, p. 323) is oftentimes audible only while the patient is standing.[1] The second aortic sound may be absent; again, it may be

[1 A very faint murmur of aortic insufficiency can sometimes be appreciated by the ear against the chest when it is not audible through the stethoscope.—ED.]

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unaffected if the aortic valves are not much diseased, or it may even be accentuated, in consequence of the increased systolic filling of the aorta; but more frequently, on account of the changes of the aortic valves, it is diminished or even disappears. In most cases of aortic insufficiency a systolic murmur can also be heard over the aorta. According to one hypothesis, this systolic murmur depends upon a roughness at the aortic valves, due to endocarditis or atheroma, which affects the blood-current during systole, although there is no actual stenosis. According to another hypothesis, the systolic murmur results from the diastolic regurgitating stream clashing with the systolic stream (p. 262), a real stenosis). According to a third hypothesis, it is the result of the increased rapidity of expulsion of the blood from the more powerful systole (p. 261). However this may be, it is well to be cautious in making a diagnosis of aortic stenosis from the presence of a systolic murmur heard over the aorta in a case of aortic insufficiency unless there are other signs of its presence. An especially rough or musical murmur speaks with some probability for the existence of an aortic stenosis; but the deciding point is the character of the pulse. If there is an actual aortic stenosis—that is, one caused mechanically—the pulse will possess more or less plainly the characteristics of the *pulsus tardus* (see p. 105 *et seq.*, p. 127). See p. 274 in regard to the presence of a doubled maximum diastolic and systolic murmur—*i. e.*, one heard very plainly both over the aorta and at the apex. A complicating mitral insufficiency or stenosis is often wrongly diagnosticated from such signs.

Austin Flint[1] described in aortic insufficiency a presystolic murmur audible over the auscultation area of the mitral valve. He explained it in this way: The regurgitating stream from the aorta spreads out the curtain of the mitral valve just as the presystolic stream from the left auricle is passing through this valve into the left ventricle. Hence the mitral curtain cannot be perfectly opened, and a sort of functional mitral stenosis results. This produces the presystolic murmur. This explanation does not seem to the writer sufficiently proved by the postmortem findings; nevertheless it deserves diagnostic attention. A certain differentiation of this condition from the combination of an aortic insufficiency with an organic mitral stenosis must be very difficult. Certainly such a functional origin of a pronounced presystolic murmur is rare, so that the diagnosis of a mitral stenosis complicated with aortic insufficiency is not so difficult.

Further important diagnostic signs of aortic insufficiency are: the *pulsus celer* (pp. 105 and 127) and a series of signs depending upon it, which have been explained above; a *capillary pulse* (p. 144 *et seq.*); the *single* and *double arterial tones* (p. 282); the *Duroziez double murmur* (p. 282 *et seq.*); the rare appearance of the *penetrating venous pulse* (p. 152 *et seq.*); and the rare *arterial liver pulse* (p. 300). All these signs are, of course, most plainly marked during good compensation; less plainly during disturbed compensation. They are not all equally plain.

During disturbance of compensation in aortic insufficiency the character of the pulse still seems exceptionally well preserved despite the poor circulation. This depends in the first instance upon the fact

[1] *Flint's Murmur*, first described in *Manual of Auscultation and Percussion*, Austin Flint, 3d ed., 1883, p. 231.

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that even with poor filling of the arteries the pulse does not lose its character of *pulsus celer*, which would naturally produce the impression of an exceptionally good pulse. Besides, measurements of the direct pressure by means of the v. Basch sphygmomanometer show that with this valvular lesion the systolic pressure (see pp. 107 and 135 *et seq.*) can be high despite disturbance in compensation. This only apparently contradicts our statement above in regard to the characteristics of disturbance of compensation. For even if we neglect the arguments against the v. Basch principles of pressure-measuring which were stated above (stasis of the wave, action of the blow), we must not forget that the systolic pressure in this valvular lesion should be considered a much less exact measure of the mean arterial pressure than under ordinary circumstances, because a considerable portion of the systolic pressure is of no use to the circulation on account of the regurgitation. And so it happens that an abnormally high systolic pressure for perfect compensation in aortic insufficiency is in reality essential to establish a mean normal pressure in the capillaries. And then even in disturbed compensation the absolute systolic pressure will almost always be normal or above normal. Moreover, in consequence of the quick and high ascent of the pulse wave, the pulse will be felt a longer time than normal on the other side of the pelotte of v. Basch's instrument, and thereby cause an overestimation of the systolic pressure. Finally, even if combined with marked arterial resistance, disturbances of compensation are not necessarily associated with a low blood-pressure (see p. 321).

AORTIC STENOSIS.

Aortic stenosis (Figs. 119 and 120) causes an obstacle to the left ventricular systole. The left ventricle overcomes this obstacle at first by means of its reserve power, and later by the hypertrophy of its walls—

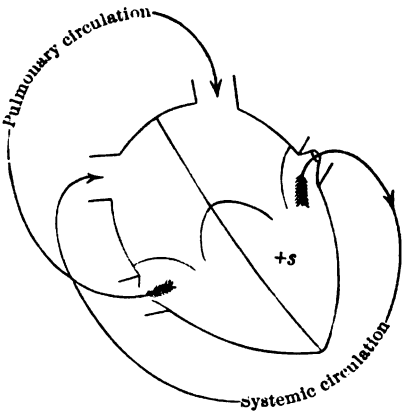

FIG. 119.—Hydraulic diagram of aortic stenosis. (Compare Fig. 113 for explanation.)

ordinarily a pure primary hypertrophy without dilatation (p. 315, Law 1). The hypertrophy of the left ventricle may not be demonstrable clinically, or it may only exhibit an accentuation of the apex

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beat (see p. 293 *et seq.*) without any cardiac enlargement evident to percussion (see p. 183). Sooner or later, however, disturbances in compensation do occur and a secondary dilatation of the left ventricle is added to the hypertrophy (Law 4). This dilatation either disappears when compensation is restored, or may persist anatomically established (see p. 316 *et seq.*). Such a permanent dilatation (p. 317) sometimes develops without being preceded by a real disturbance in compensation. (See p. 318 *et seq.* in regard to the completeness or incompleteness of contraction of such a permanently, secondarily dilated and hypertrophied ventricle.) The dilatation of the left ventricle in aortic stenosis should be sharply distinguished from its dilatation in mitral and in aortic insufficiency. It is not in any sense compensatory; it may be absent in

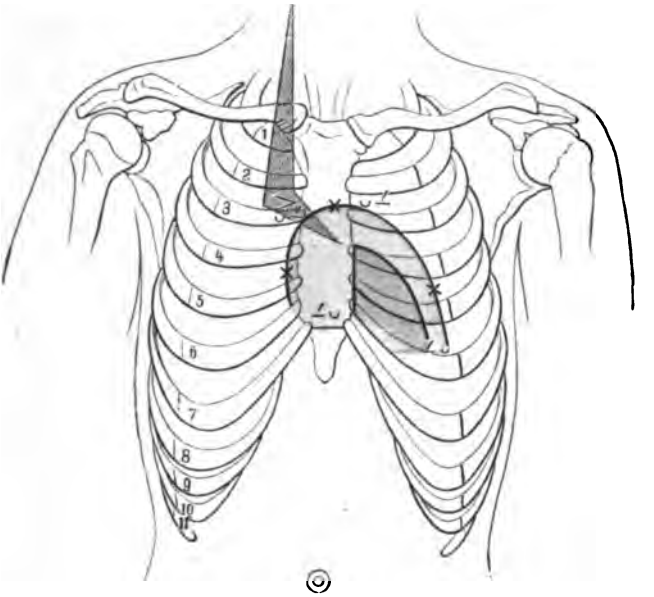

FIG. 120.—Diagnostic plan of aortic stenosis. (For explanation of the signs see p. 321.)

fresh lesions, but in older lesions it is frequently to be found. It need scarcely be mentioned that, through disturbances in the compensation, an aortic stenosis may produce the same effect upon the pulmonary circulation and the right heart (p. 330 *et seq.*) as aortic insufficiency.

The physical signs of aortic stenosis are pictured in Fig. 120. Auscultation reveals a systolic murmur audible over the aortic area in the second right intercostal space, transmitted upward to the vessels of the neck, and sometimes audible over the entire left ventricle. The murmur is also palpable at times, and may even be heard at a distance. The murmurs are, in fact, so constantly loud that the demonstration of a loud systolic murmur argues in favor of aortic stenosis and against accidental murmurs. The conditions for murmur formation (great rapidity of the current, large volume of the stream) are more favorable in aortic

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stenosis than in almost any other valvular lesion. The murmur may possess two maximum points (over the aorta and at the apex) (see p. 274). The most essential sign of aortic stenosis is the *pulsus tardus*, the presence and peculiarity of which have been explained (see pp. 105 *et seq.*, and 127). The tension of this deliberate and tardy pulse wave during the stage of compensation is not necessarily low. Frequently it is not only delayed, but also slowed—that is, less frequent than normal. Evidently the tardiness and slowness essentially lessen the heart's work. In a high grade of aortic stenosis this slowing of the cardiac action is an important condition for the preservation of complete compensation, and is therefore of especial diagnostic significance. Despite the hypertrophy of the left ventricle, the apex beat is said to be frequently weakened; but this is certainly not always the case. Where such a weakened apex beat does appear, Gutbrod-Skoda's theory that it is due to a loss of recoil depending upon slowness of cardiac emptying will not satisfy the conditions. For we know that the apex beat coincides with the closure time of the heart. Hence we must acknowledge that the recoil theory of the heart beat has been finally disproved. Rosenstein's explanation seems more plausible. He claims that the hypertrophy of the left ventricle produces a more rounded shape, and that it is more difficult for such a rounded apex to reach between the ribs, and so the apex beat is weakened. Many cases of aortic stenosis exhibit the strong, slow, heaving beat described upon p. 293 *et seq.* If the moderate force of the apex beat prevents it from readily pushing through the intercostal space, the slowness of the heaving would render its appreciation more difficult.

There is nothing characteristic about the tones in aortic stenosis. Ordinarily they persist. Under some circumstances decided weakness of the tones over the entire left heart makes it probable that a mitral insufficiency complicates the aortic stenosis (see p. 253 *et seq.*). In this lesion the shape of the cardiac dulness as outlined by percussion may be normal; hence the probability of confusion between the systolic murmur of an aortic stenosis and an accidental murmur from atheroma or roughness of the aortic intima. The pulse should distinguish the two conditions. Other kinds of accidental murmurs (anemia, fever) are less difficult to diagnose, because their maximum is not usually over the aorta. The author has already emphasized (p. 331) the frequent error of diagnosing an aortic stenosis because a systolic murmur is heard over the aorta in addition to the diastolic murmur in aortic insufficiency. The presence or absence of the *pulsus tardus* should be distinctive in this case as well. To distinguish the murmur of aortic stenosis from that of mitral insufficiency we should carefully locate its maximum intensity and notice the characteristic, although slight, difference of phase (p. 271 *et seq.*).

VALVULAR LESIONS OF THE RIGHT HEART.

In the diagnosis of valvular lesions of the right side of the heart it is important to remember that they are most frequently congenital

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lesions, and that they rarely originate during extra-uterine life; this is exactly the reverse of left-sided lesions. Nevertheless, acquired right-sided valvular lesions are not quite as rare as is sometimes claimed. When they do originate after birth they are most frequently complications of coexistent left-sided lesions.

TRICUSPID INSUFFICIENCY (Figs. 121 and 122).

As a relative insufficiency complicating left-sided valvular lesions, this lesion occurs quite frequently; but as a true anatomic valvular change, it is rare except in congenital heart disease. The weak right auricle is all that exists behind the tricuspid valve to compensate the disturbance. Its compensatory power is naturally limited, so that the disturbance to the circulation is more serious than in mitral insufficiency. The first result of a tricuspid insufficiency (see the hydraulic

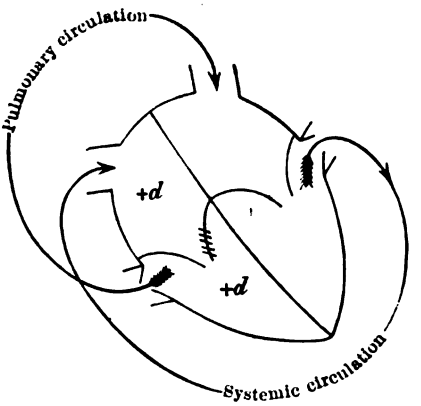

FIG. 121.—Hydraulic diagram of tricuspid insufficiency. (For explanation see Fig. 118.)

scheme, Fig. 121) is a dilatation of the right auricle, because the regurgitating blood distends this chamber during its diastole (p. 316, Law 2). The dilated auricle, having more blood to propel, necessarily hypertrophies (Law 2). It must then force an increased mass of blood into the right ventricle; the latter chamber therefore dilates and hypertrophies (Law 2), and the lesion is compensated in so far as it is possible. The right ventricle, in virtue of its dilatation and in spite of the blood leaking through the tricuspid orifice, is then able to propel sufficient blood with each systole into the pulmonary circulation. Practically this compensation has very narrow limits, because the systole of the dilated and hypertrophied right auricle is not sufficiently powerful to satisfy the right ventricle, and because the inherent muscular weakness of the right auricle does not permit of enough hypertrophy to overcome the defect; hence blood is also dammed back into the veins. When such a result is produced, the amount of venous blood which enters the right heart will depend upon the venous pressure, upon

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the elastic tension of the right auricular walls, and upon the aspiration power of the thorax. Increasing insufficiency of the tricuspid valve will then soon produce the signs of severe stasis, and, ordinarily, the circulation will stop on account of an increasing distention of the venous system. The prognosis of tricuspid insufficiency is therefore much more serious than that of mitral insufficiency unless the tricuspid insufficiency is a relative one, depending upon back pressure.

In this lesion a dilatation of the right auricle and right ventricle can be made out by percussion and sometimes by palpation (see Figs. 122 and 91). Auscultation discloses a systolic murmur over the tricuspid area (p. 267). This murmur can be differentiated from the murmurs of pulmonary and aortic stenosis by a slight difference in phase (p. 271). During compensation the second pulmonic may retain its normal

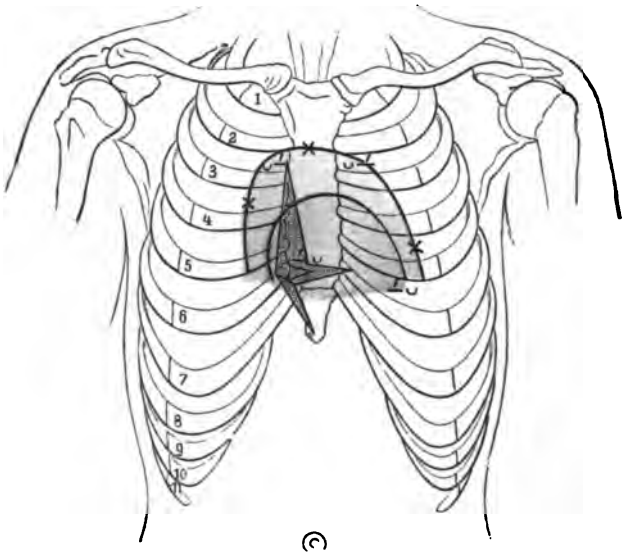

FIG. 122.—Diagnostic diagram of tricuspid insufficiency. (For explanation of the signs see Fig. 90 and p. 321.)

strength; during disturbance of compensation it may be diminished. The heart tones over the right ventricle may remain normal if the insufficiency is slight, or be weakened if the insufficiency is marked, just as in mitral insufficiency (see p. 252). Much the most important and suggestive symptom of tricuspid insufficiency is the regurgitating or positive venous pulse (p. 150 *et seq.*) visible in the jugular veins, often visible as a liver pulse, and visible sometimes even in the small cutaneous veins of the body. The positive venous pulse in the jugular veins is, under some circumstances, accompanied by a systolic tone over the jugular vein, depending upon the systolic tension of the vein-wall and of the valve at the bulb (p. 284). The positive venous pulse is present both during effective compensation and during disturbed compensation. The arterial pulse remains normal during perfect compensation; but good

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compensation is possible only with slight degrees of tricuspid insufficiency. During disturbed compensation it very quickly assumes a dangerous diminution of volume and tension.

TRICUSPID STENOSIS.

This valvular lesion, fortunately, is very rare, both in the congenital and the acquired forms (Figs. 123 and 124). Like tricuspid insufficiency, it can be compensated with difficulty, and then generally for a short time only. (See the hydraulic scheme, Fig. 123.) Compensation takes place in this way: The right auricle, in consequence of the obstruction to the entrance of the blood into the right ventricle, becomes dilated during the first period of the ventricular diastole, when the auricle is relaxed (p. 316, Law 2). To overcome the difficulty of emptying its increased contents through the narrow tricuspid opening by its systole, it must hypertrophy (Laws 1 and 2). Should this

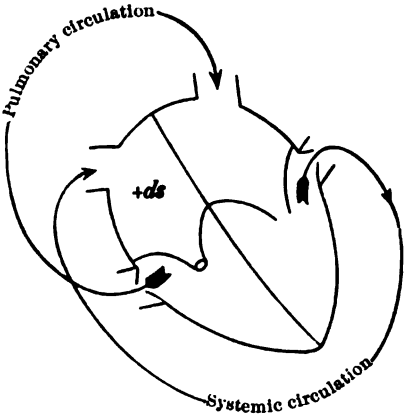

FIG. 123.—Hydraulic diagram of tricuspid stenosis. (For explanation see Fig. 113.)

hypertrophy succeed in emptying the auricle completely, the lesion is compensated. But, just as with tricuspid insufficiency, the hypertrophy of so weak a muscular organ as the right auricle has very narrow limits. Besides, the increased contraction only partially helps the ventricle, because some of the blood is regurgitated into the veins. Mild degrees of stenosis may therefore attain an approximate compensation, but usually the auricle is quickly paralyzed. There is this essential difference between mitral and tricuspid stenosis: The latter must be compensated by the aid of the auricle alone, whereas the former has the additional help of the right ventricle. In tricuspid stenosis no reason exists for a dilatation or a hypertrophy of any other heart chamber except the right auricle. The dilatation of the right ventricle, which is sometimes found, probably always depends upon a coincident insufficiency of the tricuspid.

The physical signs (Fig. 124) are a dilatation of the right auricle, demonstrable by percussion or palpation, and a diastolic, a presystolic-

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ally accentuated, or a pure presystolic murmur, audible over the tricuspid area (p. 269 *et seq.*). If compensation is well maintained the second pulmonary tone may be of normal intensity; with disturbance of compensation it becomes very faint. The tricuspid tones, like the other tones, may retain their normal character during compensation.

Under some circumstances a presystolic or diastolic tricuspid tone may be of some diagnostic service. It is analogous to the presystolic or diastolic mitral tone (p. 258 *et seq.*).

PULMONARY INSUFFICIENCY.[1]

Pulmonary insufficiency, an inability of the pulmonary semilunar valves to close perfectly, is an exceptional valvular lesion and usually congenital (Figs. 125 and 126). Its effect upon the lesser circulation is

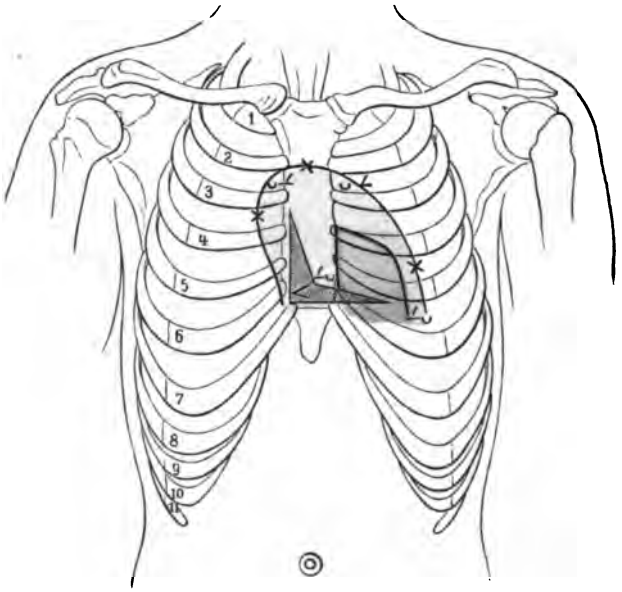

FIG. 124.—Diagnostic diagram of tricuspid stenosis. (For explanation of the signs see p. 321.)

analogous to the action of an aortic insufficiency upon the greater. Compensation (see the hydraulic scheme, Fig. 125) will be understood by referring to that lesion. In this case it depends upon dilatation and hypertrophy of the right ventricle.

The diagnostic signs consist essentially in a diastolic murmur heard over the pulmonic area (Fig. 126) and transmitted downward (p. 268), and in the demonstrable dilatation of the right ventricle. The diastolic murmur, unlike that of aortic insufficiency, is not transmitted to the

[1] See Gerhardt, "Ueber Schlussunfähigkeit der Lungenarterienklappen," *Verhandl. des II. Cong. f. inn. Med.*, 1892, p. 290.

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neck vessels. The tones are generally normal, just as in aortic insufficiency. The *pulsus celer* belongs to the pulmonary circulation, and, so,

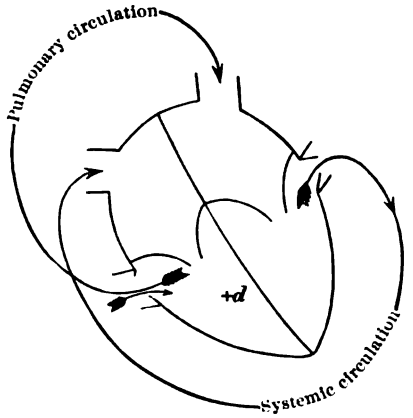

FIG. 125.—Hydraulic diagram of pulmonary insufficiency. (For explanation see Fig. 113.)

naturally escapes notice, or at most betrays itself only by an exceptional pulsation of the pulmonary area, provided the latter is not covered by lung tissue (analogous to the increased aortic pulsation in aortic insuffi-

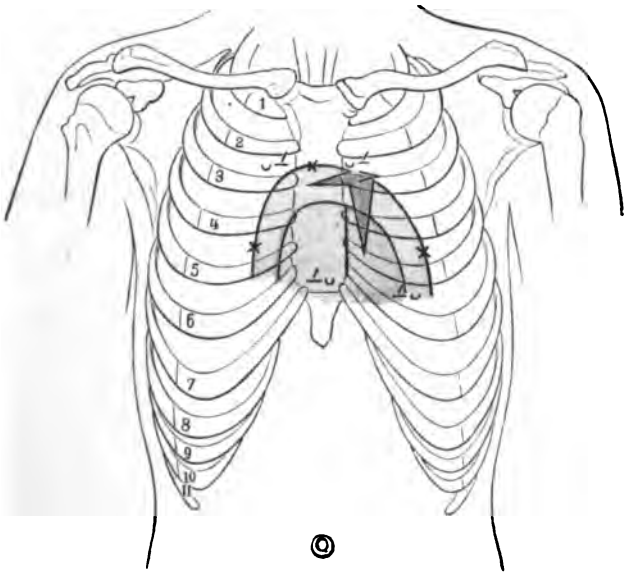

FIG. 126.—Diagnostic diagram of pulmonary insufficiency. (For an explanation of the signs see p. 321.)

ciency, p. 299 *et seq.*). The lack of a *pulsus celer* in the peripheral arteries aids in distinguishing pulmonary from aortic insufficiency.

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Gerhardt[1] has called attention to the occurrence of a double tone audible over the lung at some distance from the heart—*i. e.*, outside of the right scapula (analogous to the crural double tone of aortic insufficiency, p. 282); also of a cog-wheel or jerky respiratory murmur synchronous with the pulse and heard at some distance from the heart. He attributes the latter phenomenon to the vigorous systolic distention of the lung vessels by the pulmonary *pulsus celer*. Both signs are to a certain extent the audible evidences of a *pulsus celer* of the pulmonary artery.

Pawinski[2] has called attention to the appearance of a relative insufficiency of the pulmonary artery in mitral stenosis from the mechanical dilatation of this artery.

PULMONARY STENOSIS.

Pulmonary stenosis (Figs. 127 and 128) is the most frequent of all congenital valvular lesions, and therefore has considerable practical importance. Hypertrophy of the right ventricle (see hydraulic scheme, Fig. 127) is essential for its compensation (Law 1); and to this hyper-

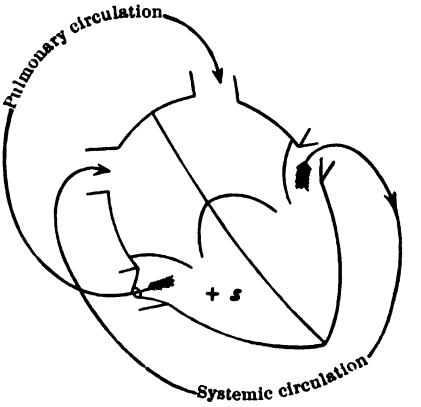

FIG. 127.—Hydraulic diagram of pulmonary stenosis. (For explanation see Fig. 113.)

trophy dilatation is added secondarily by disturbances in the compensation, or sometimes without (Law 4).

The essential physical sign is the systolic murmur audible over the pulmonic area and the right ventricle, and often over the entire lung corresponding to the branches of the pulmonary artery. In distinction to the systolic aortic murmur, the murmur of pulmonary stenosis is not transmitted into the vessels of the neck. Sometimes it may be differentiated from the systolic auriculoventricular murmur by a slight difference in phase (p. 271). There is generally nothing characteristic about the tones. We may not be able to demonstrate a dilatation of the right ventricle, because its hypertrophy takes place only primarily. But ordinarily a paralytic dilatation (Law 4) soon occurs and becomes anatomically fixed.

This lesion is almost exclusively congenital, and is frequently combined with pulmonary phthisis and clubbed fingers (p. 59).

[1] *Loc. cit.* [2] *Deutsch. Arch. f. klin. Med.*, 1894, vol. liii.

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DIAGNOSIS OF COMBINED VALVULAR LESIONS.

The diagnosis of single valvular lesions generally seems comparatively simple; but it is sometimes no easy task to distinguish correctly, when, as so frequently happens, several lesions are present at the same time. The relations become complicated, because one valvular lesion influences the other in regard to the changes of pressure within the heart. This influence may be expressed by alterations in the strength of the heart tones, by the presence of dilatation and hypertrophy of individual chambers, and by the peculiarity of the pulse. With combined valvular lesions the clinician's task assumes a special difficulty, because he must analyze complex murmurs into several individual ones, deter-

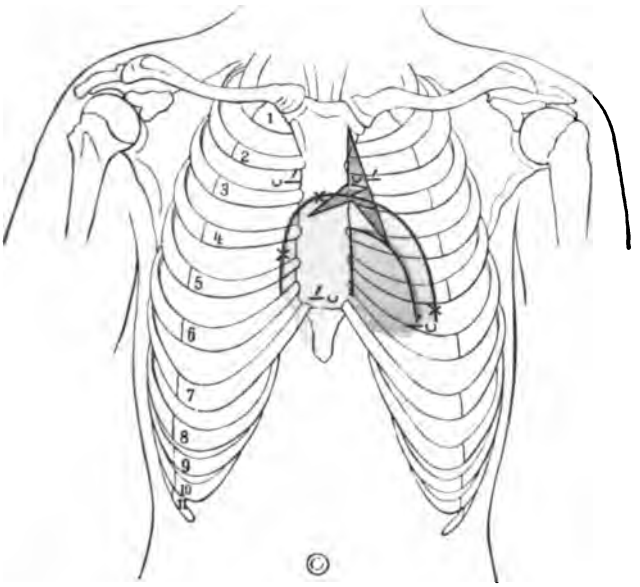

FIG. 128.—Diagnostic diagram of pulmonary stenosis. (For an explanation of the signs see p. 321.)

mining their origins, and decide which are systolic and which are diastolic.

The most important rule for the diagnosis of such complicated cases is to heed only those physical signs which are characteristic of one definite valvular lesion; then, starting with the supposition that this lesion is actually present, to analyze the other symptoms and determine whether any symptom disproves this supposition, and, further, to decide whether all the signs can be explained by this one hypothesis. If one lesion is not sufficient to explain all the signs, those not yet explained should be considered further. In this way, step by step, we can complete the diagnosis of combined valvular lesions. The task may be lightened by first considering the more frequent valvular lesions; and, in case there is no question of a congenital disease, in neglecting the more unusual lesions of the right heart, unless, of course, some signs

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persist which cannot be explained by the supposition of only left-sided changes. For illustration: suppose, in a case with several murmurs, a plain presystolic murmur can be heard at the apex. This murmur is in all probability due to a mitral stenosis, because tricuspid stenosis is so rare. We then try to see if this hypothesis will explain all the signs. If a systolic murmur can also be heard we must search for some other cause. We estimate its maximum point, its relations of transmission, its exact phase (p. 270 *et seq.*), and under some circumstances the peculiarity of the pulse, and soon decide whether this systolic murmur arises at the aortic or at the mitral orifice. We then diagnose two lesions—a mitral stenosis and insufficiency—and review the clinical picture to determine whether all the signs are satisfied, or whether,

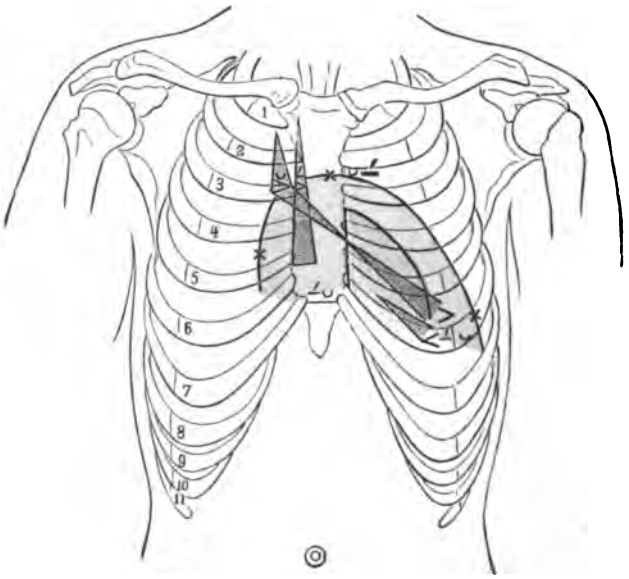

FIG. 129.—Diagnostic diagram of a complicated valvular lesion: Mitral insufficiency and stenosis and aortic insufficiency, possibly an aortic stenosis as well. The last cannot be diagnosed from the cardiac signs alone. The pulse must first be carefully examined. (For an explanation of the signs see p. 321 *et seq.*)

perhaps, we need to assume a third lesion. And so, step by step, we arrive at a complete diagnosis.

To distinguish whether a murmur audible over a large area of the heart arises at one or more places—in other words, whether it is composed of several different murmurs—we should construct a figure of the minimum points, according to the rule of p. 272 *et seq.* This method is very important in diagnosing combined valvular lesions.

A careful attention to the evidences in the vessels (peculiarity of the pulse, degree of the cyanosis, venous pulse, arterial tones, etc.) will give just as important information for the diagnosis of combined as of single lesions.

In general, here as well as everywhere else, the systematic analysis

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of a clinical picture can only be acquired by considerable practice, and even then only after thorough reflection.

Figure 129 illustrates such a combined valvular lesion.

CONGENITAL VALVULAR LESIONS; ABNORMAL COMMUNICATION OF THE HEART CAVITIES; ADMIXTURE CYANOSIS.

Experience has taught us that in making diagnoses of acquired valvular lesions we can narrow the possibilities to those of the left heart, excepting the possibility of the rather rare tricuspid insufficiency. In congenital lesions we can confine our attention mostly to the right heart. Perhaps the reason is that during intra-uterine life the right side of the heart has much more work to do than during extra-uterine life, because the right ventricle supplies blood not only to the pulmonary circulation by means of the pulmonary artery, but also to part of the systemic circulation by means of the ductus Botalli.

The great difficulty in the diagnosis of congenital heart disease is that, besides the valvular lesions which depend upon endocarditis, there are very often developed obstructions, malformations, abnormal communications between heart cavities, etc., and all these are mutually influenced, etiologically and clinically, in a very complicated way.

The most frequent and practically important conditions of malformation and obstruction to be considered in the diagnosis of congenital lesions are: a *patent foramen ovale, ventricular septum, or ductus Botalli; origin of the aorta from both ventricles, connection of the pulmonary artery, closed at its origin, with the aorta through the ductus Botalli* (the right heart must then communicate with the left through the foramen ovale, or through an opening in the ventricular septum); *transposition of the great vessels*, so that the aorta comes from the right, the pulmonary artery from the left, ventricle; *obliteration of the aorta at the mouth of the ductus Botalli* (life is then preserved by the formation of a collateral circulation between the upper and the lower part of the aorta). It need not be said how difficult it is to recognize these conditions, whether they appear isolated or as complications of fetal valvular defects. Only a few hints can be given in the present state of modern diagnosis.

Much stress is ordinarily laid upon the exceptionally pronounced cyanosis which appears so frequently in congenital heart disease, and which is often attributed to the admixture of venous with arterial blood through some communication. The conditions are, however, especially favorable for a high grade of cyanosis in right-sided valvular lesions without supposing the admixture of arterial and venous blood, because, on the one hand, compensation is here either very poor or soon disturbed (*e. g.*, tricuspid lesions), and because, on the other hand, in all right-sided, even in pulmonary, lesions, as soon as there is a disturbance in compensation, the stasis must be concentrated in a most direct and intense way upon the systemic veins. Therefore, in congenital cardiac disease it is incorrect to make a diagnosis of an abnormal communication and consequent admixture of arterial and venous blood simply from marked cyanosis. Many authors even go so far as to deny the possibility of such admixture cyanosis, on account of certain mechanical considerations. They are supported with some reason by the fact that communications between the ventricles and between the auricles have sometimes been found at autopsies without any signs during life of such a cyanosis. And they contend that this lack of admixture cyanosis (despite the communication) is not surprising, because the two abnormally communicating heart cavities contract coincidentally, so that no blood need be sent from one to the other. Certainly, every abnormal communication of the cardiac cavities does not cause admixture cyanosis; but it is probable that under certain conditions venous blood may be mixed with arterial. The following examples may be cited in evidence:

A mere defect in the septum between the auricles and the ventricles ordinarily causes no admixture cyanosis; yet it is perfectly easy to understand that such an effect may result if at the same time a right-sided valvular lesion is also present.

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With tricuspid insufficiency, for example, and an open foramen ovale, the ventricular systole may force the venous blood out of the right ventricle not only into the right, but also, in consequence of the increase of pressure, into the left auricle. With tricuspid stenosis, during ventricular diastole the blood from the right auricle may partly flow in the direction of least resistance through the foramen ovale into the left auricle and ventricle. Just so it is conceivable that in pulmonary stenosis during diastole the blood from the right ventricle is forced into the left ventricle through a patent ventricular septum, the presumption being that the right ventricle has become so strongly hypertrophied that it overbalances the aortic pressure which acts upon the other side of the communicating orifice.

The plainest and, perhaps, also most frequent example of admixture cyanosis is "straddling" of the aorta—*i. e.*, its origin from both ventricles and insertion above the defect in the ventricular septum. Here, of course, the aorta must contain mixed blood. We observe this appearance in congenital pulmonary stenosis.

Patency of the ductus Botalli alone can hardly occasion admixture cyanosis, because the aortic pressure, if no other anomaly is present, is always higher than that of the pulmonary artery, so that the blood could pass through the aorta into the pulmonary artery, but not in the reverse direction. But mixed blood must flow into the aorta if the ductus Botalli is the only outlet of the pulmonary artery, which is completely closed at its opening into the right ventricle, because the aorta must obtain its blood from the opening in the ventricular septum—*i. e.*, from the right ventricle.

In congenital valvular lesions, therefore, it is reasonable to assume the possibility of admixture cyanosis, and to be fairly certain of its presence, if the cyanosis is very pronounced while the veins are very little dilated (marked cyanosis with slight stasis).

In any given case, however, the cause of the admixture cyanosis is not so easily determined. In tricuspid lesions we should naturally first suppose an open foramen ovale; whereas, in pulmonary stenosis a patent ventricular septum, with or without a straddling aorta, would be more probable. Under some circumstances a systolic murmur at the cardiac apex may be a positive sign of a ventricular septum defect. This sign is, however, of very little practical value, because in pulmonary stenosis just where a patent ventricular septum is to be taken into consideration the systolic murmur is frequently very plainly audible over the entire heart, because it proceeds from the entire right ventricle (p. 267 *et seq.*).

It is difficult to diagnosticate patency of the ductus Botalli. We should remember that this anomaly very often accompanies congenital pulmonary stenosis. Zinn and Hermann Müller[1] have called attention to a band-like dulness reaching upward and to the left of the upper part of the sternum. An accentuation of the second pulmonic has been repeatedly stated to be a result of a patent ductus Botalli, because with this abnormality the pulmonary arteries are affected by the aortic pressure. Müller attributes diagnostic significance to the systolic and diastolic murmurs which appear over the great vessels, and states that they sometimes merge into a practically continuous murmur. The systolic murmur depends upon the systolic passage of blood from the aorta through the narrow ductus Botalli into the pulmonary aorta; the diastolic murmur, upon the diastolic continuation of this current. The congenital obliteration of the aorta at the place of origin of the ductus Botalli is, on the contrary, very easy to diagnose, whether it is associated with congenital valvular lesions or isolated. The collateral circulation by which the blood flows from the peripheral arteries of the upper part of the body (subclavian, etc.) to the lower part of the aorta becomes visible under the skin and makes the diagnosis clear. Such an obliteration may exist without any disturbances of the circulation.

There are many questions in the pathology and diagnosis of congenital heart lesions which still lack solution.

[1] *Corresp.-Bl. f. schw. Aerzte*, 1899, p. 449.

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ANEURISM OF THE AORTA.

Aortic aneurisms are most frequently situated upon the ascending aorta or upon the arch of the aorta. When an aneurism reaches a certain size we can detect a prominence in the upper intercostal spaces to the right of the sternum ; and appreciate it by palpation as well. Small aneurisms may not exhibit any prominence, but the pulsation can generally be appreciated by palpation. If the aneurism reaches to the surface, percussion will show a decided dulness, which generally merges with the cardiac dulness. If a layer of lung remains in front of the aneurism, the dulness may be either entirely absent or there may be only a moderate, deep dulness, demonstrable by strong percussion. Here, too, the pulsation may be transmitted through the overlying layers of lung up to the surface. Auscultation very frequently reveals a systolic murmur, which may sometimes be appreciated by palpation. The entrance of the blood into the aneurismal sac produces this murmur because there is a widening of lumen in the blood-path (p. 261 *et seq.* See

FIG. 130.—Large aneurism of ascending and transverse arch of aorta (New York City Hospital).

Fig. 103, III.). This murmur, like that of aortic stenosis, is transmitted most distinctly in the direction of the aortic current—*i. e.*, to the neck vessels. Diastolic murmurs are, however, sometimes due to aneurisms. If there is no complicating aortic insufficiency, such a murmur arises from the regurgitation of blood into the sac, which is stretched during diastole. Similar murmurs may be heard over the abdominal aorta.[1] Under such circumstances a diastolic murmur may be one of the earliest signs of an aortic aneurism, and may readily lead to a confusion with aortic insufficiency unless the aneurism is evident to inspection, palpation, and percussion. Frequently, however, the diastolic murmur over an aortic aneurism depends upon a combination of the aneurism with an aortic insufficiency. For the sac, continually increasing in size, gradually produces a dilatation of the aortic orifice, and so a relative insufficiency, or else the endarteritis, which is always present in an aneurismal aorta, attacks the valves as well. Unless an aortic aneurism is combined with a valvular lesion, the heart need not be hypertrophied nor dilated, because an aneurismal dilatation of the aorta by itself offers no considerable obstacle to the circulation. Large aneurisms, on the

[1] v. Leyden, *Deutsch. med. Woch.*, 1900, No. 23, p. 365 *et seq.*

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contrary, generally crowd the heart somewhat to the left, so that the apex beat lies farther outside than normally. To determine the exact seat of the aneurism in relation to the origin of the innominate and of the left carotid and subclavian arteries, we should accurately compare the exact time and strength of the carotid and radial pulses on the two sides. The aneurismal dilatation frequently delays the pulse in those arteries which arise from the aorta beyond the aneurism, because a sort of pump action retards the pulse wave. In other cases the delay is due to the fact that some of the arteries arise from the sac itself, and are narrowed at their origin. The pulse may then be also abnormally small. In many cases certain accompanying signs are important in confirming the diagnosis of aneurism. They are to be attributed to the pressure of the aneurism upon the trachea, with dyspnea; upon the esophagus, with difficulty in swallowing; upon the left bronchus, with a diminished respiratory murmur over the left lung; upon the left, rarely upon the right, recurrent laryngeal, with a unilaterial vocal paralysis. A venous col-

FIG. 131.—Aneurism of the descending arch of the aorta (Dr. Cutler, Massachusetts General Hospital) (see *x*-ray).

lateral circulation visible in the skin over the chest is of the same diagnostic importance for an aneurism as for other intrathoracic tumors (p. 55 *et seq.*). It results from the pressure of the aneurism upon the great veins inside the thorax. Mention should be made of the *Oliver-Cardarelli sign* (tracheal tug) (p. 299). The symptoms of the rare aortic aneurisms which are situated at some distance from the great vessels of the heart (thoracic and abdominal aorta) can be readily deduced.

In consequence of the increased systole of the left ventricle, an aortic insufficiency may cause a considerable diffuse dilatation of the ascending aorta and produce an abnormal pulsation and increased dulness over the upper intercostal spaces to the right of the heart (see Fig. 92). Pain is a prominent symptom in this condition as well as in aortic aneurism, so there may be some difficulty in the differential diagnosis. The most important criteria for diagnosing a sacular aneurism are the exhibition of compression signs (recurrent paralysis, etc.), of the

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Oliver-Cardarelli phenomenon, and of difference in the pulses. If an aneurism forms a circumscribed, prominent tumor, dull to percussion, there will be no possibility of confusion.

Recently the Rõntgen rays have been repeatedly employed in the diagnosis of aortic aneurism. They allow of certain conclusions, however, only when the aneurism is in the form of a pulsating tumor which is distinctly separated from the heart. These are cases in which other methods of examination usually determine the diagnosis. In contrast to these cases, there are many others in which the Röntgen examination will not certainly differentiate an aortic aneurism from a diffuse dilatation of the aorta due to aortic regurgitation, or even from a dilated right ventricle. For the details of the Röntgen examination the reader is referred to the special works upon the subject.

PERICARDITIS.

Physical examination generally reveals a *dry pericarditis* by a characteristic rubbing friction sound (see p. 279 *et seq.*). So long as the layers of the pericardium are not separated from one another by a fluid exudation throughout their entire extent, *exudative pericarditis* will also

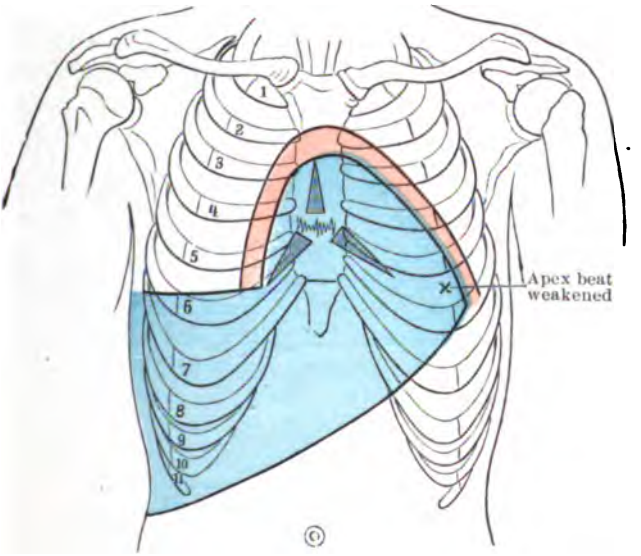

FIG. 132.—Diagnostic diagram of an exudative pericarditis (see pp. 279 and 321 for an explanation of the signs).

cause a pericardial friction rub, because there is always a deposition of exuded fibrin in addition to the fluid (p. 280). The cardinal sign of exudative pericarditis is, however, a characteristic cardiac dulness which is increased upward. It ordinarily assumes the form of a blunt triangle with its base below (p. 187 *et seq.*). The width of this dulness is increased in the sitting or erect posture (p. 187 *et seq.*), the apex beat is weakened (p. 294), and the heart tones are also enfeebled (p. 250), especially in the outer portions, on account of the interposed exudation. In some cases the apex beat lies within the borders of superficial cardiac

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dulness (p. 295 *et seq.*), and frequently there is a visible prominence of the precordia (p. 34). Beyond these features the diagram (Fig. 132) will illustrate the entire clinical picture of exudative pericarditis. Such a border of deep cardiac dulness does not always occur (see p. 185 *et seq.*), and the heart is apparently laid bare by the crowding back of the lungs, causing only a very large superficial dulness.

GRAPHIC EXPRESSIONS FOR THE PHYSICAL SIGNS IN PULMONARY CASES.

In the sections upon Percussion, Auscultation, Inspection and Palpation we have described and explained the individual physical signs of pulmonary and pleural affections, and so now we need only add a concise abstract of our method of combining these signs in order to make a general picture in any given case. For the sake of simplicity and brevity we employ graphic expressions to describe the lung conditions. These have been found, in the Clinic at Bern, both practical and comprehensive.

The rules for such graphic expression are simple and easily memorized. We represent the respiratory murmur audible over a certain area of the lung by a small right angle, and mark it at the corresponding spot upon a chest diagram. This sign may be modified in various ways, according to the modification of quality of the respiratory murmur. The vertical limb of the angle represents the inspiratory murmur; the horizontal, the expiratory murmur; the length of the limb shows its duration; the thickness, its intensity. A simple, smooth, straight line signifies normal vesicular breathing; a dotted line, cog-wheel breathing; a toothed line, rough vesicular breathing. A small cross-line on the limb signifies mixed breathing; two cross-lines, pure bronchial breathing. From these rules we can readily understand the following:

- Vesicular breathing.
- Weak vesicular breathing (diminished).
- Coarse vesicular breathing.
- Coarse vesicular breathing with prolonged expiration.
- Rough vesicular breathing with prolonged expiration.
- Bronchial breathing.
- Mixed (bronchovesicular) inspiration with bronchial expiration.
- Vesicular inspiration with bronchial expiration.
- Mixed (bronchovesicular) inspiration with prolonged expiration.
- Cog-wheel vesicular breathing.
- Cog-wheel inspiration with bronchial expiration.
- Indistinct (feeble) breathing, etc.

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The râles are designated thus:

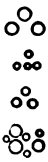
Dry râles:

Sonorous and sibilant.

Crackling (creaking).

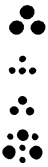
Moist râles:

(*a*) Non-consonating or non-resonant.



Large, coarse,
Small, fine,
Medium,
Mixed,
} Moist or bubbling.

(*b*) Consonating or resonant.



Large, coarse,
Small, fine,
Medium,
Mixed,
} Moist or bubbling.

Inspiratory râles are designated by prefixing the letter "i"; expiratory, by prefixing "e"; *e. g.*,

Inspiratory, non-consonating, mixed moist or bubbling râles, "*i*."

Inspiratory and expiratory sonorous and sibilant, *ie*.

Crepitant râles or crepitation are designated by prefixing Kn.

A pleuritic or a pericardial rub is designated by the sign: , . If it is possible to confuse a pleuritic with a pericardial rub, "pl" or "pe" should be prefixed.

Tone phenomena are expressed as words, or abbreviations may be used, as:

ty = tympanitic note.
hty = high tympanitic note.
lty = low tympanitic note.

In recapitulation we may mention (see p. 164) that a blue surface corresponds to the superficial dulness—*i. e.*, with light percussion—the red, to the deep dulness. A mixed color is employed for a dulness which can be demonstrated as well by deep as by superficial percussion. The intensity of the color represents the intensity of the dulness. Palpable borders are represented in black. The border lines are generally so drawn that the form of the dulness and the direction of the lines correspond as nearly as possible to the reality.

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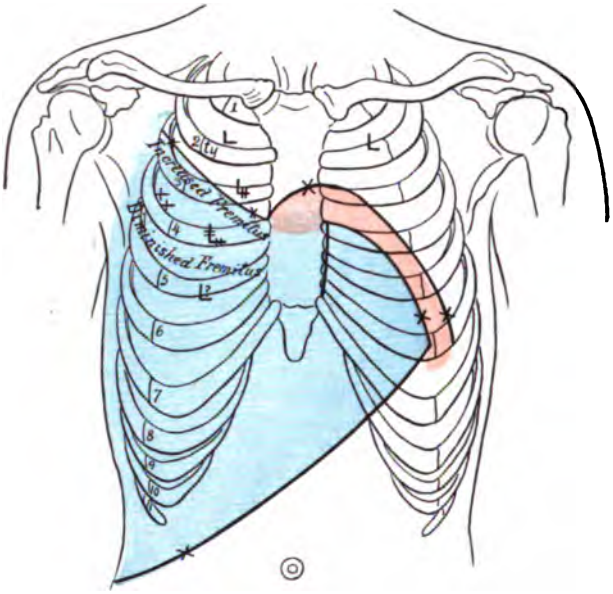

Fig. 133.—Physical signs in right pleurisy with effusions.

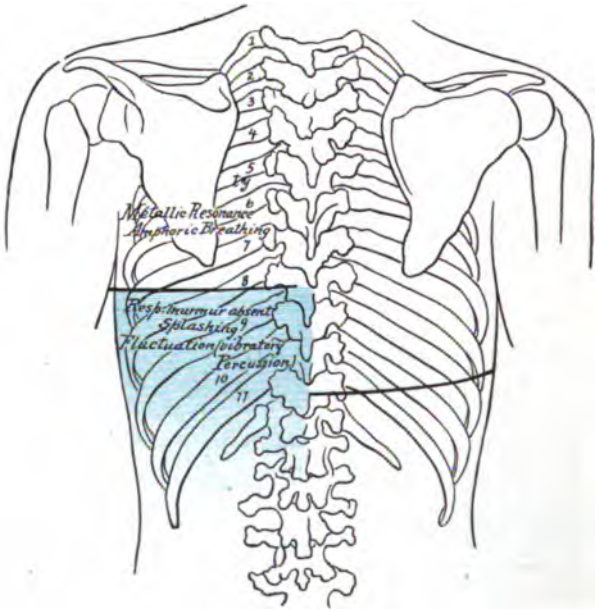

Fig. 134.—Physical examination in left pyopneumothorax (see Fig. 98.)

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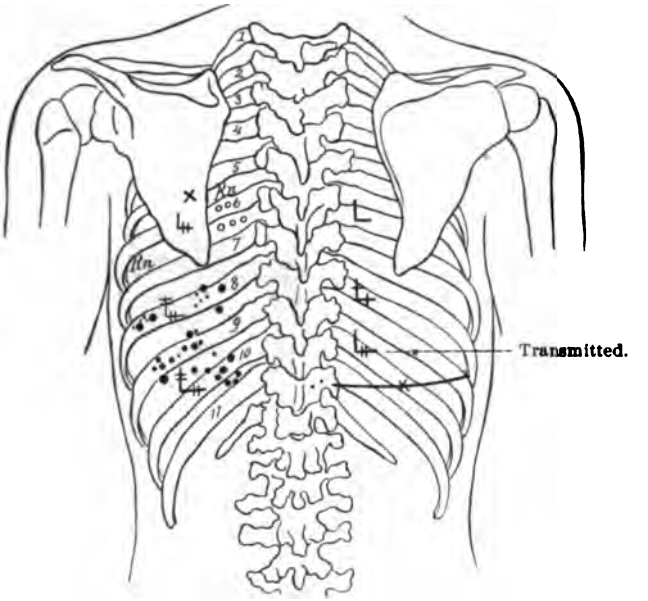

Fig. 135.—Physical examination in left croupous pneumonia.

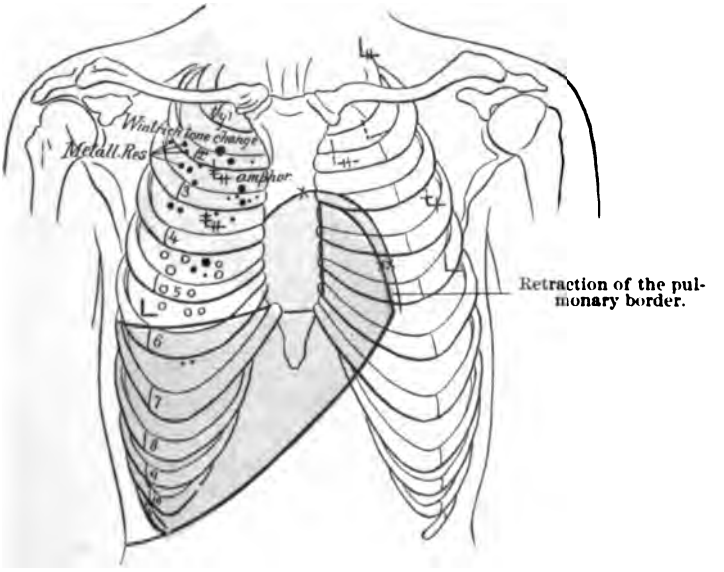

Fig. 136.—Physical signs in pulmonary phthisis: Marked change on the right; beginning on the left.

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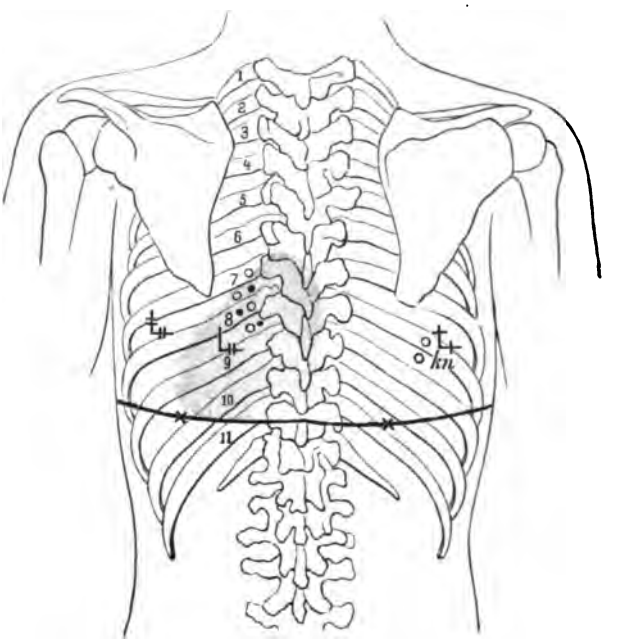

FIG. 137.—Physical signs in catarrhal pneumonias, infarcts, etc.

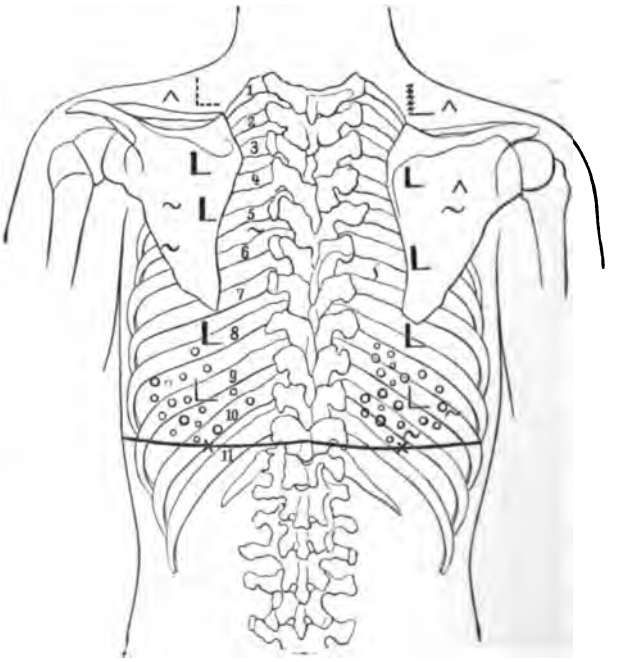

FIG. 138.—Physical examination in diffuse bronchitis.

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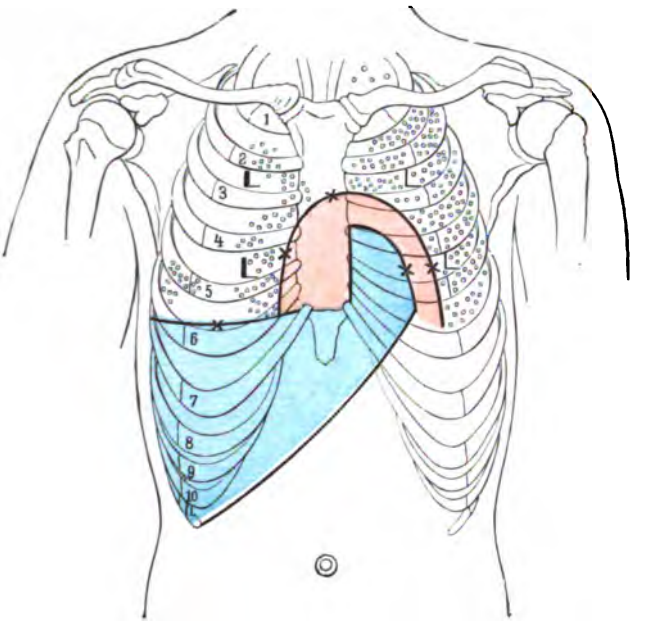

FIG. 139.—Physical examination in capillary bronchitis or miliary tuberculosis.

EXAMINATION OF THE STOMACH AND STOMACH CONTENTS.

UNDER the examination of the stomach we shall consider not only the estimation of its size and position, and whatever other results may be determined by palpation (pain, demonstration of tumors, etc.), but more particularly the methods of testing its specific functions. Purely physical methods of examination furnish such meagre results that we have to depend upon the last-named methods for most of the diagnostic points. Therefore the bulk of the following section will be devoted to the functional examination of the stomach, and especially to the study of its secretion relations. Under the examination of air-containing abdominal viscera we have already alluded to many points in physical diagnosis (compare pp. 196 *et seq.*, 215 *et seq.*, and 301 *et seq.*). We have intentionally postponed until now any more minute consideration of the physical methods of examining the stomach, because such methods reveal but isolated symptoms which are of no value except in connection with others. Many of the cases require the employment of the stomach tube, although its use is attended with certain discomforts, and at times even with injuries, to the patient. Still, quite a number of important diagnostic points may, of course, be obtained without using the tube, so that it is advisable to employ first the other methods of examination.

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METHODS OF EXAMINATION WITHOUT EMPLOYING THE STOMACH TUBE.

The subjective symptoms in the diagnosis of stomach disorders play an important part—*i. e.*, the complaints, the kind of pain or vomiting, when the latter occurs, the character of the stools, etc.—in short, the history in the broadest sense of the word. But all this belongs rather to special pathology.

For the objective examination, which chiefly concerns us, we refer to the sections upon Inspection, Palpation, and Percussion of the Abdomen. But a few special points must be taken up here.

THE DETERMINATION OF THE SIZE, POSITION, AND SHAPE OF THE STOMACH. THE SO-CALLED SPLASHING SOUNDS OF THE STOMACH.

It is difficult to estimate the size of the stomach by percussion immediately, but, if the abdominal walls are lax, inspection will often show the outlines of the stomach, the greater and sometimes the lesser curvature as well. Palpation will sometimes permit a differentiation of the tense stomach from the less tense intestines. If these methods of examination afford no definite result, as is frequently the case, we may fill the stomach either with gas or fluid. The former may be accomplished by giving the patient a teaspoonful of sodium bicarbonate, and immediately afterward the same amount of tartaric acid, each salt dissolved in a half-glassful of water. The alkaline solution should always be swallowed first, because the acid solution might cause pain if the mucous membrane is sensitive, and especially if a gastric ulcer is present. The admixture of the two solutions produces carbonic acid gas, which dilates the stomach like an air cushion, so that its outlines can be easily defined by inspection, palpation, and percussion. If this test is successful, percussion is hardly necessary. The experiment is, however, not always successful, for the stomach may rapidly expel the gas, either upward or downward. Again, a large dilated stomach might require the development of more gas, and so we should have to repeat the attempt with a second and larger dose of effervescing powder. It is not advisable to use too great an amount at first, because it may distend the stomach too much and cause pain. Inflating the stomach with air by means of an ordinary Davidson syringe is free from all these disadvantages and the amount of air can easily be controlled.

A reasonably accurate estimate of the size of the stomach can be reached in some cases by noting the change in percussion which ensues when the patient while erect swallows water little by little. It is best to commence with the fasting stomach, which is usually retracted and entirely covered by intestine. After one or two glassfuls of warm water have been swallowed, the fundus will be dropped downward and forward, and light percussion will bring out an approximately half-moon-shaped dulness in the region of the greater curvature (Fig.

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140, *a*). It is perfectly easy to see that this dulness corresponds to the liquid within the stomach, for if the patient swallows more water the size of the dull area will be increased upward, provided the stomach-wall is of normal resistance (*b*); but with a lax wall or with a pathologic dilatation of the stomach, the weight of the fluid will depress the greater curvature (*c*). This method is generally less valuable than inflation with gas; besides, it is annoying and sometimes distressing to the patient, especially so unless moderately warm water is employed.

The dislocation of the spleen in association with gastric dilatation should always be borne in mind. The enlarging stomach draws it downward, by means of the gastrosplenic ligament, until it can be readily palpated.

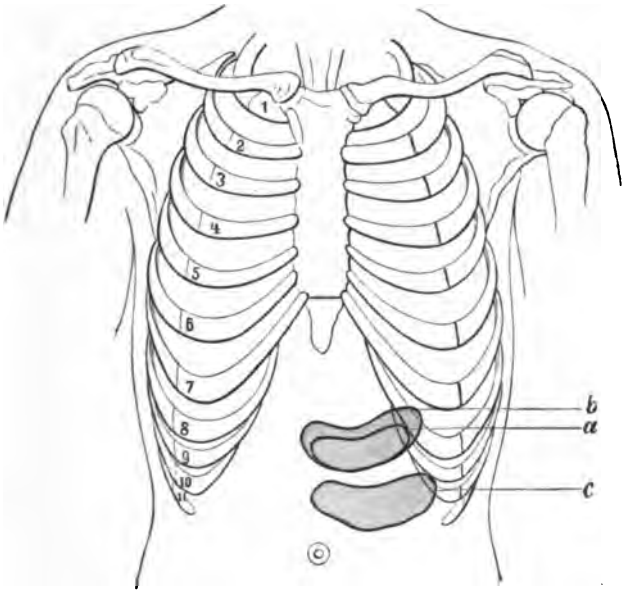

FIG. 140.—Dulness due to liquid in the stomach, percussion in the erect posture: *a*, With a small amount; *b* and *c*, with larger amounts; *b*, with normal gastric tone; *c*, with relaxed gastric tone—*i. e.*, stretching of the greater curvature by the weight of the fluid.

The normal position of the greater curvature should not be lower than perhaps two finger-breadths above the navel (compare Fig. 74). But in estimating the size of the stomach we must remember that the greater curvature is always depressed in gastroptosis, either in the congenital form or especially in that resulting from *enteroptosis*, and also in the anomalous loop-shaped type; and yet in both these instances the stomach need not be really dilated and its functions may still be normal. Therefore it is advisable to map out the position of the lesser as well as of the greater curvature, and both can be best accomplished by inflating with gas.

The capacity of the stomach varies considerably even under physiologic conditions. This variation depends largely upon the diet—*e. g.*, a

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person who lives chiefly upon vegetables, such as potatoes, exhibits a very large stomach, although it need by no means be diseased. Increased capacity then signifies only an enlargement of the stomach. Before a stomach can justly be called a dilated stomach, or "stauungsmagen," we must be sure that the enlargement is accompanied by a loss of motility —*i. e.*, by motor insufficiency.

Splashing sounds in the gastric region (see later section upon Gastric Motility) elicited by so-called "dipping" percussion depend upon the presence of air and fluid. For this purpose the patient is best examined in the dorsal decubitus. With the left hand placed flat upon the epigastrium for palpation, and the ear near the body, the examiner strikes gently with the right hand in the left hypochondrium, or upon the lower left ribs. The splashing may then be felt as well as heard. Splashing sounds do not necessarily mean a dilatation or a stomach with insufficient motor power unless they are elicited when the stomach should be empty—*e. g.*, in the morning before breakfast, or more than seven hours after an ordinary meal. If heard or felt under such circumstances the sign means motor insufficiency of the stomach, which is most common in pathologic dilatation of the stomach. Again, if the splashing can be obtained over an area larger than the normal (see above), then we may consider the organ enlarged.

A very decided splashing sound elicited by quite gentle tapping over the stomach area suggests, however, a gastric atony, even if obtained within the time limits mentioned above. We might call this "superficial splashing," in contrast to "deep splashing" elicited by vigorous blows. This superficial splashing merely shows that the stomach-walls are relaxed (gastric atony), but does not necessarily mean that the stomach is dilated (gastric dilatation). On the other hand, a pathologically dilated stomach, or "stauungsmagen," need not show any decided signs of atony—*i. e.*, it need not furnish any superficial splashing, especially when the gastric dilatation is due to stenosis of the pylorus.

A pathologic dilatation of the stomach which does not lead to a superficial splashing, and that condition which does lead to a superficial splashing—*i. e.*, atony—are two fundamentally different lesions, and should be distinguished properly one from the other. The foregoing explanation of the significance of superficial splashing should not be misconstrued, as it has been.[1] For the author does not invariably make a diagnosis of motor insufficiency of the stomach merely from the demonstration of a superficial splashing. Nor does he regard motor insufficiency and atony as identical conditions. By atony he understands merely a diminished tension. Many such *gastric atonies* are never complicated by any other symptoms, and he especially wishes to insist that the presence of food in the stomach need not be in any way prolonged by mere atony. He considers "atony" diagnostically significant, not as a name of a disease, but merely as a name of the physical condition of the stomach, with which, of course, diseased conditions of the gastric

[1] Elsner, *Berlin. klin. Woch.*, 1901, No. 16.

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function may under some circumstances be combined. So far as the significance of "superficial splashing" is concerned, it should be noted that even relaxed abdominal walls may favor the origin of "superficial splashing," because the tension upon the abdominal contents equals the sum of the pressure of the abdominal wall and of the gastric musculature, and because a superficial blow naturally has a stronger action the less the tension of the abdominal walls. From perfectly evident reasons "superficial splashing" will be favored by a low position of the stomach (gastroptosis), quite independent of the usual association of this position with gastric atony. It may still further be noted that the atony of the gastric muscularis may be entirely concealed by pronounced passive tension of the gastric wall due to an abundant meal. Then, despite the atony, the full stomach, up to a certain time, will not furnish any "superficial splashing."

Sometimes a splashing may be wrongly referred to the stomach when it really proceeds from the colon filled with liquid feces and gas, as in some cases of diarrhea. The existence of diarrhea and the demonstration of a similar splashing along the ascending and descending colon will usually prevent any confusion. Neurasthenics and hypochondriacs sometimes consult their physician in regard to a splashing sound which they produce themselves in the gastric region by contracting and retracting their abdominal walls. This sound may be a true splashing, but it is important only when it occurs under the conditions mentioned above. It may be produced when the stomach is quite normal and nearly empty by an alternate separation and rubbing together of the stomach or the intestinal walls.

EXAMINATION OF THE GASTRIC FUNCTIONS WITHOUT THE USE OF THE STOMACH TUBE.

The functions of the stomach are these: (1) digestive, (2) receptive, (3) antiseptic. In the first place it alters albuminoids and carbohydrates and partly absorbs them. In the second place it acts as a well-equipped reservoir, supplying the food for the intestines, which completely digest and assimilate it; with such help we are able to limit the ingestion of food to definite mealtimes. In the third place it acts as a sort of germicide, protecting the intestines from the injurious action of the noxious micro-organisms contained in the food.

EXAMINATION OF THE VOMITUS.

Considerable information about the chemistry of digestion may be acquired from carefully examining the vomitus. Even a macroscopic examination will enable us to draw valuable conclusions, especially in regard to the digestion of meat, if the vomiting occurs during the time when the digestion ought to be pretty well advanced physiologically. If we find bits of unchanged meat or albumin in the material vomited two to three hours after eating, we are justified in assuming some disturbance of albuminoid digestion. The changes of other kinds of

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food in the stomach are less characteristic. Bread, however, should be altered within three hours after ingestion to a uniform mass of purée-like consistence, which settles to the bottom when the vomitus is allowed to stand. So that if the vomitus contains crumbs of unaltered bread, perhaps covered with mucus, we are usually justified in assuming disturbed albuminoid digestion, depending usually upon a deficient secretion of hydrochloric acid. We may also employ a dilute iodin solution to determine the degree of amyloid digestion, as will be described later in connection with the Test Breakfast (p. 371 *et seq.*). But we must remember that after the completion of normal gastric digestion we still find in the stomach contents microscopically numerous, apparently intact, meat shreds and starch granules. Hence, very little dependence can be placed upon the microscopic examination of the vomitus. The vomitus should therefore be subjected to the same chemical tests as will be described later for the expressed test breakfast. The results will, however, not be very valuable, because gastric digestion varies markedly with the character of the food ingested and the length of time since ingestion. Free hydrochloric acid should nevertheless be found in the gastric juice within one to two hours after eating an ordinary meal; and the filtrate from the vomitus should digest fibrin or coagulated albumin.

Furthermore, the examination of the vomitus may give some conclusions about the gastric motility. If an individual vomits bits of food more than seven hours after a meal, some impairment of motility must exist, for after that interval even a hearty meal should have completely left the stomach. If this late vomiting is very noticeable, or if perhaps a larger amount is vomited than was ingested at the last meal, it indicates a very pronounced motor insufficiency, and is usually associated with *extreme gastric dilatation*. If the vomitus contains particles of food known to have been ingested at some previous meal or a day or two before (seeds of fruit, etc.), such a diagnosis is clinched.

If we can show that the excess of the vomitus over what was ingested at the last meal consists largely of a thin acid liquid containing free hydrochloric acid, we are entitled to assume a *hypersecretion* of gastric juice. The vomiting of an acid liquid without food particles from a supposedly empty stomach is especially characteristic of this condition. In the latter case the secretion of the gastric juice apparently does not depend upon the ingestion of food, but occurs continually (*supersecretion, gastrorrhœa acida*). We must, however, remember that in some of these cases, and perhaps in the majority, the hypersecretion is due entirely to a motor insufficiency—*i. e.*, to the fact that secretion is continually excited in a stomach which never empties itself completely. In contrast to these cases of hypersecretion in the strict sense, there are others characterized as *simple hyperacidity*, in which the vomitus is not abnormally abundant, but is abnormally and intensely acid (due to free hydrochloric acid). The method of determining this is by titrating the filtrate for its acidity. It will be described later. A quantitative comparison with the normal is, however, justifiable only

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when the vomiting occurred one or two hours after the meal, and when longer-retained food portions are not mixed with the vomitus. If a considerable quantity of ingested fluid which need not be digested (water, coffee, etc.) is vomited several hours after consumption, and if the acidity of the fluid does not indicate hypersecretion, it has been assumed that both the motility of the stomach and its power of absorption are affected. For it has been claimed that physiologically the absorption even of large amounts of fluids by the stomach is exceedingly rapid. But v. Mering's recent investigations seem to prove that under physiologic conditions the stomach absorbs hardly any water,

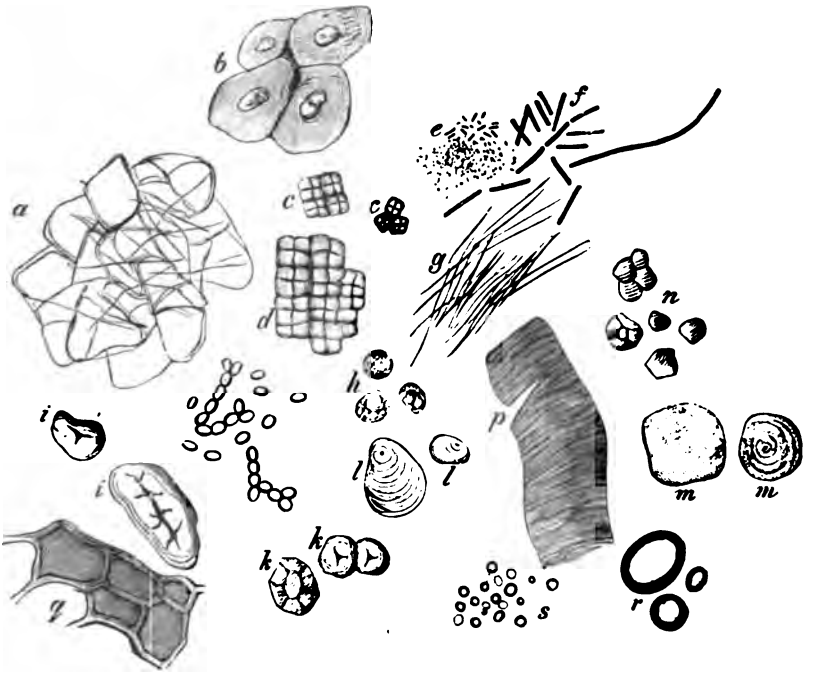

FIG. 141.—Microscopic constituents of vomitus: *a*, Fragment of an apple (crushed); *b*, pavement epithelium; *c*, Sarcina ventriculi (smaller type); *d*, Sarcina ventriculi (larger type); *e*, bacterial clump; *f*, large bacilli, such as are especially suggestive of a gastric carcinoma; *g*, needles of fatty acids; *h*, white blood-corpuscles; *i*, vegetable starch; *k*, corn starch; *l*, potato starch; *m*, white-bread starch; *n*, rice starch; *o*, yeast cells; *p*, muscle fibers; *q*, vegetable tissue; *r*, fat-drops; *s*, milk or cream drops.

so that, unless we deny that his observations upon dogs can apply to healthy man, we must attribute the retention of fluids as due to *deficient motility* alone.

An examination of the vomitus gives us important information about the *antiseptic qualities* of the gastric juice. Decomposition in the gastric contents depends either upon a diminution in the amount of hydrochloric acid contained in the secretion (for the acid acts as an antiseptic) or upon stagnation of the gastric contents—*i. e.*, upon some interference with the motility of the stomach. The vomitus is then apt to be foamy and to smell of some of the volatile fatty acids (buty-

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ric acid, acetic acid, p. 377), or to have some other disagreeable odor; and microscopically we find abundant micro-organisms, especially Sarcina ventriculi, yeast fungi, cocci, and bacilli (Fig. 141). This condition is most pronounced in a dilatation of the stomach due to stenosis, especially from carcinoma of the pylorus. The food stagnates in the stomach as a result of mechanical obstruction, oftentimes for several days. We frequently find in carcinoma of the stomach, and more rarely in gastric dilatation due to other causes, peculiar thin bacilli, sometimes arranged in long threads (Fig. 141). Cocci and bacilli are usually found in larger quantities in gastric juice which is poor in hydrochloric acid; whereas sarcinæ and yeast fungi may flourish in gastric juice rich in hydrochloric acid. We meet with two types of gastrorrhœa acida (supersecretion). In both the stomach always contains material with free hydrochloric acid, and this is apt to be vomited from time to time. In *the milder type* this material is free from micro-organisms. In *the more severe type* the large amount of hydrochloric acid present is no longer capable of keeping the stomach contents aseptic, but micro-organisms grow abundantly, owing to the high degree of motor insufficiency. In this case chiefly yeast fungi and sarcinæ are found in the vomitus, and may be present in abundance even with 30 per cent. HCl.

It must be noted that bread itself contains greater or less numbers of bacteria and yeast cells. Hence, before conclusions are justified, the bacteriologic examination of the gastric contents after the ingestion of bread should first be compared with a similar examination of the bread.

If the vomitus contains *abundant tough, slimy masses,* it indicates a mucous catarrh of the stomach or a diminution in the hydrochloric acid, for under normal conditions the mucus produced by the mucous membrane is in part digested. (Compare later Examination of the Mucous Secretion of the Stomach.) An *admixture of blood* may be found in the vomitus under many different conditions. A slight streak-like admixture of blood in the vomitus is of no diagnostic importance whatsoever, for the violent act of vomiting may produce some slight mechanical lesion of the stomach, esophagus, or pharynx. An abundant admixture of fresh arterial blood or of blood stained dark and coagulated by the acid gastric juice is especially suggestive of ulcer of the stomach. Brown or black material resembling coffee grounds, if granular and intimately mixed with the stomach contents, is suggestive of gastric carcinoma; but the same condition frequently results from hemorrhagic erosions of the gastric mucous membrane, especially if associated with hyperacidity or hypersecretion. We can prove that this appearance is due to the altered blood by microscopic or spectroscopic examination, or by Teichmann's test.[1] (See under Urine, p. 470.)

Pus may be found in the vomitus due to the very rare condition, *suppurative* or *phlegmonous gastritis. Admixture of bile* produces a yellow or greenish discoloration of the vomitus. In a doubtful case we can

[1] The author recommends the modification of the turpentine-guiacum test described upon pages 447 and 471 (Examination of the Feces for Blood).

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apply Gmelin's test for biliary pigment. See Examination of the Urine, p. 472 *et seq.*). Admixture of bile may occur with any type of vomiting, for the contents of the duodenum may be forced into the stomach mechanically by the act of vomiting. It is, however, observed most frequently in vomiting from an empty stomach, perhaps because there is then no counter-pressure from gastric contents to prevent the regurgitation from the duodenum. This is probably the reason that biliary vomiting is so frequently observed in peritonitis. The vomitus of *cerebral meningitis* is less apt to be mixed with bile, because these patients still take considerable nourishment, and the vomiting generally starts with a partially filled stomach—*i. e.*, the vomiting in this disease is both peripheral and cerebral in origin. Green biliary vomitus is, of course, by no means pathognomonic of peritonitis. Biliary vomiting, for evident reasons, accompanies *stenosis of the duodenum.*

Fecal vomiting is a characteristic sign of complete motor insufficiency of the intestines (in peritonitis), or of *intestinal obstruction* in the lower part of the small intestine or in the large intestine. The odor and brownish color of the vomitus are distinctive. Microscopically, it is found to contain large numbers of bacteria. *Cholera nostras* and *cholera asiatica* are characterized by the vomiting of abundant *alkaline rice-water-like* material (exactly like the cholera stools). It usually contains bits of white mucus. In cholera asiatica the comma bacillus (Koch) (see Examination of the Feces) may be found in the vomitus. We are not yet certain about the organism of cholera nostras. The Finkler-Prior comma bacillus, the colon bacillus, various forms of the proteus group and streptococci (see Pathognomonic Bacteria of the Feces) have all been found in the feces, and also in the vomitus of cholera nostras. The occurrence of streptococci in the vomitus has assumed some importance recently in the etiologic diagnosis of other gastro-intestinal infections.

The color, odor, and chemical examination of the vomitus may sometimes aid in tracing the occurrence of stomach disturbance to some sort of poisoning (hydrocyanic acid, alcohol, etc.). In uremia the vomitus often possesses the odor of ammonia, for the urea eliminated in the stomach contents may be converted into carbonate of ammonia while still in the stomach.

Intestinal worms, more especially ascarides, are sometimes found in the vomitus. (See later, Animal Parasites in the Stools.)

It is sometimes difficult to decide whether food particles which the patient vomits soon or some time after ingestion come from the stomach itself, or whether they are only regurgitated from the esophagus (*diverticulum* from *stenosis of the esophagus*). The presence of free hydrochloric acid will generally settle the doubt.

EXAMINATION OF THE POWER OF ABSORPTION OF THE GASTRIC MUCOUS MEMBRANE BY MEANS OF POTASSIC IODID.

Penzoldt and Faber[1] have published a direct method of testing the absorbing power of the gastric mucous membrane. The procedure depends upon the prin-

[1] Penzoldt and Faber, *Berlin. klin. Woch.*, 1882; *Zweifel. Deutsch. Arch. f. klin. Med.*, vol. xxxix.

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ciple that under physiologic conditions potassic iodid is very rapidly absorbed by the gastric mucous membrane and then eliminated immediately in the saliva. The length of time from the ingestion of the potassic iodid until the saliva exhibits a distinct iodin reaction may be considered as a measure of the rapidity of absorption, provided that slight differences in the time of elimination be neglected. The potash is administered in a gelatin capsule filled carefully enough to avoid any particles of the salt sticking to the outside, which would be absorbed by the mouth or esophagus.

To demonstrate the iodin, the patient spits upon the edge of a white plate, a little starch powder is added to the saliva, and then, while it is being stirred carefully, 1 or 2 drops of nitric acid are added. The nitric acid sets iodin free from the potassic iodid in the saliva and produces the well-known violet color (starch iodid). Any excess of acid will oxidize the iodin to iodic acid, which does not give this violet color. The acid should therefore be added drop by drop and stirred. The reaction is less sensitive if the saliva remains any length of time in contact with the starch, because the saliva changes the starch to erythrodextrin and achrodextrin. If these substances unite with the iodin, colorless or only pinkish combinations result; hence the admixture should be made promptly. If a few drops of chloroform are added to the saliva before adding the acid, the chloroform will dissolve the free iodin and produce a rose color.

Experience shows that under normal conditions the first trace of iodin appears in the saliva ten or more minutes after the administration of 0.1 gm. potassic iodid upon an empty stomach. If, however, the stomach is full, or if food is given simultaneously with the drug, the absorption and test will be delayed. Hence the experiment should always be performed either upon an empty stomach or coincidently with a test breakfast. Bauer, in the Bern Clinic, found from a series of experiments that if the drug was taken with an Ewald test breakfast the reaction appeared in the saliva, under normal conditions, in from five to twenty minutes. If retention is suspected, the stomach should, of course, be first washed out. If the reaction is delayed, although the stomach is supposed to be empty, retention is the probable cause, because experience shows that there is rarely much retardation of the reaction in an absolutely empty stomach, even under pathologic conditions.

Of course, this method does not give any information as to the absorption of food substances, for, as a matter of fact, potassic iodid, even under the most unfavorable pathologic conditions, is absorbed with very noticeable ease.

If v. Mering's[1] experiments are confirmed, this method becomes entirely useless. He finds in dogs, and very likely the same is true in men, that potassic iodid is not absorbed at all from the stomach, not even within two or three hours. Hence we must acknowledge that the rapid appearance of the iodin reaction in the saliva depends upon the absorption of the iodid in the intestines, and is in reality only a measure of the gastric motility (see the following):

EXAMINATION OF THE MOTILITY OF THE STOMACH WITHOUT THE USE OF THE STOMACH TUBE.

Palpation (p. 354, *et seq.*), especially eliciting the splashing sound of the stomach, gives us some idea of the gastric motility; examination of the vomitus (p. 357 *et seq.*) reveals still more. Ewald and Sievers and Huber recommended the employment of salol to test the motility. Salol or salicylate of phenol passes through the stomach unchanged, and splits up, only after it reaches the intestine, into salicylic acid and phenol, under the influence of the pancreatic juice and the intestinal bacteria. Salicylic acid, soon after absorption, is eliminated in the urine as salicyluric acid (chlorid of iron produces a violet color when added to the urine; see Examination of the Urine).

Assuming that the salol is always broken up with equal rapidity, this reaction should measure the rapidity with which it reaches the intestines; in other words, the gastric motility. Ewald and Sievers administer the drug with the test break-

[1] *Klin. Jahrb.*, vol. vii, 1899.

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fast; then the patient urinates at regular intervals during the next few hours, and the chlorid of iron test is performed upon each specimen. Normally, the salicyluric reaction occurs within seventy-five minutes. If delayed beyond this period, motor insufficiency may be diagnosed.

This method of testing is liable to a number of errors. The most important one is that we can never be sure whether the movements of the stomach produce a uniform mixture of the salol and stomach contents, so that whether the salol leaves the stomach with the first portion of the food mass or later on must largely depend upon chance. Hence the salol should be intimately mixed with the food, or else taken in small portions (as an emulsion in water) during the meal. The rapidity of splitting up, of absorption, and of elimination is not always a definitely fixed and constant quantity. However, in spite of these sources of error, the method gives quite reliable information regarding the more pronounced disorders. In marked gastric retention the salol may remain for hours in the stomach.[1]

Huber has modified this method by determining the length of time which elapses before all the salicyluric acid is excreted. The reasoning is clear. The moment of the appearance of the salicyluric acid reaction depends principally upon the rapidity with which the stomach passes the first amount of salol into the intestine; but the persistence of the reaction in the urine (assuming rapid splitting up and rapid elimination of the salol in the intestines) is largely due to the fact that there is still some salol not yet split up in the intestines. This is, of course, due to the fact that the stomach has not yet passed on all the salol. Therefore the duration of the salicyluric acid reaction may be a better measure of stomach motility than the period of its first appearance. Huber's investigations have shown that in healthy individuals who have taken 1 gm. of salol at a meal, the salicyluric reaction has always disappeared inside of twenty-six or twenty-seven hours. Motor insufficiency may therefore be assumed if this time is exceeded. With this method the urine need not be tested until about twenty-seven hours after taking the salol. Of course, the urine should be voided a little beforehand—*e. g.*, one-half hour—so that no residual urine remains. If the salicyluric reaction is present twenty-seven hours after the ingestion of the salol, the test should be repeated every three hours till it disappears. The degree of motor insufficiency is in direct proportion to the duration of the reaction.

The author has observed more reliable results from combining Ewald's and Huber's methods, as follows: 1 gm. of salol is taken with the meal, either intimately mixed or in very small quantities, and then the time of appearance and of disappearance of the salicyluric acid in the urine is noted. This combined test will reveal impairment of motility in all sorts of stomach disorders. The appearance of the reaction may be delayed for several hours, and not infrequently it persists for forty hours or more. *Iodipin* has recently been recommended instead of salol. Iodin is set free and absorbed in the intestines and then tested for in the saliva (see p. 361).

Personally, the author is convinced that none of these methods is perfectly trustworthy for estimating the gastric motility. In the first place, we are not sure that the substances employed are intimately mixed with the gastric contents, nor are we certain that they may not be precipitated by the gastric juice and reach the intestines too soon or too late. Again, the results will be very decidedly influenced by the rapidity of the splitting up and of the absorption in the intestines. If v. Mering's idea is confirmed (see p. 362), that potassic iodid is not absorbed at all in the stomach, then Penzoldt and Faber's method for testing the absorption in the stomach can be utilized as a test of its motility (p. 361 *et seq.*).

Further investigations upon the point are necessary.

[1] There is, however, reason to believe that in cases of grave disturbances of motility, when the salol remains for hours in the stomach, some of the drug may also be absorbed by this organ. Part of it is, perhaps, first split up by micro-organisms, which are rarely absent in a stagnant stomach, or by fat-splitting ferments. Such a slow absorption, however, would rarely, if ever, lead us to assume that the motility was normal

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TESTING THE DIGESTION WITH POTASSIC IODID FIBRIN—RUBBER CAPSULE.

Although this method was detailed at length in the previous edition of this book, it has since led to no practically useful results, and so it need only be alluded to.

METHODS OF EXAMINING THE STOMACH WITH THE AID OF THE STOMACH TUBE.

INSTRUMENTS.

Kussmaul was the first to employ the stomach tube for the treatment, and v. Leube the first to use it for the diagnosis, of gastric affections. With its help we cannot only obtain the contents of the stomach at any period of digestion, but we can inflate it with air or fill it with fluid at will. The best tubes are made of rubber, and are quite soft and pliable. The English (Jack's patent[1]) are as good as any. They are provided with a lateral opening and a rounded closed end. They might well be even softer. The surface of the tube must be smooth, the rubber soft but not too collapsible, the lumen large in relation to the entire diameter, and the opening at the lower end as large as possible. The smaller-sized tubes are generally easier to pass and less disagreeable to the patient, but become stopped up easily. In passing the smaller sizes we require the patient's help in swallowing, whereas the larger can be more readily pushed down the esophagus. No. 22 (Jack's patent), with a diameter of about 12 mm., is, the author believes, the most convenient size. With the aid of a small glass tube for connection, and a rubber tube about 2 feet long, the stomach tube is connected to a large funnel (preferably of glass). These three pieces together constitute a siphon apparatus, by means of which we can easily empty and wash out the stomach.

If the stomach contains large solid food particles which clog up the tube again and again, it may be convenient to have some sort of a pump which can be attached in order to suck out these pieces. The same pump is useful for distending the stomach with air in order to estimate its size. Katsch, of Munich, makes a very good one (Fig. 142). The pump in Potain's aspirating set, attached to a large glass bottle (Fig. 143), may be used.

Any syringe may be employed with a stomach tube—*e. g.*, Davidson's syringe, a fountain syringe.

TECHNIC OF INTRODUCING A SOFT STOMACH TUBE.[2]

The patient should be in the sitting posture, with his mouth wide open, and the rounded end of the tube should be pushed back and down

[1] [We have found the Jack tubes very good, but a rather softer and more pliable tube will sometimes cause less gagging, especially upon the first introduction. We refer the reader to *Hemmeter's Diseases of the Stomach*, 2d ed., pp. 114–118, for additional details and hints upon the introduction of the tube.—ED.]

[2] See Examination of the Esophagus.

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over the base of the tongue. If there are no obstructions in the esophagus, we need not lubricate the tube with anything but water. If the patient can be persuaded to relax and breathe quietly and deeply, the tube can be quickly pushed into the stomach. The patient need not

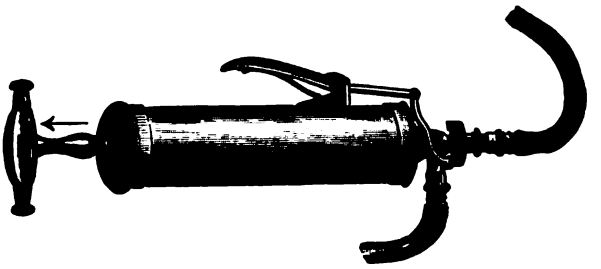

FIG. 142.—Stomach pump.

attempt to swallow if we use the rather larger tubes. It is important for the examiner to reassure him in every possible way, and to explain beforehand the steps of the process and its disagreeable features, so as to avoid frightening the patient during the ordeal itself. As soon as the tube has entered the esophagus but a few centimeters, the complete

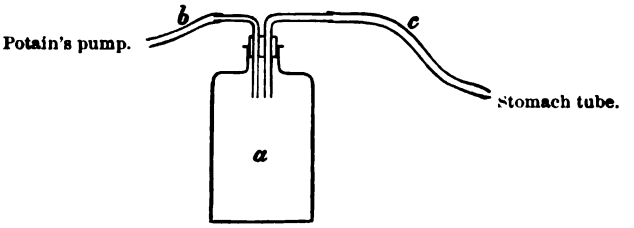

FIG. 143.—Arrangement of Potain's pump used as a stomach pump: *a*, A wide-necked bottle fitted with a rubber stopper, through which are passed two glass tubes bent at right angles; *b*, tube leading to Potain's pump; *c*, tube connecting with the stomach tube.

passage is readily effected. The distance from the teeth to the cardiac orifice in an adult is usually about 40 cm. (15¾ in.). Continued deep breathing, even after the tube has been completely inserted, is very helpful to the patient.

Some patients exhibit so much dyspnea that an inexperienced examiner may think the tube is in the larynx or trachea, although the difficulty in breathing is due only to the psychic influence, perhaps to the pressure of the tube, or to obstruction from involuntary swallowing movements (see above). Sometimes audible whistling respiratory sounds produce the impression that the patient is breathing through the tube (esophageal breathing). (Compare section on Examination of the Esophagus.) As a matter of fact, it is extremely difficult to introduce the tube into the larynx, especially if soft tubes are used, because the larynx closes the instant any foreign body touches its entrance, and because the epiglottis is depressed and the tube pushed into the esophagus by the voluntary or reflex swallowing act at the moment the tip of the tube gets into the pharynx.

If a fit of coughing occurs as the tube is being introduced, it is well to withdraw it and try again. Any marked resistance, marked dyspnea, or much gagging upon the part of the patient usually means that the tube is caught in one

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of the fossæ pyriformes at the side of the larynx, and rolled up over the entrance of the larynx like a spiral. Then, if the examiner attempts to push it farther, the tube usually snaps back suddenly into the mouth. To avoid this difficulty we should endeavor to introduce the tube exactly in the median line.

In some very difficult cases it may be necessary to paint the pharynx with a 4 per cent. solution of cocain. (See section on Laryngoscopy and Tracheoscopy.)

INDICATIONS AND CONTRA-INDICATIONS TO THE INTRODUCTION OF THE STOMACH TUBE FOR DIAGNOSTIC PURPOSES.

Indications.—Every slight gastric complaint does not indicate the employment of a stomach tube, for it causes the patient too much discomfort; but its use is indicated where an absolute diagnosis seems impossible at the onset, or where the treatment employed has failed and the physician appreciates the necessity of revising the diagnosis by more exact methods.

Contra-indications.—The stomach tube is contra-indicated in all grave disorders of respiration or circulation, in cardiac failure, cardiac neurosis—*e. g.*, when the excitement accompanying the passing of the tube may in itself lead to danger—in aortic aneurisms (on account of the danger of perforation), in patients liable to cerebral hemorrhage, and in those who have recently had hemorrhage from the lungs or the stomach.

THE VARIOUS STEPS IN THE EXAMINATION.

The best time to introduce the tube is in the early morning, upon a fasting stomach, the patient having ingested the previous evening a rather abundant mixed meal.

We first cleanse the fasting stomach, extracting any residue or secretion which it may contain. After the tube has been introduced the patient is instructed to strain or cough gently, to aid in expelling any stomach contents. If nothing appears we employ gentle suction with a pump or bulb. If this obtains nothing we pour in a little lukewarm water at first, and later a pint at a time, and then empty the stomach again by depressing the funnel below the level of the stomach. If employing a funnel, the examiner must see that it is kept filled, so that the tube does not become empty, and hence stop the siphonage. In case any particles stop up the tube, it may be possible to force them back into the stomach by elevating the funnel again. The stomach is washed out in this way till the fluid recovered comes clear. If any material was removed before beginning the washing, it should be saved for examination (see p. 368). If this was not possible, then whatever was obtained after the first dilution.

After emptying and cleansing the fasting stomach, the next procedure is to determine its size and position by inflating it with air.[1] This can be accomplished quite conveniently with the aid of a Davidson's syringe. The patient should be in the dorsal decubitus, with the lower chest and abdomen exposed to view. We must inflate cautiously

[1] This will be superfluous if we have already determined the measure accurately by palpation and by percussion.

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and stop whenever any pain is produced. After it is moderately distended, inspection will generally disclose the position and shape of the organ; but to avoid confusing a dilated stomach with a gastroptosis we must determine the lesser, as well as the greater, curvature. (Loop-shaped stomach may also occur.)

The "hour-glass" stomach is very rare. It consists of two cavities connected by a constricted passage, and under certain conditions may be recognized by inspection of the distended stomach.[1]

When inspection alone fails to determine the shape and size of the stomach (*e. g.*, on account of abnormally thick walls), palpation and percussion of the organ after it has been inflated with gas will usually settle the shape and, at least, the position of the greater curvature. Luschka has estimated that a normal stomach contains between 1½ and 2 liters of gas; but these figures were determined upon cadavers, and are subject to considerable variations according to the class of the individual and the type of his nourishment.[2] A large stomach is not necessarily diseased. An increase in size becomes pathologic only when motor insufficiency exists and food remains. If continued insufflation produces a diffuse distention of the abdomen instead of a localized bulging in the epigastrium, we are justified in diagnosing an insufficiency of the pylorus (Ebstein).

When distending the stomach we should note if any tumors are present, and also any changes in the position of the tumors. As soon as all of these points have been determined, the air is allowed to escape, so as not to cause the patient any unnecessary discomfort. This is accomplished by pressing or kneading over the upper abdomen with the tube *in situ*. The tube should then be quickly pulled out.

The fact that a patient's distention can be instantly relieved (p. 354) is one of the chief advantages of employing the tube instead of an effervescing powder. This method of gastric inflation furnishes such accurate results in regard to the size and position of the stomach that it seems very questionable if "gastrodiaphany" can supplant it for practical purposes. The latter process consists in introducing a tube, with a small electric lamp at the tip, into the stomach, and thus reflecting light through the stomach. Röntgen rays seem quite as unnecessary and impractical for the purpose. Tumors which cannot be appreciated by palpation will probably escape both these methods.

Inflation may be supplemented by palpation of the soft tube along the greater curvature of a relaxed stomach. Its tip can often be felt especially plainly, and the results of inflation thereby confirmed. Such palpation should be practised very cautiously.

TESTING THE GASTRIC FUNCTIONS BY MEANS OF A TEST BREAKFAST.

The principle involved follows: The patient takes a definitely determined meal upon a fasting stomach—*i. e.*, after the stomach has been emptied artificially or the first thing in the morning. We then draw conclusions as to the functions of the stomach from the amount and from the chemical character of the contents removed by the tube after the lapse of a certain time.

[1] *Virchow's Archiv*, 1895, vol. cxl., No. 3, p. 459.

[2] [The German figures are generally larger than the American.—Ed.]

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Various test breakfasts have been advocated. For some purposes one kind is better; for others, another kind. If, for instance, we wish to test the behavior of a certain food in the stomach, then that food should be selected for a test breakfast. Again, if we wish to determine the behavior of the stomach under an ordinary mixed meal, we select such a meal for our test—*e. g.*, Riegel's test meal consists of a plate of mutton broth, a beefsteak (150 to 200 gm.), potato purée (50 gm.), and a roll. Ewald-Boas' test breakfast is the most serviceable for general use. It is convenient and uniform. It consists of 1 roll (about 35 gm.), or the same amount of white bread, and 2 cups (400 c.c.) of water or weak tea without milk or sugar. The patient should chew the bread very thoroughly. More useful and accurate information will be obtained from the "Sahli-Seiler's Universal Test Meal" (see p. 397 *et seq.*). The test meal is allowed to remain in the stomach a definite length of time until acted upon by the gastric secretion, and then withdrawn through the tube. Riegel's meal is ordinarily withdrawn at the end of four hours; the Ewald-Boas and the "Universal" at the end of an hour. If no contents can be obtained after the usual interval, another meal should be ingested, and expressed after a shorter interval (better upon the succeeding day).

Ewald and Boas' method of expression is the most convenient way of obtaining the stomach contents. The patient is instructed to cough and strain down. The food is usually expelled better if the patient is in the right or left lateral, rather than in the sitting, posture. If the tube becomes clogged we can generally free it by forcing a little air through, which is more convenient than diluting the contents by adding more water. When but little is expelled it is advisable to employ pressure, so as to be sure that the stomach is empty, and thus to correctly estimate the amount of the meal contained in the stomach. (See p. 401 in regard to the procedure of the "residue estimation." This is only available when the universal meal is employed. (See p. 370 *et seq.* and p. 397 *et seq.* for the examination of the expressed meal.)

EXAMINATION OF THE CONTENTS OF A FASTING STOMACH.

The materials removed from a fasting stomach are to be examined in the same way as the vomitus so far as their microscopic and macroscopic peculiarities are concerned. For the chemical examination the same rules apply as for testing the expressed breakfast. (See p. 370 *et seq.*). If the fasting stomach contains remnants of the previous day's food, there must be a considerable degree of motor insufficiency, because a normal stomach would be empty within seven to eight hours, after even quite an abundant meal, and in any event should be empty in the morning.

Cranberry or Currant Test.—If we have not the opportunity to test the fasting stomach for determining motor insufficiency, nearly the same result can be accomplished, according to Ewald and Strauss, by giving the patient, at his evening meal, a tablespoonful of cranberry or currant preserve. We can decide upon a certain degree of motor insufficiency of the stomach if the expressed meal the

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next day contains any of the fruit or seeds. The advantage of the test is that it does not make any difference whether or not the patient has commenced upon his ordinary nourishment.

If we can obtain from the fasting stomach a considerable quantity of fluid containing free hydrochloric acid without any food remnants, hypersecretion may be diagnosed, because, if empty, the stomach should not secrete hydrochloric acid. The irritation of the tube or of the saliva swallowed may suffice to excite a mild secretion. Hence a small amount of such fluid is not sufficiently characteristic. The amount of acid fluid which can be withdrawn from a fasting stomach and still remain within the normal limits varies considerably, according to the individual authority. Ordinarily not more than 10 c.c. are found. Cases have been reported, however, where 20 to 50 c.c. have been recovered without any noticeable disorder. Boas considers 100 c.c. or over pathologic for a fasting stomach. Evidently there are no very sharp limits between a physiologic and a pathologic amount of secretion. To differentiate between primary hypersecretion and hypersecretion secondary to stagnation, compare p. 390, Note 2. Normal gastric juice from a fasting stomach is thin, not at all slimy, with a specific gravity of only 1004 to 1005. It thus differs from gastric juice which contains the products of digestion, such as is obtained after a test breakfast or when the food is retained (compare p. 371).

If much lactic acid is present we know that it is due to bacteriologic fermentation and, hence, indicates a diminished production of hydrochloric acid, for normally the hydrochloric secretion of gastric juice prevents lactic acid fermentation.

Not infrequently a little yellowish or greenish bile-containing fluid, often alkaline, is expressed from the fasting stomach. It consists of duodenal juice which entered the stomach from the gagging and vomiting while passing the tube. But if repeated examinations show that such fluid is always present, even if there have been no vomiting efforts, or, again, if it is constantly vomited in some chronic disease, there must be some stenosis of the duodenum below the ductus choledochus (Boas). This statement has recently been corroborated by various authors.[1] Gmelin's test will, of course, prove that the color is really due to bile (see p. 472 *et seq.*). The reaction of the stomach contents in such cases may be alkaline if the stomach no longer secretes enough acid to produce a neutral or acid reaction. If, on the other hand, the stomach secretes an abundance of hydrochloric acid and the fluid is stagnant for a considerable period, then the reaction may remain acid. In case the reaction is neutral or alkaline, we can prove that the fluid comes from the duodenum, because it will digest in an alkaline solution. For this purpose the fluid is placed with a few fibrin flakes colored with Magdala-red in an incubator, the reaction having been rendered (if necessary) distinctly alkaline by the addition of 1 per cent. soda solution. The fibrin flakes will be digested by the trypsin contained in the fluid, and the fluid will be colored red. If the fluid is of acid reaction, it is usually

[1] Compare *Deutsch. med. Woch.*, 1896, No. 23, "Herz, Über Duodenalstenosen."

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impossible to demonstrate the presence of trypsin, because in acid solutions trypsin is rapidly destroyed by the pepsin of the gastric juice.

For further methods of demonstrating the action of trypsin and for recognizing intestinal juices in general, see later, in section upon Boas' Method of Obtaining the Intestinal Juice (p. 421 *et seq.*).

Gastric carcinomata which cannot be palpated may sometimes be diagnosed by washing out the empty stomach and finding macroscopic and microscopic constituents of the growth in the wash water. The best method is to wash out the stomach thoroughly the evening before, and then to repeat the lavage the next morning without allowing the patient to take anything overnight. Sometimes a little undiluted fluid can be expressed through the tube for microscopic examination before the morning lavage. Characteristic bits or cells can thus often be obtained for examination, especially if the carcinoma is considerably ulcerated. The fluid should, of course, be centrifugated, and only the sediment examined microscopically. (Compare Urine Analysis, p. 553 *et seq.*). The following is a very illustrative case: Clinically it presented the features of pernicious anemia, and *intra vitam* the diagnosis of carcinoma of the stomach could not be made with any certainty. No tumor could be felt. Free hydrochloric acid was absent, to be sure, but the stomach was of normal size and emptied itself even abnormally rapidly. As is commonly known, anacidity with hypermotility of the stomach occurs in a very large number of so-called pernicious anemias. Autopsy showed a very large carcinoma of the lesser curvature. It could not have been palpated, because it was entirely hidden under the liver. It had not produced any stenosis of the pylorus. The method above recommended might easily have permitted a differential diagnosis from pernicious anemia, because the tumor was soft, friable, and ulcerated.

EXAMINATION OF THE GASTRIC FUNCTIONS BY MEANS OF THE ORDINARY (EWALD-BOAS) TEST BREAKFAST.

The expressed breakfast is, first of all, filtered. In what follows the filtrate is called the "gastric juice."

APPEARANCE AND AMOUNT OF THE EXPRESSED MATERIAL; SPECIFIC GRAVITY OF THE GASTRIC JUICE; JUDGMENT OF THE GASTRIC MOTILITY.

The conclusions to be drawn from the *appearance* of the test breakfast in relation to the digesting function of the stomach are the same as were mentioned upon p. 357 *et seq.* with reference to the appearance of vomited gastric juice (*cf.* especially the peculiarity of the particles of bread).

The *amount* of the expelled material is of special diagnostic importance. The Ewald-Boas test meal does not usually furnish physiologically more than 30 to 70 c.c. of filtrate. But too much importance should not be given to these figures, because many variations occur and much larger amounts are within physiologic limits. A filtrate to the amount of 200 to 300 c.c. makes motor insufficiency or hypersecretion probable. If there is a large volume of fluid and it is strongly acid from free hydrochloric acid, hypersecretion is very probable. Hypersecretion is, however, sometimes secondary to motor insufficiency, and is due to the continual stimulation of residual contents. If the expressed contents contain a large proportion of solid material, motor insufficiency is

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suggested. This diagnosis is confirmed if the fasting stomach contains particles of food. If the organ, eight hours after an ordinary meal, contains any remains of the meal, we have corroborative evidence of motor insufficiency. To determine the existence of true hypersecretion (independent of motor insufficiency), we should administer a roll with but very little of any fluid. Then if we can express a goodly amount of acid fluid, hypersecretion is definitely determined. Hypermotility may very properly be assumed if the volume of expressed material is decidedly diminished, but only when we have become convinced after lavage that the stomach is thoroughly emptied.

The specific gravity of the filtrate varies normally between 1012 and 1020. In hypersecretion and in sluggishness of the digestive mechanism it is diminished either by dilution or by deficient formation of soluble digestive products. Pure gastric juice without digestive products has (see p. 369) a specific gravity of only 1004 to 1005.

EXAMINATION OF STARCH DIGESTION.

As is well known, the salivary enzyme changes starch first into soluble starch (amylum to amidulin or amylodextrin). Iodin still colors the latter violet. The next change is to *erythrodextrin;* but this gives a reddish to a mahogany-brown color with iodin. Finally, *achroodextrin* and *maltose* are formed (not, as was formerly believed, dextrose). These last two products no longer produce characteristic colors.

In testing the gastric contents for the character of starch digestion, the point to determine is whether, besides starch, which always exists in considerable quantity, even in the upper portion of the intestines, there are any of the first products of starch digestion. With a solution of iodin this can be easily accomplished. Achroödextrin produces no color, but it has a greater affinity for iodin than has starch itself. Consequently, when slight traces of iodin are added to a uniform mixture of starch and achroödextrin, the iodin combines with the achroodextrin before it affects the starch. Therefore, if we add traces of iodin to a mixture of starch and achroödextrin, no color will result until an excess of iodin brings out the violet. A drop of a very dilute (wine yellow) Lugol's iodin solution[1] upon a glass rod is added to the residue on the filter paper, or even to the filtrate (because the latter always contains soluble starch). If achroödextrin is present, no color will appear until an excess of iodin is added. If, on the other hand, no starch digestion has occurred, the slightest trace of iodin will produce a violet color.

If a large volume of gastric contents is at our disposal, the test may be performed as follows: Lugol's solution (0.1 gm. iodin, 0.2 gm. potassium iodid, 200 c.c. water) is added drop by drop to the filtrate. The amount added before a violet color appears will furnish an approximately accurate quantitative measure of the degree of starch digestion. The latter, of course, depends not only upon the rapidity of enzyme

[1] A few drops of tincture of iodin added to a 1 per cent. solution of potassic iodid.

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action, but also upon the degree of the absorption of the product formed.

Starch digestion is a function of the salivary enzyme. Its action is apparent partly in the mouth and partly in the stomach for a short time after the meal. Hence, starch digestion would naturally seem of little importance in the diagnosis of stomach diseases. But this is not really so, because an increased acidity will interrupt starch digestion very quickly. On the contrary, with hypoacidity starch digestion progresses very favorably and completely. Hence, the degree of starch digestion serves as a rough test of the hydrochloric acid secretion. Boas claims that a gastric fluid containing 0.15 per cent. hydrochloric acid will arrest starch digestion. Nevertheless, even with normal gastric digestion achroödextrin is present in the Ewald breakfast one hour after its ingestion. It was, of course, produced before enough acid had been secreted to reach the above degree of concentration. Achroödextrin is often absent in hyperacidity. Lactic acid does not impede starch digestion until it is more concentrated than hydrochloric acid; so that starches are frequently well digested in the gastric contents from a passively congested stomach (Stauungsmagen) in case it contains lactic, instead of free hydrochloric, acid.

QUALITATIVE EXAMINATION OF THE FILTERED GASTRIC JUICE FOR ACIDS.

The reaction of the filtered gastric juice is first of all tested with litmus paper. If the reaction is acid (as is almost universally the case), we get no information about the presence of hydrochloric acid, and especially of free acid, in the gastric juice, since other salts, particularly the acid phosphates, also turn blue litmus red.

To determine whether the acid reaction is due to free acid or only to acid salt we employ Congo-red, in the form of commercial Congo-red paper. If the gastric juice turns a strip of this paper blue, some free acid must be present; either free hydrochloric or some organic acid.[1]

To decide whether the acid is organic or free hydrochloric we employ various qualitative tests. In the following we describe only those tests which in our experience have proved to be the best.

Tests for Free Hydrochloric Acid.—The tests for free hydrochloric acid which are of clinical value all depend upon color reactions.

Methyl-violet Reaction.—A pale-violet solution is made by adding considerable water to one drop of a moderately concentrated aqueous or alcoholic stock solution of methyl-violet in a test tube. This solution is divided into equal parts. To one a little of the filtered gastric juice is added; to the second, an equal quantity of water. If free hydrochloric acid is present, the methyl-violet in the first changes to a beautiful blue. The second half is used for a control. Even a very slight

[1] If the paper is colored intensely blue, an expert would nearly always decide that it was due to free HCl, because organic acids—in the concentrations in which they are found in the gastric juice—always produce lighter shades.

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amount of free hydrochloric acid in the gastric juice will produce this change from violet to blue.

Tropæolin OO Reaction.—Tropæolin OO is a yellow pigment. Even a minute trace of free hydrochloric will turn a solution of this substance red or reddish brown. It is better not to employ too concentrated a solution. The best is a pale-orange solution, prepared freshly by diluting with water a concentrated alcoholic stock solution. If the test is made in the same way as with methyl-violet (see above), the reaction is not as sensitive. Boas increases the delicacy of the reaction by the following modification: Two or three drops of the concentrated alcoholic tropæolin OO solution are dropped into a porcelain dish, and then distributed in an irregular smear to the edges by tilting the dish. The same quantity of gastric juice is added and mixed with the tropæolin. The dish is then gently warmed over a small flame or over a water bath, and if free hydrochloric acid is present, beautiful violet to blue stripes form.

Phloroglucin-vanillin Reaction (Günzburg's Reagent).—The reagent consists of phloroglucin 2 gm., vanillin 1 gm., alcohol 30 gm. One or two drops of this solution are placed in a porcelain dish and mixed with the same amount of gastric juice. The dish is then gently warmed over a small flame. If free hydrochloric acid is present, the drying margins of the mixture will develop a beautiful carmin-red color. If absent, the solution dries up, leaving a brown or yellow color. The reaction becomes a little more sensitive in doubtful cases if we employ rather more gastric juice than reagent.

Value of These Tests for Hydrochloric Acid.—An objection to two of these tests, methyl-violet and tropæolin, is that free organic (especially lactic) acid will produce the same color. But, as a matter of fact, organic acids are never present in sufficient concentration in the stomach to produce these reactions. Günzburg's reaction, however, admits of only one interpretation—the presence of free hydrochloric acid. It not only admits of but one meaning, but also is the most sensitive of all the tests.

Another objection to these tests cannot be disposed of so easily. They may be negative and still free hydrochloric acid be present, but "masked" by other substances—*e. g.*, proteids or peptones. We can easily prove this experimentally by adding a little neutral commercial peptone (this consists chiefly of proteoses) or common salt to a solution of methyl-violet which has been turned blue by a trace of hydrochloric acid. The violet color immediately returns, as if there were no free hydrochloric acid. The same objection applies to tropæolin and to phloroglucin-vanillin; in fact, to all the tests for free hydrochloric acid which are known. But this is no reason against employing these color reactions clinically. The reason the reaction no longer occurs is because in such a mixture of free hydrochloric acid and proteids and peptones the hydrochloric acid is no longer free enough to act upon the coloring matter (Riegel, Boas). The proteids, as is well known, form combinations with acids, just like the typical acid albumin. These combinations

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still react acid to litmus and phenolphthalein, but the affinity of the acid is just as loosely held as in the so-called acid salts. Hence these color tests react negatively when considerable quantities of proteids or peptones are present because a positive reaction appears only in the presence of perfectly free acids. From a clinical point of view, this fact should really be considered advantageous, because it is of no diagnostic importance to learn that the gastric mucous membrane has preserved traces of its acid-secreting power. What we want to determine is whether, after saturating the proteids introduced with the test breakfast, there still remains enough perfectly free acid to continue the digestion. Experience has proved that one hour after the ingestion of an Ewald's test breakfast there should still be free HCl in the mixture. The more recent methods, which try to demonstrate roughly the hydrochloric acid which is combined with proteid, as well as the free acid, are of no diagnostic value except for quantitative purposes. The great difference between a gastric juice which reacts to the color tests for free hydrochloric acid and one which does not would no longer be striking and useful if we should select tests to demonstrate quantitatively the amount of hydrochloric acid combined with the proteid. Experiments with artificial gastric juice prove the same thing; if the juice contains free hydrochloric acid, it is effective in digestion, otherwise not. This would seem to show that for the artificial digestion of proteids only that portion of the hydrochloric acid is of use which is not already combined with the proteids contained in the stomach contents, and this portion still reacts to color reagents. Such an excess of acid should be present normally, and it must be of diagnostic importance to determine whether this is the case or not. Therefore the so-called "masking" of the color reaction by proteids is not a disadvantage, but, on the contrary, a very marked diagnostic advantage. The "masking" of the reaction by salts (the cause of which is not known) has no practical importance in gastric diagnosis, because with the usual test meal the gastric juice never contains salts in sufficient concentration to hinder the reaction.

Ewald quotes the following figures to show the degree of sensitiveness of the three reagents:

Reaction positive when there is present:

	Hydrochloric acid. Per cent.	Lactic acid. Per cent.	Butyric acid. Per cent.
Methyl-violet	0.24	4	——
Tropæolin OO	0.30	2.4–3	5– 6
Phloroglucin-vanillin	0.05	——	5–10

Tests for Lactic Acid. — *Uffelmann's reagent* is commonly employed to test the gastric juice for free lactic acid. It consists of a mixture of 20 c.c. of a 1 per cent. carbolic acid solution, with 1 drop of dilute ferric chlorid. This solution is diluted until the color becomes a transparent amethyst blue. The reagent should be freshly prepared before each test, since the violet color disappears after a short time. This is a very delicate reagent: even traces of lactic acid change it

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to a beautiful canary yellow or greenish yellow. Mere decoloration without the formation of any pronounced yellow shade does not suggest lactic acid, because proteids, salts, and hydrochloric acid might produce such a result. Uffelmann's reaction may also be performed by adding to the fluid to be tested 1 to 2 drops of a very weak (5 per cent.) solution of ferric chlorid (without carbolic acid). If the fluid contains lactic acid, it will become distinctly yellow to green. In a doubtful case the same amount of ferric chlorid should be added to water for comparison. Uffelmann's reaction is not quite reliable, because certain sugars, peptones, alcohol, tartaric acid, citric acid, oxalic acid, and various other substances produce a similar color. Another objection is, that the reaction may be prevented by the presence of phosphates and a considerable excess of hydrochloric acid. The yellowish tint of the stomach contents, and sometimes the turbidity due to the addition of the ferric chlorid, may also affect the reaction. Greater accuracy will result if we isolate the lactic acid by extracting the gastric juice with ether, removing the ether by evaporation, and then perform the test upon the ether residue dissolved in water. Uffelmann's test has been very appropriately modified by H. Strauss.[1] By this method all sources of error are avoided and a quantitative result obtained.

Strauss' Method of Determining Lactic Acid.—He extracts the gastric contents with ether, but does not evaporate the ether. He shakes the ethereal extract with a dilute solution of ferric chlorid, according to Fleischer's method. If the watery layer shows a greenish coloration the reaction is positive. By employing definite quantities of reagent and solution the intensity of the coloration can be made to furnish an approximate estimate of the amount of lactic acid. The procedure is as follows: 5 c.c. of gastric juice are put into a separatory funnel (Fig. 144) having two division marks, one corresponding to a volume of 5 c.c., the other to that of 25 c.c.; ether is now added to the 25 c.c. mark. The solutions are then shaken vigorously. After the mixture has settled into layers, the gastric juice at the bottom is drawn off by means of the stopcock. Distilled water is then added until the liquid rises again to the 25 c.c. mark. Then 2 drops of a dilute solution of ferric chlorid (containing 1 part ferric chlorid to 9 parts of distilled water) are added and the mixture again vigorously shaken. If more than 1 per cent. of lactic acid is present, the layer of water below is colored an intense greenish yellow. Smaller amounts do not produce any distinct color change. Strauss' method eliminates all the sources of error of the ordinary Uffelmann's reaction (above mentioned). None of the substances which produce a similar reaction (peptones and carbohydrates), nor substances which mask the reaction (hydrochloric acid and phosphates) will be taken up by the ether in any appreciable quantities. The ether employed should be free from alcohol. This method suffices for all practical purposes. It should be remembered that Strauss' test may be negative if the lactic acid present is completely combined with the proteids of the gastric juice. The acid albuminate of lactic acid does not then give up lactic acid to the ether. In such a case, which can occur only when the gastric juice does not react for free

FIG. 144.—Separatory funnel.

[1] *Berlin. klin. Woch.*, 1895, No. 37.

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acids, it is advisable to repeat the shaking after hydrochloric acid has been added to the gastric juice up to a beginning of the Congo-red reaction.

Boas' Method for Detecting Lactic Acid.—Boas'[1] method depends upon the fact that oxidizing agents change lactic acid into aldehyd, and that the aldehyd can be easily distilled off and transformed into iodoform by adding some alkaline iodin solution, very much as acetone in Lieben's reaction is transformed into iodoform. (See Urine Analysis, p. 495.) The iodoform is easily recognized by its characteristic crystalline form (compare *ibid.*) and by its peculiar odor. By oxidation carbohydrates are also changed to aldehyd; hence the test should be performed with an ethereal extract of the gastric contents, which will not contain any carbohydrate. The technic of Boas' method is as follows: 10 to 20 c.c. of the filtrate from the stomach contents are evaporated in a small dish over a water bath to a syrupy consistence. If free acid is absent (Congo test) this can be done directly; but if present the lactic acid should first be combined and its volatilization prevented by the addition of a saturated solution of barium carbonate. A few drops of phosphoric acid are then added to the syrupy fluid to set the lactic acid free again, and it is boiled just once to remove the carbon dioxid; then it is allowed to cool, and finally extracted with 100 c.c. of ether free from alcohol.[2] After digesting for a half-hour the clear layer of ether is poured off, the ether evaporated, the residue extracted with 45 c.c. of water, carefully shaken and filtered, and 5 c.c. of sulphuric acid and a little powdered manganese dioxid are added to the filtrate and distilled into the receiving vessel, which contains 5 to 10 c.c. of an alkaline solution of iodin.[3] If aldehyd passes through into the receiving vessel a cloudiness is produced. This consists of iodoform crystals, and the characteristic odor of iodoform is noticed.

Martius' experiments (see p. 388 *et seq.*), contrary to the old idea, prove that the formation of lactic acid is not a part of normal gastric digestion, but is rather due to bacterial fermentations, which are normally prevented by the presence of the hydrochloric acid of the gastric juice. The occurrence of lactic acid in the expressed meals, as demonstrated by Strauss' method, must therefore be considered a pathologic phenomenon. This points either to a stagnation of the gastric contents or to the absence or diminution in the amount of the free hydrochloric acid, or to both of these factors, for only when they occur together is opportunity furnished for abundant lactic acid fermentation. Hence a large quantity of lactic acid is found with *motor insufficiency of the stomach*, more particularly from a stenosis of the pylorus, when associated with diminished hydrochloric acid production. As experience shows, this is generally due to carcinoma of the pylorus; hence recently a certain amount of significance has justly been attached to the demonstration of lactic acid in the stomach contents as suggesting the diagnosis of carcinoma of the stomach. But the opinion originally advanced by Boas, that the presence of lactic acid in the gastric juice was pathognomonic of this lesion, has not been confirmed. *Benign stenosis* and *gastric insufficiencies*, when combined with deficient secretion of hydrochloric acid, have repeatedly furnished a positive test to lactic acid.

[1] *Deutsch. med. Woch.*, 1893, No. 39.

[2] Because alcohol when oxidized becomes aldehyd.

[3] Prepared by mixing equal parts of a solution of 56 gm. of dry potassium hydroxid in 1000 gm. of distilled water and of a $\frac{N}{10}$ iodin solution. (Compare text-books on chemistry.)

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One objection to all the lactic acid tests which demonstrate lactic acid fermentation in the stomach is, that ordinary food, and in particular Ewald's test breakfast, always contains preformed lactic acid. Boas, for this reason, substituted a thin oatmeal porridge for the ordinary test meal, there being no lactic acid in oatmeal. However, this precaution seems really unnecessary, considering the slight amount of preformed lactic acid which is contained in the ordinary test breakfast, if diagnostic importance should be attached only to a very pronounced lactic acid reaction. The advantage of Strauss' method is, that while it estimates fairly accurately the amount of lactic acid, it is not sufficiently sensitive to give a positive reaction from the lactic acid in an ordinary roll made with water (not milk), without lactic acid fermentation in the stomach. This is a marked advantage over Boas' test, which, on account of its sensitiveness, is only of practical value when employed as a quantitative method (see p. 386).

Detection of Volatile Fatty Acids.—If the volatile fatty acids—*butyric*, *acetic*, or *valerianic*—are present to any extent, they are easily recognized by their characteristic odor. More accurate tests are too complicated and hardly practical. Traces of butyric acid occur normally after the ingestion of foods containing butter. Its presence is to be attributed to the action of the fat-splitting ferment of the gastric juice, recently more exactly studied by Volhard. The two other volatile fatty acids, like lactic acid, arise only as the result of fermentation and stagnation of the gastric contents, although traces do, of course, occur from what is contained in the various foodstuffs.

QUANTITATIVE TESTS FOR ACIDS.

The quantitative analysis of the gastric juice is peculiarly difficult because organic acids, and especially acid salts, may be present as well as hydrochloric acid, and because we must also differentiate between free hydrochloric acid and that which is combined loosely with the proteids, as we have already explained in connection with the qualitative hydrochloric acid tests. To make clear the complexity of the condition, we may arrange in the following table the acid-reacting components of the gastric juice—*i. e.*, those turning blue litmus paper red—in one group, and the chlorin-containing constituents, which we must consider in testing for hydrochloric acid, in another:

1. Acid-reacting constituents of the gastric juice.
   (*a*) Free, and combined with proteids, hydrochloric acid.
   (*b*) Organic acids.
   (*c*) Acid salts (acid phosphate).
2. Chlorin-containing constituents of the gastric juice.
   (*d*) Free hydrochloric acid (producing color reactions).
   (*e*) Hydrochloric acid which is combined—*i. e.*, with proteids—producing no color reaction, but reacting to litmus and phenolphthalein acid. (This is usually called combined hydrochloric acid).
   (*f*) Chlorids, neither reacting acid nor producing any color reaction.

Titration of the Total Acidity of the Gastric Juice.—In

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order to determine the total acidity, we first of all titrate the gastric juice with a $\frac{N}{10}$ sodium hydroxid solution—*i. e.*, we determine the number of cubic centimeters of the sodium hydroxid solution which is necessary to neutralize a definite volume of gastric contents. The technic is as follows: As is well known, a normal sodium hydroxid solution is one which contains in each liter as many grams of the substance in question as its equivalent weight. A decinormal ($\frac{N}{10}$) solution is made from this by diluting 10 times. According to the definition, equal volumes of normal solutions neutralize each other; 1 c.c. of a $\frac{N}{10}$ NaOH solution neutralizes exactly 0.1 c.c. of a N—HCl, etc. In preparing a normal sodium hydroxid solution, it is best to start with a normal oxalic acid solution: 63 gm. of a well-crystallized and chemically pure oxalic acid are dissolved in distilled water and the volume made up to exactly 1 liter. Now, a normal sodium hydroxid solution will require, in order to neutralize a given volume of the normal oxalic acid solution, exactly the same volume. A few drops of alcoholic phenolphthalein solution are added as an indicator to 10 c.c. of normal oxalic acid solution. Then to this the approximately normal sodium hydroxid solution[1] is added from a buret until the acid is neutralized—*i. e.*, until the mixture takes on a permanent reddish color. If the normal NaOH solution is correct, it will require exactly 10 c.c. Generally speaking, less is needed. For instance, if we employed 9.5 c.c. of the $\frac{N}{10}$ NaOH for neutralization, we must add 0.5 c.c. of water to each 9.5 c.c. of the solution. Then 10 c.c. of the normal NaOH solution will correspond to exactly 10 c.c. of the normal oxalic acid solution. From this we can easily estimate how much water to add to the 1000 c.c. of solution—*i. e.*, $(\frac{1000}{9.5} \times 0.5)$. Normal sodium hydroxid solution is too strong to use for the titration of the gastric juice; hence we prepare $\frac{N}{10}$ by diluting the normal 10 times. We fill a graduated buret, provided with a stopcock, with this $\frac{N}{10}$ sodium hydroxid solution. We measure 10 c.c. of the filtered gastric juice[2] into a porcelain dish (or into a beaker if we have a white background) and add a few drops of alcoholic phenolphthalein solution as an indicator.

While stirring the mixture continually we add enough of the $\frac{N}{10}$ NaOH solution to make the red color persistent. The amount of the $\frac{N}{10}$ solution employed represents the acidity of the gastric juice. It may be expressed in two ways—either with reference to some fixed acid, or in so-called acidity percentages (Jaworski). The acidity of the gastric juice is perhaps most frequently expressed in terms of HCl. For this purpose we must remember that, according to the definition, equal volumes of normal solutions will react—*i. e.*, neutralize each other. Therefore 1 c.c. of normal sodium hydroxid solution corresponds (for neutralization) to exactly 1 c.c. of normal hydrochloric acid solution. Normal HCl

[1] This is made by dissolving about 42 gm. of pure sodium hydroxid (in sticks) in 1 liter of distilled water.

[2] Martius has showed that we really should employ the unfiltered gastric juice to prevent errors in the quantitative acid tests. This is because the solid material contains a larger proportion of the acid. For practical purposes, however, titration of the filtrate will suffice.

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contains to a liter 1 equivalent—*i. e.*, as the equivalent weight of HCl is 36.5, 1 c.c. of normal HCl acid contains 0.0365 gm. of hydrochloric acid. This amount of hydrochloric acid corresponds to 1 c.c. of normal NaOH solution; 1 c.c. of $\frac{N}{10}$ NaOH therefore corresponds to 0.00365 c.c. of HCl. From this the acidity as expressed in hydrochloric acid is easily figured out. If 5 c.c. of $\frac{N}{10}$ sodium hydroxid solution are required to neutralize 10 c.c. of gastric juice, the acidity of the 10 c.c., in terms of hydrochloric acid, would equal $5 \times 0.00365 = 0.01825$ gm. of hydrochloric acid; or, expressed in percentage—*i. e.*, calculated for 100 c.c. of gastric juice—0.1825 hydrochloric acid. This method of calculating the percentage of free hydrochloric acid does not give us the actual amount of HCl contained in the gastric juice, for other factors aid in determining the acidity. But it does give us an estimate of the maximum of hydrochloric acid which the gastric juice in question may contain. To calculate the so-called degree of acidity we simply estimate how many cubic centimeters of $\frac{N}{10}$ NaOH are used to neutralize 100 c.c. of the gastric juice—*i. e.*, the percentage of its own volume necessary for the neutralization of the gastric juice under examination. To express the acidity in per cent., we multiply by 10 the number of cubic centimeters of the $\frac{N}{10}$ NaOH solution necessary to neutralize 10 c.c. of stomach contents. A gastric juice, 10 c.c. of which require 5 c.c. of $\frac{N}{10}$ NaOH solution for neutralization, has therefore an acidity of 50. This method of calculation possesses an advantage in corresponding exactly to the actual conditions, whereas the reckoning in terms of HCl incorrectly assumes that the acidity may be due exclusively to hydrochloric acid. On the other hand, changing over to hydrochloric acid perhaps gives us a more distinct idea.

Quantitative Estimation of the Hydrochloric Acid and Chlorids in the Gastric Juice.—The estimation of the total acidity of the gastric juice is a very simple chemical procedure; but the determination of the amount of hydrochloric acid contained in the gastric juice appears more difficult, because we have to consider separately the factors *d*, *e*, and *f* (p. 377). A chlorin estimate alone is of no value in judging of the amount of hydrochloric acid, for it would give only the total sum of $d + e + f$, and include chlorids of the food as well. What we wish to determine is the sum of $d + e$ (*i. e.*, the amount of free and combined hydrochloric acid secreted by the stomach).

Estimation of the Total Unneutralized (Secreted) Hydrochloric Acid of the Gastric Juice ($d + e$, p. 377).—*Sjöqvist's Method.*—This depends upon the fact that the acids which are absolutely free, as well as those which are but loosely combined with proteids, are changed to their corresponding barium salts if mixed with barium carbonate. If the mixture is then evaporated to dryness and incinerated, the barium salts of the organic acids will be transformed to barium carbonate, but the hydrochloric acid will remain fixed as barium chlorid. This can then be isolated from the ash by extraction with water, and then titrated with a solution of potassium bichromate, and the amount of hydrochloric acid be estimated from this.

The steps of Sjöqvist's method are as follows: 10 c.c. of gastric juice are slowly evaporated to dryness, in a silver or platinum dish, with an excess of barium carbonate (free from chlorin). The residue is then kept at a red heat for a few

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moments. After cooling, the residue is first digested with 10 c.c. of water, and then extracted repeatedly with hot water until the filtered extract amounts to 50 c.c.

In order to titrate for barium we add to the solution one-fourth to one-third its volume of alcohol, and 3 to 4 c.c. of a solution of 10 gm. of acetic acid and 10 gm. of sodium acetate in 100 c.c. of water. These additions facilitate the separation of the barium chromate and prevent the precipitation of calcium chromate from any calcium salt that may be present. An 8.5 per cent. solution of chemically pure potassium bichromate is added from a buret to the solution until the barium is completely precipitated. So-called "tetra paper" (tetramethyl-paraphenyldiamin paper) will serve to indicate the end of the reaction. Any excess of potassium bichromate will produce a blue color. Every cubic centimeter of bichromate solution which it was necessary to add in order to complete the end-reaction corresponds to 4.05 mg. of hydrochloric acid. Sjöqvist has recently improved his method by changing the final titration with bichromate, because the reaction is not sharply defined, to a much more sharply defined iodin titration. Iodin is quantitatively liberated by adding KI and HCl to the bichfomate, and the iodin is determined in the well-known way by titrating with sodium thiosulphate and starch. For the technic of this modification we must refer to the original communication in Vol. V. of the *Scandinavian Archives of Physiology*, 1895.

Leo's Method.—Leo's method is based on the fact that both free and combined hydrochloric acid are neutralized by calcium carbonate, while acid phosphates and other combinations reacting in the titration with sodium hydrate show the same acidity after treating with calcium carbonate as before.

Leo first removes the fatty acids by distillation, and the lactic acid by extraction with ether, and then estimates the total acidity of the specimen of gastric juice. In another portion he combines with calcium carbonate both the free hydrochloric acid and that combined with the proteids, and then titrates again. The amount of the acidity which disappears after the addition of calcium carbonate corresponds to the hydrochloric acid present. Calcium chlorid formed in the neutralization of the hydrochloric acid necessitates (on account of the probable presence of acid phosphates) especially skilful manipulation when performing the test. If calcium chlorid is absent, the titration of the acid phosphates will be represented by the following formula: $PO_4H_2K + NaOH = PO_4HKNa + H_2O$. But if calcium chlorid is present in the solution at the same time, the following formula will represent the titration: $2PO_4H_2K + 4NaOH + 3CaCl_2 = (PO_4)_2Ca_3 + 2KCl + 4NaCl + 4H_2O$. In the latter case we need double the quantity of sodium hydroxid for neutralization. Leo adds an excess of calcium chlorid to both specimens before titration, in order to exclude the influence of the calcium chlorid formed by the neutralization with calcium carbonate, when comparing the two titrations. For the details of the technic consult Leo's own communication.[1]

Leo's method will also serve the purpose of estimating at the same time the quantity of acid phosphates.

Lütke-Martius' Method.—Lütke-Martius' is one of the simplest methods for the determination of the total amount of hydrochloric acid secreted (free HCl + that combined with proteids).

We first estimate the total amount of chlorin in a specimen of gastric contents[2] (see below). This is represented by *a*. Another specimen of the gastric contents is incinerated, the chlorin in the ash estimated, and represented by *b*. The latter then represents the chlorin of the chlorids, because the HCl is volatile. By subtracting *b*, the chlorin of the chlorids, from *a*, the total chlorin, we get $a - b$, the chlorin of the hydrochloric acid secretion.[3]

[1] *Diagnostik der Erkrankungen der Verdauungsorgane*, Berlin, 1890.

[2] Martius employs the plain gastric contents exclusively, instead of the filtrate (compare note 2, p. 378).

[3] See following pages. For a correction of this method see Reissner, *Zeit. f. klin. Med.*, vol. xliv., p. 75.

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Volhard's method also estimates the total amount of chlorin. An excess of strongly acid silver nitrate solution is added to a measured quantity of the gastric contents so that all the chlorin present will be combined with the silver. We can then determine whatever excess of silver remains uncombined in the filtrate by titration with ammonium sulphocyanid. The silver will be precipitated as insoluble silver sulphocyanid. Iron alum or sulphate will serve as an indicator to show when the precipitation is complete. It may be added to the silver solution at the beginning. In adding the ammonium sulphocyanid, the red color of the sulphocyanid will persist only after all of the silver has been completely precipitated as silver sulphocyanid. The details of the technic are contained in the following quotation from Lütke:

The following normal solutions are essential:

1. $\frac{N}{10}$ silver solution containing 17 gm. of silver nitrate in a liter: Ferrous sulphate is also added to the solution as an indicator, and an excess of nitric acid. The method of preparation is as follows: 17.5 gm. of silver nitrate are dissolved in about 900 c.c. of 25 per cent. nitric acid, and 50 c.c. of liq. ferri sulphurici oxydati are added to the solution and the volume made up to 1 liter. The solution is standarized with a $\frac{N}{10}$ HCl solution in the usual way.

2. $\frac{N}{10}$ ammonium sulphocyanid solution: 7.6 gm. of $CNSNH_4$ in a liter. About 8 gm. of ammonium sulphocyanid are dissolved in 1 liter of water, and the absolute quantity contained in this solution estimated by means of the $\frac{N}{10}$ silver solution. For this purpose 10 c.c. of the (iron-containing) silver solution are placed in a beaker, and 150 to 200 c.c. of water added; then the sulphocyanid solution is allowed to flow in from a buret until a faint reddish color appears and persists—*e. g.*, if 9.7 c.c. had been used for this purpose, then 970 c.c. of the sulphocyanid solution should be diluted up to 1000 c.c. After such dilution a further trial is made to determine accurately that it is a $\frac{N}{10}$ solution.

(*a*) *Estimation of the Total Chlorin.*—Ten c.c. of the stomach contents, well mixed, are placed in a 100 c.c. graduated flask. The graduate in which the 10 c.c. are measured must previously be rinsed out twice with water. Then 20 c.c. of $\frac{N}{10}$ silver solution are added; the mixture is well shaken and allowed to stand ten minutes. If there is a marked color to the stomach contents, we may decolorize by adding 5 to 10 drops of a potassium permanganate solution (15 : 1). This is not often necessary. The permanganate solution should not be added until all the chlorin is already combined with the silver, otherwise the permanganate will split up the hydrochloric acid and form free chlorin, which volatilizes, so the result of the analysis would be questionable. After decolorization has taken place we add up to 100 c.c., shake well, and filter through a dry filter paper into a dry glass.

Fifty cubic centimeters of this filtrate placed in a beaker are now titrated with the $\frac{N}{10}$ sulphocyanid solution.

The total amount of chlorin is calculated as follows: The number of cubic centimeters of sulphocyanid solution required is multiplied by 2, and the product deducted from the volume of the silver solution used (20 c.c.).

(*b*) *Determination of the Chlorids.*—Ten c.c. of well-mixed gastric contents in a platinum dish are evaporated to dryness over a water bath. The residue is incinerated over the direct flame so long as the ash burns with a bright luminous flame. Excessive and prolonged heating is superfluous and to be avoided, because the chlorids volatilize at red heat. After the burning of the residue the moistened ash is pulverized with a glass rod and extracted with about 100 c.c. of warm water, and the fluid then thrown upon a filter. Experience has shown that this amount

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of water is sufficient for a complete extraction of the ash. If it is doubtful, however, whether all the chlorin has been washed out, a few drops of the silver solution may be added to the last few drops of the filtrate. Any cloudiness would indicate the presence of chlorin and necessitate further extraction. The entire filtrate is then placed in a beaker with 10 c.c. of $\frac{N}{10}$ silver solution, and titrated with the $\frac{N}{10}$ sulphocyanid solution. The amount of chlorin held in combination is calculated by subtracting the number of cubic centimeters of the sulphocyanid solution from the amount of silver used (10 c.c.).

(*c*) *Determination of the Hydrochloric Acid.*—The amount of hydrochloric acid contained in 10 c.c. of gastric contents is calculated from the two values found (total chlorin (*a*) and chlorin of the chlorids (*b*)) by subtraction. From the amount of total chlorin, expressed in cubic centimeters of $\frac{N}{10}$ solution, the amount of chlorin of the chlorids, expressed in the same way, is deducted. By multiplying the result by 0.0365, the absolute amount of hydrochloric acid contained in 100 c.c. of stomach contents, or the percentage of the hydrochloric acid, is obtained.

The author's own experience shows that the amount of $\frac{N}{10}$ silver solution employed in both determinations (a=20 c.c., b=10 c.c.) has been found quite sufficient to combine with all the chlorin. If it should happen that a gastric content be observed with a greater content of chlorin, then more of the $\frac{N}{10}$ silver nitrate solution must be added.

O. Reissner[1] has called attention to the fact that the hydrochloric acid values obtained by means of the Lütke-Martius method are erroneous in one respect, since the gastric juice contains ammonium chlorid, the ammonia of which, as shown by Sticker, is obtained chiefly from the saliva, although under pathologic conditions it may also arise within the stomach, from the putrefaction of albumin. When the residue of the gastric contents is incinerated in the Lütke-Martius method, to remove the hydrochloric acid and to obtain the value *b* (see p. 381), the ammonium chlorid is volatilized as well. The value *b* for the chlorin of the chlorids is consequently too low, and the value $a - b$ for the secreted acid will be correspondingly too high. This explains the fact that the Lütke-Martius method sometimes gives a value for the secreted hydrochloric acid which is greater than the total acidity, a condition of affairs which is manifestly impossible, since the total acidity includes not only the hydrochloric acid, but the acid phosphates as well. Reissner has also shown how to avoid this source of error. He eliminates the ammonium chlorid before determining the value of a; the subtraction $a - b$ will then give a correct result. Instead of determining a directly by titrating the gastric contents with the silver and sulphocyanid solutions, Reissner advises neutralization of a measured quantity of the gastric contents, its incineration in a platinum dish, the solution of the ash in hot water, and the titration of the filtrate in the customary manner. By this modification the ammonium chlorid is volatilized and eliminated from the chlorin value a. In the neutralization Reissner believes it is best not to go beyond the neutral reaction upon litmus, since, when phenolphthalein is employed as an indicator, the end-reaction is postponed, and there is danger that the chlorin of the ammonium chlorid will be partly fixed by the sodium as sodium chlorid.

Hehner-Maly's Method.—Hehner-Maly's method (recommended by Leube for clinical work) is based on the fact that where a mixture of organic and inorganic acids is neutralized and reduced to an ash, the organic salts are changed to alkaline carbonates by the process of combustion, but the inorganic salts remain neutral. If this alkalinity—*i. e.*, an equivalent acidity—is deducted from the total acidity, the remainder is the acidity due to mineral acid.

[1] *Zeit. f. klin. Med.*, vol. xlviii., parts 1 and 2, p. 106.

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For the gastric juice the process is as follows: 10 c.c. of gastric juice are titrated in the usual way with $\frac{N}{10}$ NaOH, and with phenolphthalein as an indicator. The neutralized solution is evaporated to dryness in a platinum dish and then incinerated, the ash dissolved in distilled water, and the alkaline solution titrated with $\frac{N}{10}$ acid (oxalic acid or hydrochloric acid, with phenolphthalein again added as an indicator) till the decoloration is complete. The $\frac{N}{10}$ acid used in the titration corresponds to the acidity caused by organic acids. To determine the amount of hydrochloric acid, this value is simply to be deducted from the total acidity. This method is very simple and well adapted for clinical purposes. The only disadvantage is that the acid phosphates are estimated as HCl, for they act as mineral acids when reduced to ashes. Very accurate results can be obtained by estimating the acid phosphates according to Leo's method (p. 380), and deducting the acidity belonging to them from the HCl as obtained by Hehner-Maly's method. Of course, the hydrochloric acid would be determined at the same time by Leo's method, so that Hehner-Maly's method would be needed only for the determination of the organic acids.

This method, however, gives incorrect results in respect to organic acids in case these include the higher fatty acids, which are insoluble in water, and consequently not completely neutralized by the addition of $\frac{N}{10}$ NaOH. Should it be desired to estimate these higher fatty acids, the gastric contents, neutralized by means of the $\frac{N}{10}$ NaOH, must be well shaken with a generous quantity of ether, and any acidity arising from these fatty acids neutralized by the addition of an alcoholic $\frac{N}{10}$ NaOH. This neutralized ethereal solution is then added to the neutralized watery solution of the gastric contents, and the mixture evaporated. By proceeding in this manner we are certain that the hydrochloric acid, as well as all of the organic acids, has been neutralized in the residue. The alkalinity of the ash is the measure of the previously present organic acids. By means of this modification the Hehner-Maly method is also of value in estimating the higher fatty acids.

If it is desired to estimate the higher fatty acids by means of this procedure, the *unfiltered* gastric contents must be employed, since these acids (with the exception of butyric acid), on account of their insolubility, are simply suspended in the gastric contents, and would consequently remain upon the filter.

Determination of the Free Hydrochloric Acid (Even Free from Proteids) which Gives the Color Reactions Previously Mentioned —*i. e.*, of the Excess of Acid (p. 377).—Mintz's method of titration is the best—that of adding a $\frac{N}{10}$ NaOH solution to the gastric juice from a buret until we fail to get the usual color reaction for free hydrochloric acid (see p. 372 *et seq.*). Phloroglucin-vanillin is the most highly recommended of the reagents by Mintz. It requires but a few drops of the gastric contents, it is not influenced by any organic acid, and the final reaction is sharply defined. A still more convenient method than that described upon p. 373 is to add 25 to 30 drops of phloroglucin-vanillin to the 10 c. c. of gastric contents before titrating, for then, by heating the end of a glass rod moistened in the contents, we can observe the reaction directly. Of course, the glass rod should be cooled and washed carefully each time.

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Another reaction, the addition of methyl-violet to normal gastric contents, and then $\frac{N}{10}$ NaOH solution until the blue color produced changes again to violet, has not given the author any satisfactory results, because it is so difficult to recognize the final reaction. But the end-reaction may be very well recognized with Congo paper, adding the $\frac{N}{10}$ NaOH solution until the Congo paper is no longer colored blue. Rugil recommends this method. The Tüpfel method is still more convenient, carrying the fluid in a platinum-loop to the Congo paper. We must, however, remember that the titration with the phloroglucin reaction will not necessarily give the same results as with Congo-red, on account of the degrees of delicacy of the indicators, and also on account of the sensitiveness of Congo-red to organic acids.

Determination of the Hydrochloric Acid Deficit in a Gastric Contents which Does Not Give a Reaction for Free Hydrochloric Acid.—By hydrochloric acid deficit we mean the amount of HCl which must be added to a definite volume of the stomach contents in order to bring out the color reactions for free hydrochloric acid. This figure varies, of course, on the one hand, with the amount of proteids and peptones present, and perhaps with the alkaline components of the secretion, all of which combine with the acid; and, on the other hand, with the amount of hydrochloric acid already attached to the proteids. We might therefore call it a deficit in HCl saturation. To determine this deficit we add a $\frac{N}{10}$ HCl solution from a buret to 10 c.c. of stomach contents until the reaction for free hydrochloric acid is obtained in the mixture; this reaction is indicated most accurately by phloroglucin-vanillin or Congo-red.

[The *Töpfer method* for the estimation of the total acidity of the gastric contents, consisting of free and combined acid, has found wide use in the chemical laboratories in the country. The results obtained by it are sufficiently accurate for the purposes of diagnosis, for which they are employed.

The basis of the method is as follows:

The *total acidity* is estimated by means of *phenolphthalein;* the indicator reacts acid to free and combined acid and acid salts, and can, therefore, be used for this purpose.

The *combined acidity* is determined by subtracting the acidity as found by *alizarin S* from the total acidity. This value corresponds to the acid combined with proteids, since *alizarin S* reacts acid to all acid compounds except the acid proteid combinations.

The *free acidity* is found by titration, using *dimethylamidoazobenzol.* This indicator is only sensitive toward free acids.

The following solutions are necessary for the performance of the method:

1. A 0.5 per cent. alcoholic solution of dimethylamidoazobenzol.
2. A 1 per cent. aqueous solution of alizarin S.
3. A 1 per cent. alcoholic solution of phenolphthalein.
4. A $\frac{N}{10}$ NaOH solution.

The details of the method follow:

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The total acidity is found by titrating 5 or 10 c.c. of the gastric contents with the $\frac{N}{10}$ NaOH solution after the addition of 3 drops of phenolphthalein. The titration is continued until the pink color, which becomes permanent near the end of the reaction, can no longer be deepened by the further addition of a drop of the $\frac{N}{10}$ NaOH solution. The number of cubic centimeters required for the end-reaction corresponds to the total acidity.

The combined acidity is determined by adding 3 drops of the alizarin solution to 5 to 10 c.c. of the gastric contents and titrating with the NaOH solution. The color of the solution, when the end-reaction is reached, is a pure violet. The red color which appears after the yellow of the indicator has disappeared must be completely removed, and the color must be a decided violet.

The difference between the number of cubic centimeters found in this titration and that of the total acidity equals the combined acidity of the gastric contents.

The free acid is estimated by taking 5 to 10 c.c. of the gastric contents, adding 4 drops of dimethylamidoazobenzol, and titrating with the $\frac{N}{10}$ NaOH. The titration is continued until the pink has disappeared and the color has become greenish yellow. This number of cubic centimeters is equivalent to the free acidity.

The value of these various acid factors can be expressed in many ways. If it is required that they be in terms of HCl, then it is simply necessary to multiply the number of cubic centimeters found in the titration by 0.00365. The product will equal the number of grams of HCl in the volume of gastric contents employed for the titration. If percentage is required, the above product must, of course, be further multiplied by the fraction $\frac{100}{x}$, where x = the cubic centimeters employed in the titration.

As Prof. Sahli has suggested, it is more usual to express these values in terms of the number of cubic centimeters of the $\frac{N}{10}$ NaOH which are required to complete the respective end-reactions for 100 c.c. of gastric contents. If 10 c.c. of the stomach contents are used, the number of cubic centimeters used up in titration is multiplied by 10, etc.

In cases where only small volumes of gastric contents stand at the disposal of the investigator, Einhorn's modification may be found to be very useful. It consists in the performance of the dimethylamidoazobenzol and phenolphthalein reaction upon the same portion of contents.

Ten cubic centimeters are measured off, and to these are added 3 drops of phenolphthalein and 4 drops of the dimethylamidoazobenzol. Since the former is colorless in acid solution, the color of the mixture is entirely dependent upon the latter indicator, and the titration may be carried on with the $\frac{N}{10}$ NaOH in the same manner as in the determination of free acid—*i. e.*, to the appearance of the greenish-yellow color. The reading is then made upon the buret, and the $\frac{N}{10}$ NaOH solution further added until the pink color appears and allows of no further deepening upon the addition of another drop of the alkali. This second

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reading will correspond to the total acidity, and the first reading to the free acidity, when both readings are made from the original position of the fluid in the buret.—H. C. J.]

Determination of the Chlorids of the Gastric Contents.—O. Reissner[1] has recently pointed out that the estimation of the combined chlorin or of the neutral chlorids has a certain diagnostic significance in the recognition of gastric carcinoma. He has found a greater increase in the chlorids in this disease than in other affections of the stomach, a fact which is the more striking since the secretion of hydrochloric acid is usually greatly diminished in gastric cancer (see p. 390). Reissner believes this to be due to the neutralization of the hydrochloric acid, provided such be secreted at all, by the alkaline juice of the carcinoma, and to the chlorids contained in this cancerous secretion. If it is desired to estimate the chlorids in this connection, the directions will be found in the description of the method of Lütke-Martius (p. 380). In carrying out this procedure the modification of Reissner must be employed (see p. 382). By this method Reissner found that in other gastric diseases 100 c.c. of gastric contents corresponded to a chlorid value of 24 to 40 c.c. of $\frac{N}{10}$ silver solution; while in carcinoma the chlorids in the same quantity of gastric contents required 50 to 70 c.c. of $\frac{N}{10}$ silver solution. It has not been determined whether gastric ulcer resembles carcinoma in this respect.

Quantitative Determination of the Total Organic Acids of the Gastric Contents.—The amount of total organic acids may be calculated after an Ewald test breakfast by subtracting the hydrochloric acidity, as found by Lütke-Martius' method, from the total acidity, allowing, of course, that the acid salts do not play much of a part in the acidity. Martius considers that normally organic acids are absent during the digestion of the test breakfast in the stomach (see p. 382 *et seq.*); hence, this remainder should equal nil.

The organic acids may also be estimated by Hehner-Maly's method (p. 382). This gives the acidity due to the organic acids directly.

Quantitative Estimation of Lactic Acid.—Strauss' modification of Uffelmann's method (p. 374) is practical and sufficiently accurate for the quantitative estimation of the lactic acid in the gastric contents. Boas[2] employs the iodoform reaction (p. 376 *et seq.*) when a more accurate estimation is required. The amount of iodin required to change the aldehyd formed from the lactic acid to iodoform is determined by titration.

If there is required a more accurate quantitative estimation of these acids than can be obtained by the Strauss modification of Uffelmann's test, the Hehner-Maly method for the organic acids may be employed (p. 382); for other organic acids have the same diagnostic significance as lactic acid, and are quantitatively, at least, subordinate.

Practical Utilization and Choice of the Quantitative Methods for the Determination of Acids.—At first sight the most accurate methods would seem absolutely essential for cor-

[1] *Zeits. f. klin. Med.*, vol. xliv.

[2] *Deutsch. med. Woch.*, 1893, No. 34.

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rect gastric diagnosis. We should, then, naturally select Lütke-Martius' or Sjöqvist's method to determine the total HCl secreted, Leo's method for the acid phosphates, Mintz's for the excess of acid—*i. e.*, the acid deficit according to Mintz—and, finally, Boas' for the determination of the lactic acid. But, as a matter of fact, none of these methods is absolutely accurate; and so many would make gastric examination too complicated and tedious for practical use. Even the washing out of the stomach after all the test breakfast capable of expression has been removed, and the including of this wash water in the tests, will not furnish absolutely accurate values for the secretory powers of the stomach. For the rinsing of the stomach, as well as the efforts in expressing the meal, will produce new and inconstant stimulants to secretion. Besides, before the test breakfast is removed an unknown portion of the acid has been already passed on into the duodenum.

Fortunately, in practice it is much more simple. The acidity due to acid phosphates is of no practical importance for titration if we employ Ewald's test breakfast (compare p. 388). The investigations of Moritz[1] and Martius show that the acidity is almost entirely made up of the organic and hydrochloric acids.

The hydrochloric acids are composed of hydrochloric acid combined with the proteids, and of the free acid—reacting to Congo-red and phloroglucin-vanillin. A few examples will show that (if these facts are considered) even a simple acidity titration of the gastric contents furnishes considerable information about the gastric chemistry, provided the qualitative tests have been already performed.

Let us assume a case where the tests for free hydrochloric acid are negative. The acidity is found to be quite high—*e. g.*, reduced to terms of HCl, 0.25 per cent. We may then be pretty certain that most of this high acidity is due to free organic acids, and that the hydrochloric acid secretion is slight. If the lactic acid test is very marked, if the odor of butyric or acetic is strong, and if the stomach contents contain an abundance of bacteria, this assumption is practically proved. Conversely, if the hydrochloric acid reaction is very pronounced, the lactic acid reaction very faint or absent, and the acidity very high, in all probability there is an abnormal or excessive secretion of free HCl. If the acidity is relatively low and the hydrochloric acid reaction positive, the probability is still greater that the acidity depends largely upon free hydrochloric acid. The titration for the total acidity is valuable, quite independent of the tests for free hydrochloric acid, for it indicates the maximum limit of HCl which is possible. For example: If the acidity is found to be 0.08 per cent. (calculated as HCl) we are sure that the amount of free hydrochloric acid is not more than 0.08 per cent., anyway; which means that it is considerably diminished.

The excess or deficit of acid (see p. 383 *et seq.*) is easily estimated and gives us an important insight into the gastric chemistry. Some authors, as is well known, consider that in the determination of hydro-

[1] *Deutsch. Arch. f. klin. Med.*, 1889, vol. xliv., p. 279.

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chloric acid the free hydrochloric acid is the important part to be estimated, because in artificial digestion that is the only part which is effective. But other authors, on the contrary, consider that the free HCl can be neglected, as being, so to speak, useless, while the hydrochloric acid which is combined with the proteids is the important part, because it is accomplishing digestion in the stomach. Both are probably correct in a measure. The combined acid is doing the work, but the presence of a moderate excess is a favorable sign. It shows that there is enough to saturate the proteids and still leave more ready for anything else to be digested. The excess also aids in the antiseptic function of the stomach. But at the same time the hydrochloric acid deficit cannot be considered to measure an actual deficiency for the organism.

A comparison of the total acidity, as indicating the degree of acid secretion, with the excess of acid or acid deficit will aid in determining the condition of the other gastric functions, especially the motility and the power of absorption. For example: If the total acidity is high, and we suppose that this is mainly due to hydrochloric acid secreted, and if further examination reveals no excess or only a slight excess of hydrochloric acid, or perhaps even an acid deficit, the high acidity can be due only to a deficient motility and absorption, so that much of the proteids remained in the stomach. A low total acidity with an excess of hydrochloric acid proves, on the contrary, that the motility and the absorbing power of the stomach are excellent.

If a qualitative examination of the gastric juice shows that lactic acid is present, it may be quantitatively determined by Strauss' method (p. 375), the result deducted from the total acidity, and the remainder of the acidity calculated as above. Generally speaking, when much lactic acid is present we find low HCl values and only combined HCl—*i. e.*, diminished secretion and diminished motility.

PHYSIOLOGIC RELATIONSHIP OF THE ACIDS OF THE GASTRIC JUICE.

Under physiologic conditions, one hour after Ewald's test breakfast we find that the total acidity of the gastric juice varies between 0.15 and 0.2 per cent. (calculated in terms of HCl). The hydrochloric acid tests with methyl-violet, tropæolin, and phloroglucin-vanillin all react positively; the lactic acid test is negative. In healthy individuals, Martius has shown that these values for the total acidity during the whole of the digestion correspond fairly accurately to the amount of hydrochloric acid secreted (estimated by Lütke-Martius' method). This proves that the organic acids are absent, and, at the same time, that the acid phosphates influence the total acidity but very little. The occurrence of organic acids in the stomach during the digestion of an Ewald test breakfast is pathologic if we disregard the very slight amount of lactic acid which the test breakfast itself contains. As a matter of fact, the antifermentation action of the hydrochloric acid inhibits the formation of these organic acids. Miller claims that lactic

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acid fermentation will cease if the amount of hydrochloric acid contained in the mixture amounts to 0.16 per cent., and Cohn puts it even as low as 0.07 per cent. Schüle[1] has contributed valuable facts in regard to the normal course of the acidity of the gastric juice and its individual components with different diets. He pictures the following curves (Fig. 145) to show the relations of the acidities after the ingestion of an Ewald test breakfast.

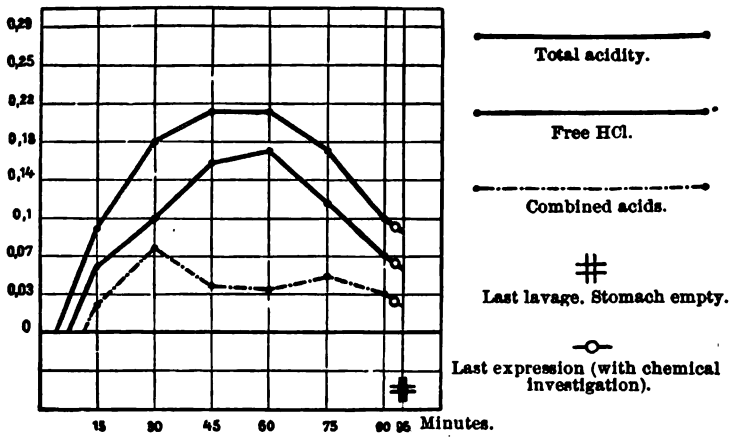

FIG. 145.—Course of the acidity of the gastric juice after a test breakfast of 300 gm. of tea and 50 gm. of milk-bread (after A. Schüle).

We quote his observations upon healthy persons in the following:

"1. The values of the free as well as of the combined HCl and of the total acidity often differ considerably in the same individual, and in different people, too, without any demonstrable cause. 2. The maximum of free HCl varies between 0.05 to 0.07 and 0.2 per cent.; the combined acids, between 0.012 and 0.11 per cent. The maximum of the total acidity lies, in round figures, between 30 (0.11 per cent. HCl) and 70 (0.26 per cent. HCl) at the height of digestion. 3. The height of digestion occurs sixty minutes after an Ewald test breakfast, varying in individual cases from forty-five to seventy-five minutes. 4. The upper limits of these figures must be regarded as pathologic for many individuals with sensitive gastric mucous membranes."

Diagnostic Notes upon Acid Contents of the Gastric Juice.

So long as we do not attempt to draw conclusions in regard to the volume of the secretion from the percentage of acidity (see p. 397), experience teaches as follows:

(*a*) Normal hydrochloric acid secretion is present:
  (1) Very often in gastric ulcers, and in the pyloric stenosis due to a healed gastric ulcer.
  (2) In some of the gastric neuroses.
  (3) In simple atony of the stomach.

(*b*) The hydrochloric acid secretion is found to be increased:[2]
  (1) In the majority of the cases of gastric ulcers.

[1] *Zeits. f. klin. Med.*, 1895, vol. xxviii.

[2] If the free hydrochloric acid after an Ewald's test breakfast exceeds 0.2 per cent., or if the total acidity amounts to more than about 70 (0.26 per cent. HCl), we may speak of it as an increased secretion. Pathologically, total acidity as high as 0.8 per cent. has been observed, due chiefly to HCl, but values over 0.35 per cent. are rare.

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(2) In passive congestion of the stomach (Schreiber) with maintained secretion of acid.

(3) In diseases of continual hypersecretion[1] where the secretion continues to be excessive after the removal of the meal.

The most important differential diagnostic sign between true continued hypersecretion (gastrorrhœa acida) and Schreiber's so-called "stagnant stomach" (where prolonged secretion of the gastric juice occurs, due to motor insufficiency with or without stenosis of the pylorus) is the condition of the fasting stomach in the morning, after it has been completely emptied the evening before. If it, then, the next morning contains fluid rich in hydrochloric acid, the condition is one of true hypersecretion.

(4) In simple hyperacidity and hypersecretion where (in contrast to (3)) the increased hydrochloric acid production occurs only during digestion.[2]

(5) In temporary hypersecretion (gastroxynsis). This occurs in nervous individuals, as a result of severe mental exertion, associated with migraine, acid vomiting, and all sorts of nervous disturbances.

(6) In the early stages of chronic gastric catarrh.

(7) In some types of mental disorders.

(*c*) Diminished secretion of hydrochloric acid occurs:

(1) In anemic conditions, especially in the severe forms.

(2) In most cases of chronic gastric catarrh.

(3) In some disorders of the stomach developing in the course of general neuroses (neurasthenia).

(4) In some types of mental disorders.

(5) In continued icterus.

(6) In some chronic cachexias—*e. g.*, tuberculosis of the lung (not constant).

(7) Sometimes in diseases with congestion (heart disease, emphysema).

(8) At times in chronic nephritis.

(9) After prolonged use of alkalies and saline purgatives.

(10) In chlorin hunger.

(*d*) Free hydrochloric acid[3] is absent in those conditions which are mentioned under (*c*) when the disorder is very pronounced, and, besides this, more or less typically:

(1) In severe febrile, especially infectious, diseases.

(2) In carcinoma of the stomach.

(3) In atrophic gastric catarrh.

(4) In pernicious anemia.

In reality numerous deviations are observed in all of the conditions which are grouped in the above scheme.

We are justified in attaching great weight to the absence of free hydrochloric acid—*i. e.*, to a negative result in the color tests—for the diagnosis of gastric carcinoma, especially if this HCl absence can be noted regularly before any tumor can be felt. Nevertheless the presence of free HCl does not necessarily exclude the diagnosis of a carcinoma. One case was especially instructive to the author in this respect. For months he found a normal amount of hydrochloric acid after a test breakfast, although the patient presented a carcinomatous stenosis of the pylorus with marked nocturnal retention of food. In the retained material he

[1] See p. 368 for the amount of secretion occurring in the fasting stomach of healthy individuals.

[2] There is really no sharp boundary line between these two conditions. If merely the percentage of acid contained is too high, we call it hyperacidity; but if, besides this, the volume of the gastric juice is also increased, we call it also hypersecretion. Hyperacidity and hypersecretion may occur independently, or they may result from a motor insufficiency (stagnation).

[3] The more exact quantitative methods for testing the total and the combined hydrochloric acid—*e. g.*, Lütke-Martius'—would still generally show that a slight amount of hydrochloric acid was secreted. Yet the secretion may even be completely suppressed and the gastric juice then give a neutral or weakly alkaline reaction.

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found a very greatly increased amount of hydrochloric acid (up to 0.4 per cent.). Moreover, other disturbances cited in groups *c* and *d* are frequently accompanied by a complete absence of the color reaction for free HCl, so that an absence of free HCl can hardly be claimed as distinctive of gastric carcinoma.

(*e*) A considerable amount of organic acids, especially of lactic acid, occurs only when hydrochloric acid is absent and when, at the same time, motor insufficiency exists, especially when the latter depends upon some stenosis of the pylorus. This is the reason that a considerable amount of lactic acid in the gastric contents strongly suggests a carcinoma.

Testing of the Digestive Power of the Gastric Juice; Examination for Pepsin.

The digestive power of the filtered stomach contents from the expressed test breakfast depends, on the one hand, upon the amount of pepsin contained, and, on the other hand, upon the amount of free acid contained, especially free hydrochloric acid. Artificial digestion is the only means at our command to test the amount of pepsin. This furnishes also indirect evidence of the amount of acid contained in the gastric juice.

For artificial digestion we usually employ fibrin stained with carmin (Grützner), or disks of coagulated egg albumin.

The fibrin is best prepared by beating freshly drawn ox blood. The stringy coagula are washed in running water until decolorized, and then cut into small pieces of uniform size, which are then placed in alcohol for several days. They are then transferred for one to two days into a neutral concentrated solution of carmin and kept cool until they are thoroughly stained, washed in water until the wash water is no longer colored, well squeezed, and then preserved in carmin containing glycerin. Before being used they should be washed in water until they do not give off any colored glycerin.

The egg-albumin disks are prepared by boiling an egg hard,[1] and then with a cork borer punching out the white cylinders of about 5 mm. diameter.

For the digestive test a few bits of fibrin or some egg-albumin disks are put into a test tube with a measured quantity of the gastric contents and the tube then set in an incubator. The digestion of the fibrin releases the carmin and becomes evident from the red color of the fluid. The digestion of the egg albumin always progresses much more slowly. It shows itself first in the rounding of the edges of the disk, followed gradually by complete solution.

The following methods are to be recommended for a more accurate quantitative examination:

Hammerschlag's[2] Method for Estimating Pepsin.—Ten c.c. of an approximately 1 per cent. filtered solution of egg albumin[3] in 0.4 per cent. HCl are poured into two tubes. To one 5 c.c. of gastric contents are added, and to the other 5 c.c. of distilled water. They are both set in the incubator. An hour later the albumin in both tubes is estimated volumetrically according to Esbach's method (p. 505 *et seq.*). The difference between the precipitate of albumin in the

[1] The egg should be boiled only long enough to coagulate completely the egg albumin, otherwise it becomes too difficult to digest. The necessary boiling-period must be found experimentally. These cylinders are sectioned with a razor into small disks of 1 mm. thickness, preserved in glycerin, and also washed off before using.

[2] *Internat. klin. Rundschau*, vol. viii., No. 39, 1895.

[3] Fresh egg albumin contains about 13 per cent. of dry proteid, and should therefore bediluted about 13 times in order to make approximately a 1 per cent. solution of egg albumin.

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two tubes is equal to the amount of albumin which has been digested, and forms, therefore, a measure of the peptic activity of the gastric juice. The square root of the amount of pepsin is proportional to the quantity of albumin dissolved, according to Schütz,[1] Borrissow,[2] and Linossier.[3] The objections to this method are that the Esbach test is not very accurate and that the albumoses are also partly precipitated. The error in regard to the precipitation of the albumoses is negligible, since they come down so finely divided and settle with so much difficulty that practically they do not influence the amount of the sediment to any appreciable extent. The method is sufficiently accurate for practical utility in judging of the quantity of pepsin in the stomach contents. It would be better, however, to employ a diluted gastric juice, for the reasons given in the description of Mett's method.

Mett's[4] Method of Pepsin Determination.—Glass capillary tubes, from 1 to 2 mm. in diameter and 20 to 30 cm. in length, are filled by suction with the fluid portion of fresh egg albumin. In order to avoid accidental variations in the egg albumin, the whites of several eggs should be mixed, and the fluid portion of the mixture employed. These filled capillary tubes are closed with bread crumbs and placed horizontally in a boiling water bath, in order to coagulate the albumin quickly. At the end of five minutes they are withdrawn, wiped off, and their ends plunged into melted paraffin, in order to prevent drying. A number of these capillary tubes may be prepared and kept in stock; but before they are employed it should be noted that the cylinders of albumin are still in contact with the walls of the tube and have not dried out and contracted, in which case they are unfit for use. At first the capillary tubes contain countless bubbles, which, however, gradually disappear, and in two days they are ready for use. They are then cut into lengths of about 2 cm. with glass scissors, and 2 of them are placed in a little dish with 5 c.c. of the acidulated gastric contents, the pepsin value of which is to be determined. The manner in which the digesting mixture is to be prepared from the gastric contents will be subsequently stated. Digestion is now allowed to proceed in the incubator; it is manifested by the fact that the albumin gradually disappears from the ends of the tubes, the disappearance gradually becoming more rapid.

According to Nirenstein and Schiff,[5] the length of the cylinder of albumin digested by any gastric juice is proportional to the duration of digestion and independent of the diameter of the capillary tube, provided that the length of the digested cylinder does not exceed 7 mm. within the time of observation. If the digested cylinder exceeds 7 mm., however, the length is not proportional to the time, since the digestion beyond this point seems to proceed more slowly as a result of the difficulty of the diffusion of the digested products into the surrounding fluid. To obtain a correct quantitative estimate of the amount of pepsin in the gastric contents we must consequently keep within this limit, which may be done by preparing the digesting mixture in the manner to be presently given, and measuring the mass of albumin dissolved at the end of twenty-four hours. This may be roughly estimated macroscopically, and more exactly by a low-power microscope provided with an objective micrometer. If two capillary tubes are employed, their four ends furnish four digestion-lengths, from which an average may be obtained.

For solutions of pure pepsin, Schütz has discovered the law that the relative quantities of pepsin in digesting mixtures containing the same quantity of hydrochloric acid are proportional to the squares of the quantities of albumin digested in the same time. Borissow has confirmed this, particularly in reference to this method of Mett, so that in this connection Schütz's law is, that the square of the length of the digested cylinder of albumin is proportional to the pepsin concentration of the solution. From the studies of Nirenstein and Schiff, however, this

[1] *Zeit. f. physiol. Chemie.*, 1885.
[2] Quoted by Samoiloff, *Arch. des sci. biol.*, St. Petersburg, vol. ii., No. 5.
[3] *Jour. de phys. et de path. gen.*, vol. i., No. 2, 1899.
[4] J. A. D., Petersburg, 1889, from Pawlow's Laboratory.
[5] *Arch. f. Verdauungskrankh.*, vol. viii., part 6, 1903.

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is true only for the less concentrated pepsin solutions. If the quantity of pepsin in the digesting fluid is so large that more than 3.6 mm. of albumin are digested in twenty-four hours, the law of Schütz does not obtain reliable values for the quantity of pepsin in any gastric juice by Mett's method ; then it must be diluted so that the digestion length will not exceed 3.6 mm. in twenty-four hours. We have already given two reasons which make it necessary to dilute the gastric juice in determining the amount of the contained pepsin. A third and far more important reason for such dilution, to which the author referred in the last edition of this book, and which has recently been more critically discussed by Nirenstein and Schiff, is that the gastric juice, as obtained from the patient by filtering the gastric contents after a test breakfast, always contains an uncertain quantity of substances which inhibit pepsin digestion. These inhibiting substances are the products of the peptic digestion itself; Nirenstein and Schiff state that they are particularly the dissolved carbohydrates, and also sodium chlorid. Gastric juices with diminished amounts of hydrochloric acid, on account of the large quantities of carbohydrates which they contain, are the richest in such inhibiting substances. The important rôle played by these inhibiting substances is shown by the fact that, if Mett's method is carried out with different slight dilutions of the same gastric juice, the relative pepsin values, as obtained by applying Schütz's law to the digestion lengths, never agree with the relative amounts of pepsin as calculated from the dilution. As a matter of fact, it will be found that if the gastric juice be diluted to two or three times its volume, a higher digestive value will frequently be obtained than if the undiluted juice be employed. This is evidently due to the fact that in such a dilution the diminished amount of pepsin is more than compensated for by the weakening of the activity of these inhibiting substances. The presence of these substances consequently gives rise to conditions beyond computation, which make it impossible to arrive at accurate conclusions if the pepsin value is calculated from the pure gastric juice.

It is apparent that the way to avoid this difficulty is to eliminate this inhibitory action by diluting the gastric juice. In the last edition of this book, having this end in view, the author recommended a tenfold dilution of the total gastric juice in diluted hydrochloric acid. Nirenstein and Schiff, however, have shown that it is better to carry the dilution still farther. They recommend a sixteenfold dilution with $\frac{N}{20}$ hydrochloric acid (= 0.18 per cent. HCl), and state that Schütz's law obtains with this and all further dilutions. The latter requisite is absolutely necessary if we wish to have a relative expression for the quantity of pepsin as estimated from the digestion length. This marked dilution also decreases the amount of pepsin in the mixture, so that the digestion length is kept within the limits of Schütz's law (3.6 mm. in twenty-four hours). The author must nevertheless observe that this is not always the case with a very active gastric juice, so that if the digestion length exceeds 3.6 mm. with a sixteenfold dilution, it becomes necessary to repeat the pepsin test with a dilution of 1 : 32.

According to Nirenstein and Schiff, the method of Mett will consequently be carried out in the following manner if exact quantitative results are to be obtained : 1 c.c. of the filtered gastric contents is diluted with 16 c.c. of $\frac{N}{20}$ HCl (= 0.18 per cent. HCl). Two Mett's tubes are then laid in this mixture, which is placed in the incubator. At the end of twenty-four hours the digestion lengths are read [and the mean digestion length obtained]. The square of this digestion length is the measure for the relative amount of pepsin in the sixteenfold diluted gastric juice, and this number, multiplied by 16, gives the relative amount of pepsin in the undiluted gastric juice. In those cases in which the length of the digested cylinder of albumin exceeds 3.6 mm. in length, in spite of the sixteenfold dilution, the estimation must be repeated with a dilution of 1 : 32. By squaring the digestion length as before, and multiplying this quantity by 32, the relative amount of pepsin in the undiluted gastric juice will be obtained. It is clear that in this method of estimation the unit of the relative amount of pepsin will

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be that quantity of pepsin by which 1 mm. of albumin in a Mett's tube will be digested in twenty-four hours with an acidity of 0.18 per cent. free hydrochloric acid. In this estimation we do not consider the absolute quantity of the pepsin, but simply the degree of its concentration, for the result of Mett's method is the same whether large or small quantities of the digesting mixture be employed; at least for quanties above the 16 c.c. above recommended. By employing this method Nirenstein and Schiff have found striking differences between individual gastric secretions, which vary between 0 and 256 pepsin units. The pepsin concentration is consequently entirely independent of the amount of acid in the gastric juice, as we might expect from the fact that physiologists have demonstrated a different localization for the formation of pepsin and hydrochloric acid in the gastric glands.

It is usually assumed that the pepsin secretion is of less diagnostic significance than that of hydrochloric acid, because it is less often disturbed. In view of the Mett's method as just described, however, this belief must be more critically tested, since it may be due to the fact that the earlier methods of pepsin estimation were faulty.

Pepsin and pepsinogen may be entirely absent, but then usually only in grave stomach disorders, especially in atrophic gastric catarrh, in carcinoma of the stomach, and in certain types of pernicious anemia. It is a very unfavorable prognostic sign, so far as the possibility of restoring the gastric function is concerned.

Examination of the Gastric Juice for Rennin and Rennin Zymogen.

Normal gastric juice contains, besides hydrochloric acid and pepsin, rennin ferment and rennin zymogen as additional secretory products of the mucous membrane. As we well know, rennin possesses the property of coagulating milk, independently of the help of acid.[1] Rennin zymogen, inactive in itself, is converted into rennin by acids. Rennin is rapidly destroyed by alkalies, but the zymogen is much more resistant to them.

To demonstrate rennin, 3 to 5 drops of gastric juice are added to 5 to 10 c.c. of fresh uncooked neutral or amphoteric milk, and the mixture is placed in an incubator. If the rennin is present in normal amounts, curdling should take place in from ten to fifteen minutes (Leo). In this process the slight amount of acid added in the gastric juice will cause no precipitation. If the curdling takes place very slowly, it is questionable whether it is due to the action of the rennin or to the formation of lactic acid, and so, to be exact, the reaction of the mixture is taken before and after curdling has taken place. Rennin action is sure to have occurred only when curdling occurs without any change in the reaction.

In the absence of rennin ferment, Boas claims that rennin zymogen may be demonstrated by rendering 10 c.c. of the gastric juice feebly alkaline by the addition of lime water, mixing it with an equal volume of unboiled milk, and placing the mixture in an incubator; if zymogen be present, a dense coagulum will form within eleven to fifteen minutes. The utility of this method of demonstrating rennin zymogen must be questioned, since Fuld has shown that zymogen is not converted into ferment by calcium salts. It is probable that in Boas' method there is simply an acceleration of a weak rennin action by the action of calcium salts.

Boas claims that an approximately quantitative estimate of the rennin action can be made by determining to what degree the gastric juice can be diluted with-

[1] This process, in which insoluble casein is formed from the caseinogen of the milk by the simultaneous action of rennin ferment and calcium salts, must not be confounded with the precipitation of unchanged caseinogen by acids (the curdling of milk by lactic acid fermentation).

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out arresting its function. The gastric juice or contents is first almost completely neutralized and then diluted. A certain amount is added to an equal volume of milk. If coagulation can still be demonstrated, another similar specimen is prepared with still further diluted gastric juice. Normally the dilution may exceed 1:100; but if the secretion is decidedly insufficient, the limit of dilution will be reached at 1:5 or 1:10. The action of rennin and that of pepsin are of about equal importance in the study of the gastric contents. Pepsin and rennin formation usually run a parallel course, although, according to Hammarsten, rennin is different from pepsin. The test for the rennin, however, has the advantage of simplicity, and it can be performed more quickly than that for pepsin. A decided diminution or absence of rennin secretion, like that of the pepsin secretion, is associated only with very grave affections of the gastric mucous membrane. Boas considers that the limit to which the gastric juice can be diluted without inhibiting the rennin action is of important diagnostic and prognostic significance. A complete absence of both rennin and its zymogen and of pepsin is found in carcinoma of the stomach, in atrophic gastric catarrh, and in certain cases of pernicious anemia.

Examination of the Mucous Secretion of the Stomach.

The gastric juice always contains mucus (mucin), in the form of more or less tough transparent or cloudy flakes. If the conditions are normal, these mucous flakes are always very small. They are easily stirred up in the rinse water. Swallowed mucus from the pharynx, esophagus, or bronchi is generally distinctive. It usually occurs in isolated larger lumps and contains more pus corpuscles than the mucus from the stomach. If it is foamy or pigmented (lung pigment), and if it contains no food particles, which are so often intimately mixed with stomach mucus, the distinction is usually easy. The microscope will often solve the origin of the mucous constituents, for frequently in the interior of the mucous flakes a characteristic type of epithelium may be found—*e. g.*, pigmented lung epithelium. True stomach mucus, if the digestion is good, contains only the nuclei of large round or oblong epithelium cells and of leukocytes, because the cell plasma has been destroyed by the digestion. If the digestion is impaired we can often see the cells of the stomach epithelium *in toto*. An increase in the amount of mucus with an abundance of the polymorphous nuclei of leukocytes is characteristic of gastric catarrh. We must be very cautious before assuming the presence of an increased mucous *production*, for an abundant admixture of mucus in the stomach contents quite as frequently depends upon a diminished solution of the mucus in consequence of a diminished production of HCl. Besides, we must remember that the mucus swells considerably if free hydrochloric acid is absent, and so the amount may seem increased. For this reason very noticeable amounts of mucus are found with carcinoma of the stomach. We may finally mention that a quantitative estimation of mucus, which is so frequently advocated, is usually impossible by chemical means. The mucus cannot be precipitated in the filtrate of a test breakfast, because the mucin which is dissolved in a gastric juice that contains hydrochloric acid, or that which is even partly digested, can no longer be precipitated by acetic acid.[1]

The view that mucus is not digestible has been changed by modern investigations.

Examination of Gastric Contents for the Products of Proteid Digestion.

Although it would seem valuable and practical to obtain some insight into the digestive functions of the stomach from an analysis of the chemical changes that the various foods undergo during digestion, yet experience has proved this is not feasible. The processes are too com-

[1] Compare A. Schmidt, "Über die Schleimabsonderung im Magen," *Deutsch. Arch. f. klin. Med.*, 1896, vol. lvii., parts 1 and 2, p. 65.

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plicated, for the changes from proteids to albumoses and peptones depend not only upon chemical reactions, but also to a high degree upon the power of absorption and of motility of the stomach. Because a test meal is rich in albumoses, for instance, we cannot conclude that the digestion is especially good, but rather the contrary, because the albumoses formed are normally either quickly absorbed or passed on into the intestine. An abundance of proteid, therefore, or an increase in the amount of peptone contained in the stomach contents, would indicate (generally speaking) disturbed gastric digestion. But any such deductions could be based only upon an accurate quantitative determination of the proteids of the stomach contents, and as yet we possess no available clinical methods for such a determination.[1]

EXAMINATION OF THE STOMACH CONTENTS FOR GAS FERMENTATION.

The fermentative processes which take place in the stomach when the digestion is affected, especially if there is also a motor insufficiency, have been studied recently, partly by bacteriologic examination and partly by a chemical analysis of the gases which develop in the fermenting gastric contents—*i. e.*, in the expressed test breakfast. These examinations as yet have been productive of no practical results. The existence of fermentation can be demonstrated by placing some of the gastric contents in a fermentation tube (like the one used for estimating sugar in the urine) and letting it stand in an incubator (Fig. 170). If gas is forming it will accumulate above the fluid. If we wish to examine it more closely, we can collect the gas formed from a considerable amount of the stomach contents in a larger vessel by conducting it through a tube and a pneumatic trough into an inverted glass filled with mercury. The gases which are found most frequently in the gastric contents are carbon dioxid and marsh gas, CH_4. The former is easily recognized by its characteristic of clouding baryta water, and the marsh gas, by the peculiar flame with which it burns.

DIAGNOSTIC IMPORTANCE OF THE USE OF RIEGEL'S TEST MEAL.

Concerning the value of Riegel's test meal, it is to be noted that, since from a motor as well as a chemical standpoint it makes greater demands upon the stomach than the Ewald test breakfast, it at the same time acts as a greater stimulus to digestion, and consequently exhibits to better advantage existent anomalies. It can easily come about that a stomach whose motility appears perfectly normal, as indicated by the test breakfast, may, when tested with Riegel's meal, show itself decidedly insufficient in motility by not having completely emptied itself for hours afterward. By the use of this test meal, there are obtained two diametrically opposed results to those given by means of the test breakfast. For example: a chemical insufficiency of the stomach (hyperacidity, lack of enzyme) may be made to appear more plainly by the use of the test meal than by the breakfast; on the other hand, a hyperacidity or hypersecretion that has escaped notice in the test breakfast may become

[1] This subject is treated in *Neumeister's Text-book of Physiologic Chemistry.*

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manifest by the strong stimulation of secretion by the Riegel meal. From these facts it is evident that in stomach affections where the use of the test breakfast does not lead to a clear interpretation, the examination is to be repeated by means of Riegel's test meal.

Concerning the most advantageous time for the siphonage of the Riegel meal, compare p. 368. There unfortunately still exist too few results for us to be able to calculate normal figures for the acid and enzyme contents of the stomach contents, as well as for the volume of the fluid removed by siphonage, at various times after administration. As a rule, it will be found that after four hours the percentage acidity of the Riegel test meal will correspond to that of the Ewald-Boas test breakfast after one hour, and that after seven hours a normal stomach will have easily emptied itself of the Riegel meal.

PROOF OF THE ABSORPTIVE ACTIVITY OF THE STOMACH BY MEANS OF v. MERING'S TEST BREAKFAST.

The method, in principle, is as follows: There is introduced into the stomach a mixture of a sugar solution and a fat-emulsion of known composition. After a definite period this mixture is siphoned off, and the quantitative relation between the fat and the sugar is determined.

This ratio will be changed during the sojourn of the mixture in the stomach only in so far as sugar becomes absorbed, since it can be altered neither by secretion nor by the passage of the contents into the intestine. This allows of the drawing of a conclusion as to the amount of absorbed sugar from the altered ratio of the quantities of sugar and fat.

The practical application of the method is as follows:

There are prepared 300 c.c. of a watery solution containing about 90 gm. of dextrose and 5 to 6 gm. of the yolk of egg. Of this mixture, 250 c.c. are introduced into the empty stomach. After two and a half hours the solution obtained by expression is examined quantitatively for dextrose and fat (ether extract), and from the altered proportion of these two components the sugar absorption is calculated. v. Mering was able to show that, although water was not absorbed from the stomach, considerable quantities of sugar were.

Volhard[1] raised the objection that the method did not give correct results, since the emulsion was destroyed by the splitting of the fat in the stomach.

It is likely that the soup prepared from browned flour, as suggested by the author, would be better adapted for this experiment than is the emulsion (see p. 411).

EXAMINATION OF THE FUNCTIONS OF THE STOMACH BY MEANS OF THE BUTYROMETRIC METHOD OF SAHLI AND SEILER.

It can hardly escape the thoughtful reader how great must be the uncertainty attached to conclusions as to the functions of the stomach which are drawn from examinations of test meals, even those in which the most exact chemical methods are employed. The main difficulty lies in the fact that the results of the quantitative examination of the stomach contents are influenced by many different factors which do not allow of separate calculation. For example, the amount of the expressed meal is not entirely dependent upon the motility of the stomach, but is altered also by the quantity of the secretion. Again, the acidity or the percentage content does not represent directly the amount of the secre-

[1] *Münch. med. Woch.*, 1900, parts 5 and 6.

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tion, but is influenced to a great degree by the motor activity. For the acidity which is present indicates a very different secretory activity according to whether the expressed meal contains besides the secreted gastric juice a greater or less part of the ingested test meal, or according to whether a greater or less part of the secretion has passed, before expression, through the pylorus. If the greater part of the test meal has passed quickly into the intestine during the hour before the expression, then even a small secretion may apparently be normal or even hypernormal; and the reverse is also conceivable—namely, that even a normal secretion may present subnormal acidity, and perhaps even indicate a lack of free hydrochloric acid, in case the food which was introduced has passed only in small amounts into the intestine during the time of the observation. For the percentage content of free hydrochloric acid is influenced not only by the motility of the stomach, but also by the solution and absorption of the proteids in that organ. It is, for example, clear that if the proteids are quickly absorbed, the excess of acid will appear much more quickly than if at the time of the examination all the proteids of the food have been saturated by the acid.

It must be emphasized that, if we determine the presence of a chemical abnormality in the gastric contents according to the older methods, it is not possible to say whether the disturbance is dependent upon the secretion or upon the motility or upon absorption, or upon all these three factors together. If, then, we consider this, it must be admitted that the experiments of Pawlow, who succeeded in collecting pure gastric juice free from the admixture of water and food, were the first to give us exact data concerning the laws of the secretion by the stomach.

From the preceding description of the usual methods of examination it is evident that the apparent accuracy of the results of these methods as applied to the stomach is really a delusion, and that a part of the trouble which has been expended upon them must be considered as actually lost, since it has not been possible to differentiate in the results the separate factors mentioned above. We have to do with an equation containing several unknown quantities which cannot be solved so long as other equations are not at our disposal. Proceeding along similar lines, Pfaundler, in his very interesting work, has brought forth several equations for the chemical process of digestion, and so has found a method of getting more exact information regarding the secretion and motility of the stomach. The essentials of Pfaundler's method consist in removing, during the period of the digestion of the test meal, portions of the stomach contents at different times, either on the same day or with a repetition of the test meal on different days. The analytic data obtained from the examination of these various portions have been used by Pfaundler to prepare equations from which he has been able to calculate, in absolute figures, the motor activity and the amount of the secretion of the stomach. But this method is entirely too complicated for practical purposes, and for the patient is much too troublesome; besides this, it is not entirely free from physiologic objections.

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Principle of the New Method.

In order to obviate, in part at least, the indefiniteness of our examination of the stomach, the writer reasoned as follows: If it is possible to add to the test meal a substance which is not easily absorbed and yet allows of ready quantitative determination, then after introduction and subsequent expression, from the amount of this substance present in siphonage, it can be calculated how much of the test meal has passed into the intestine, how much is still remaining in the stomach, and consequently what fraction of the expressed content can be ascribed to the secretion of gastric juice. One obtains, therefore, a measure not merely of the motor activity of the stomach, but also of the secretion as well. For the success of such a method it is evident that: first, the test meal must be homogeneous throughout, and that in order to serve as an indicator of the motility the unabsorbed substance must remain thoroughly mixed in the stomach contents—*e. g.*, present no inclination to separate or sediment; and, second, that absorption of water must not take place from the stomach. According to the experiments of v. Mering, this latter fact may be considered as definitely settled. Concerning the choice of indicators for the motility of the stomach, the writer thought first to add an insoluble colored powder or lycopodium to the test meal, and to determine this quantitatively by colorimetric means or by counting the lycopodium granules. It was soon shown, however, that neither the insoluble colored particles nor the lycopodium was sufficiently well mixed throughout the test meal to serve as a measure for the amount of the test meal remaining in the stomach. The powders attached themselves so intimately to the solid particles of the test meal that the experiment was abandoned. Moreover, there was still another point to be considered. The substances mixed with the test meal must not give it the character of a non-physiologic food, even if this character consist merely in an abnormality of appearance or taste. On this account the writer was convinced that the only substance which appeared feasible for the purpose in question was fat.

The use of fat in test meals essentially for the sole purpose of the determination of motility had previously been proposed by Matthieu.[1] This author added to a test meal consisting of 60 gm. of bread, 20 to 70 gm. of oil, in the form of an emulsion with gum, and 250 gm. of tea. He then obtained by means of the determination of fat, data concerning the motility and secretion of the stomach, although the latter was merely incidental and was not carried out to any definite conclusion. The writer does not know whether this method of Matthieu has been found to be practicable. In any case, this test meal did not appear to the writer to be available for the point in question, because it did not fulfil the postulate of homogeneity, in that the pieces of bread exhibit a decided inclination to sediment, and thereby mechanically remove the greater part of the fat. In consequence of this the fat, other food constituents, and the secreted juice do not allow of quantitative determina-

[1] *Arch. f. Verdauungskrankh.*, vol. i., 1896.

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tion. Besides this, such a mixture of an emulsion of oil with the ordinary test meal cannot be considered as a usual diet, nor on account of its unpleasant taste is it a test meal of physiologic composition. The experiments of Pawlow show sufficiently how very easily the physical characteristics of the test meal influence the functions of the stomach.

Again, v. Mering, in his method of proving the power of absorption of the stomach, employed fat in the form of an emulsion of the yolk of egg. T. Volhard, nevertheless, has shown that such emulsions of the yolk of egg are not permanent within the stomach; as a result of the influence of the hydrochloric acid the egg yolk separates, and the postulate of homogeneity is consequently not fulfilled.

The attempt was next made to use milk, since this presents a mixture of proteid, carbohydrate, and finely divided fat, which is easily obtainable and nearly ideally constituted for the purposes of nutrition. The experiments with milk resulted unfavorably, in that the coagulation of this substance which took place in the stomach disarranged its homogeneous composition. It was easy to show that in the coagulation the fat was carried down almost completely by the precipitation, and that the coagulum and whey in the expressed test meal of milk contained very different amounts of acid. We were not able by various artificial means to compel the milk to coagulate in fine flocks, and thus to avoid any considerable appearances of sedimentation in the stomach. The employment of milk was consequently also excluded.[1] Finally a soup made of flour browned in fat proved to be adequate for our purpose, and yet completely suitable for ordinary nutrition. At the writer's suggestion, his assistant, Dr. Seiler, tested the feasibility of the employment of this test meal for the purpose in hand, and made a number of examinations, which have been published in his dissertation.

Preliminaries of the Method.

Preparation of the Flour Soup.—This can be very readily prepared by the physician immediately before the examination, and requires only a few minutes' time. The method is as follows: 25 gm. of flour and 15 gm. of butter are fried in an iron pan until well browned; and about 350 c.c. of water are then added gradually with constant stirring. The mixture is then boiled for five minutes, and the loss in volume replaced by fresh water. Salt (sodium chlorid) is now added to give the soup a pleasant flavor. No lumps must remain in the mixture.[2] This soup presents a well-mixed emulsion of fat which, probably because it is mechanically well mixed with the flour, remains intact, in spite of the action of the acid in the stomach, and shows no

[1] A further reason for abstaining from the use of milk was the fact that, in opposition to the results of Schüle (*Zeits. f. klin Med.*, vol. xxviii.), the author was never able to detect free hydrochloric acid in an expressed test meal of milk of 200 to 300 c.c. This difference is attributable to the better quality (fat and proteid content) of the cows' milk used in his laboratory.

[2] Quite a number of the adverse criticisms of this method are due to the fact that this soup has not been prepared with sufficient care in reference to its homogeneity.

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inclination within the time of the examination to form a sediment or for the fat to separate.[1] In fact, at the end of the digestion such a flour soup is expressed in about the same physical condition as it was introduced. It is merely more or less diluted.

The patient takes with a spoon 300 c.c. of this soup, while the remaining 50 c.c. are retained for control determination of the content of fat. The stomach must have been thoroughly washed before administration, and after one hour the contents are expressed.

The next step to be taken is first to determine the absolute amount of fat remaining in the stomach after the test digestion, and, secondly, to compare this amount with that introduced. As it is not possible to be sure that the entire stomach contents are expressed, this determination can be exact only when the amount of the stomach contents which escaped expression is obtained. For this purpose the determination of the residue[2] according to the *method of Matthieu* is resorted to.

The principle of this method consists in the dilution of the residue in the stomach by means of a definite volume of water, which is introduced by means of a tube, and in the expression of the diluted stomach contents after the water has been thoroughly mixed in the stomach by means of light kneading. The acidity of the undiluted, as well as of the diluted, stomach contents is then determined by titration (p. 377), and from the differences in these two values conclusions may be drawn as to the degree of dilution; or, since the amount of water which was added is known, as to the residual amount of stomach contents which was not expressed. The following is the method of calculation according to Matthieu:

Let a = acidity of the undiluted gastric contents.

Let b = acidity of the diluted gastric contents.

Let x = amount of the test-meal remaining in the stomach after expression.

Let 300 c.c. = the amount of water introduced into the stomach for dilution.

Then $ax = b\ (x+300)$.

$$x\ (a-b)\ 300\ b.$$

$$x = \frac{300\ b}{a-b}.$$

For the next step in the procedure it is necessary to have a clinical method for the *determination of the fat* in the fluids to be examined. The usual method, according to Soxhlet (extraction with ether), is too detailed and lengthy to be suited for clinical purposes. On the other hand, the writer has found that the butyrometric method of Gerber[3] for

[1] That sedimentation and separation do not take place even when the flour soup remains quietly in a glass is a further proof that even the slightest peristaltic movements of the stomach cause the homogeneous character of the meal to be maintained, and completely exclude any sedimentation which might be caused by the addition of the gastric juice.

[2] Matthieu and Remond, *Soc. de biol. de Paris*, Dec., 1890, and *Arch. f. Verdauungskrankh.*, vol. i., p. 348.

[3] *Die praktische Milchprüfung*, Bern, K. J. Wyss, 1900.

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fat-determination, which is often employed in the examination of milk, answers all the demands of simplicity and exactness. He will therefore next describe this method of fat-determination, which has not been used before for clinical purposes. The principle is as follows: The solutions in which the fat is to be determined are mixed with a definite amount of concentrated sulphuric acid and a very small volume of amyl alcohol and thoroughly shaken; since the mixture becomes quite hot, the action of the sulphuric acid tends to decompose the organic substances, with the exception of the fat, to such an extent that a thin solution results; from this the total amount of fat dissolved in the amyl alcohol can be separated in the form of a clear layer by means of centrifugalization. The volume of the fat-amyl-alcohol layer is then read off in a butyrometer specially constructed by Gerber. The readings on the graduated scale are calculated as fat per cent. by weight. The butyrometer, made of hard glass, is about 20 cm. long. Its shape may be seen from Fig. 146. The apparatus has the disadvantage that centrifugalization cannot be performed with the ordinary clinical centrifuge, which has too small a radius; this necessitates the employment either of a much larger centrifuge or of one designed especially for this purpose, such as can be obtained from Hugershof, in Leipzig.

Fig. 146.—Butyrometer with rubber stopper.

The butyrometer is filled by means of pipets, also especially designed for the purpose. Of these are required a 10 c.c. (for the sulphuric acid), a 1 c.c. (for the amyl alcohol) and an 11 c.c. (for the milk, the flour soup, or stomach contents).

The process is as follows: 10 c.c. of sulphuric acid (specific gravity 1.820 to 1.825 at 15° C.) (corresponding to 90 to 91 per cent. acid) are added to the butyrometer by means of the pipet, and upon this is stratified 1 c.c. of pure amyl alcohol (specific gravity 0.815 at 15° C.), and then 11 c.c. of the flour soup or stomach contents. The butyrometer is then carefully closed by means of a rubber stopper, and thoroughly shaken. On account of the heat the tube must be covered with a cloth. It is finally centrifugalized, during which process the graduated end of the butyrometer must lie toward the center. By the centrifugalization the fat and alcohol separate from the mixture, and lie as a clear, transparent layer on top of the solution, which is now darkly colored. Centrifugalization is carried on as long as the height of the alcoholic layer increases. In case the operation is not undertaken immediately, the butyrometer must be placed in water at a temperature of at least 70° C., in order to prevent the hardening of the fat. In order to obtain exact results the reading should be made while the fluid

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is still warm. For the purpose of reading, the rubber stopper must be pushed into the mouth of the butyrometer so that the upper surface of the layer of fat (with its under meniscus) comes to lie at the zero point of the scale.[1] The scale (empirically graduated) is then read off at the point corresponding to the lower meniscus of the fat-layer. The centrifugalization should be repeated, particularly by those who are inexperienced in the method, in order to insure the accuracy of the estimation. If the estimation be correct, the second centrifugalization must give exactly the same reading as the first. Gerber found that this method for the determination of fat is exact for milk to about 0.1 per cent. Control experiments which were undertaken at the writer's suggestion, in order to compare the results from this instrument with those from the Soxhlet apparatus, have shown that this degree of accuracy holds good for the flour soup before and after the action of stomach digestion.

The writer must here make reference to the *presence of a lipolytic enzyme in the gastric juice*, which has been of late carefully studied by F. Volhard.[2] The thought presents itself that perhaps in an examination of the digested stomach contents, since a part of the digested fat may have been split in the stomach, the amount of fat after siphonage cannot serve as an indicator for the amount of the residual test meal. The separated glycerin and butyric acid (if butter fat be employed) are both soluble in water, and, if they are not absorbed by the stomach, consequently remain in the watery, instead of in the alcoholic, layer, and thus escape estimation. The action of the enzyme in this connection was studied by the writer's assistant, Dr. Seiler, and his results, recently published in the *Archiv für klinische Medicin* (1904–05), show that the amount of fat decomposed is so slight that it may practically be neglected. The flour soup is expressed after one hour, and Seiler has shown that the lipolysis in this length of time is quite inconsiderable. Furthermore, almost all of the fatty acids contained in butter fat are insoluble in water, so that they pass almost entirely into the alcoholic layer of the butyrometer. The butyric acid, which is soluble in water, is present in such exceedingly small quantities that it may be disregarded. Owing to the relatively small amount of glycerin contained in the fat, it follows that even if there be considerable lipolysis, the loss in the alcoholic layer due to the solution of the glycerin in water is so slight that it is practically unimportant. In other words, the fatty acids liberated by lipolysis in butyrometry may be read off as fat, and the result will be fairly accurate.

Although lipolysis does not interfere with the butyrometric determination, Volhard has shown that if the hydrochloric acid of the gastric

[1] In centrifugalizing it will be found more practical to introduce the rubber cork so that the upper level of the fluid reaches only to about the 80 mark of the butyrometer scale, since the fat accumulates most readily in the conical part between the neck and the body of the butyrometer. By observing this precaution, moreover, the disturbing layer of insoluble substances (cellulose, etc.) collecting below the fat may be kept back in the wide portion of the butyrometer by cautiously bringing the instrument into the vertical position when the rubber cork is pushed in.

[2] *Münch. med. Woch.*, 1900, parts 5 and 6; *Zeit. f. klin. Med.*, vols. xlii. and xliii.

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juice be small in amount, the acidity due to the liberation of the fatty acids by lipolysis may markedly influence the total acidity; it would consequently be incorrect to refer this total acidity entirely to hydrochloric acid. The simplest method for avoiding this error is to employ the filtered gastric contents for the titration of the acidity. With the exception of the quantitatively unimportant butyric acid, the fatty acids are insoluble in water, and so, since they remain upon the filter, the acidity of the filtrate may be referred directly to hydrochloric acid.

It is also to be remarked that it is advisable to determine the amount of fat in the undigested flour soup, as well as the amount in the expressed meal. This can be done without any especial trouble. This is particularly advisable because, on account of the varying content of water in butter, we cannot, by weighing the amount of butter put into the soup, assume the weight of the fat-content.

Next follow the technical conditions which must be insured in the use of Matthieu's determination of the residue contents and Gerber's butyrometric fat-determination in order to be able to draw exact conclusions concerning gastric digestion.

Performance of the Method.

After a thorough rinsing of the stomach the patient is given in the morning the 300 gm. of flour soup, prepared according to the method already described. It must be eaten with a spoon. The remaining 50 c.c. are retained for the determination of fat. After one hour the contents are expressed, and 300 c.c. of water introduced for the purpose of the determination of the residue. The undiluted stomach contents and those expressed after dilution are titrated for their acidity, and from these two results the amount of stomach contents remaining after the first expression, or the residue, is calculated according to the formula of Matthieu. Next, the amount of fat in the undiluted stomach contents and also in that portion of the soup which was retained as a control is determined butyrometrically according to the method given above. The undiluted stomach contents still remaining can be used for whatever qualitative or quantitative determinations may be deemed advisable. Qualitative examinations in this case normally show the same results as in the use of the ordinary test meal. Free hydrochloric acid should be present; lactic acid should not. The flour-soup meal is very well suited for the determination of the pathologic formation of lactic acid, because it is free from this substance. The titrations are best carried out upon unfiltered gastric contents (compare p. 377). Besides the total acidity, the excess or deficit of acid may also be titrated. If the qualitative reaction shows considerable amounts of lactic acid, the determination of the hydrochloric acid must be made by Lütke-Martius' method, or that of the hydrochloric acid and the organic acids by Hehner-Maly's. Of course, all other advisable examinations can also be made upon the expressed gastric contents—namely, the qualitative and quantitative tests for pepsin and rennin (pp. 391 and 394) and the ordinary tests for starch digestion (p. 371).

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Calculation of the Results.

The following calculations are possible from a consideration of the residue, from the acidity of the gastric filtrate, and from the difference between the amount of the fat found in the ingested flour soup and that found in the expressed contents.

By the addition of the value x, found in the calculation of the residue (p. 401), to the amount of contents expressed after one hour, there is obtained the volume of the contents which were actually present in the stomach at the end of that period. This we designate as *To* (a total gastric contents after one hour). From the absolute fat-content of *To* found butyrometrically, there can be determined how much of the volume *To* can be ascribed to the ingested flour soup; provided the expressed contents present a thoroughly mixed emulsion whose homogeneity has suffered no disturbance. Since the results of v. Mering's investigations prove that absorption of water from the stomach plays no considerable rôle, and since the absorption of fat in the stomach may also be neglected, the percentage relation of the fat to the stomach contents is disturbed neither by the motility of the stomach nor by the absorption of water, but only by the secretion of gastric juice. The amount of fat remaining in the stomach serves, therefore, as a measure for the amount of flour soup also remaining;[1] for example, if 300 c.c. of flour soup with a content of 4 per cent. fat, or altogether 12 gm. of fat, were introduced into the stomach, and the determination of fat for *To* showed a fat-content of 3 gm., we can conclude that the amount of ingested soup which remained at the end of the period was equal to $\frac{3}{12} \times 300 = \frac{1}{4} \times 300$—*i. e.*, 75 c.c. We designate this amount as *Su* (soup).

Since we can neglect the absorption of water and the amount of the saliva which was swallowed, the volume *To* — *Su* is equivalent to the volume of gastric juice present in the expressed contents. This we designate as *Ma* (gastric juice). For example, if the amount *To* = 150 c.c., and the amount *Su* = 75 c.c., then 150 — 75, or 75 c.c., will be the volume of gastric juice contained in *To*. If the acid content of *To* has been determined, it is possible from these data to proceed further, and to calculate what acidity was possessed by the pure gastric juice as it was excreted from the mucous lining of the stomach.[2]

If 75 c.c. of pure gastric juice are present in the stomach contents whose volume amounts to 150 c.c., with 2 per cent. acidity, then the acid content of the pure gastric juice (*Ma*) must evidently be 4 per cent. In a similar way the enzyme content of the pure gastric juice can be calculated if it has been quantitatively determined for the mixed stomach contents.

In order to avoid misunderstandings in regard to the value of all these calculations, attention must be called to the following points:

[1] The saliva which has been swallowed can be very well neglected.

[2] In the content of this secretion is included any exceptional osmosis caused by the ingestion of the flour test meal.

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The flour soup contains such small quantities of soluble carbohydrates and salts that such osmotic effects can be in evidence only when the absorption of the soluble products of the digestion of the flour soup is interfered with.

Naturally the volume *Ma* is not identical with the total volume of gastric juice which has been secreted up to the time of expression. An unknown amount of the gastric juice mixed with the food has been passed on into the duodenum before expression. Nevertheless the calculation gives interesting results as to the volume of the secretion independent of the percentage acidity of the pure gastric juice, since it shows us the relation of the amount of the gastric juice present in the stomach at a given time to the amount of test meal present in the stomach at the same time. As later examples will show, this relation is approximately constant under normal conditions. The gastric juice and flour soup are contained in the volume *To* in approximately equal parts. From this it is evident that variations in this relation are of pathologic and diagnostic interest.

Concerning the conclusions as to the motility of the stomach which may be drawn from these results, the following is to be noted: The difference between the volume of soup introduced and that recovered by expression (namely, the volume $300 - Su$) can be considered as a measure of the motility of the stomach. Really this value does not correspond to the total motor power of the stomach, for an unknown amount of secreted gastric juice has also been passed on into the duodenum. At the same time it is probably a more reliable measure of stomach motility than the value of the total stomach contents, which is used in the older methods for the same purpose. For, as already mentioned, the expressed contents contain under normal conditions about equal amounts of soup and gastric juice, so that it is easy to judge, when variations from this normal occur, whether a hypersecretion or diminished secretion is present. This gives the actual value of the motility already mentioned.

The calculations from the results of the examination can be presented algebraically as follows:

Let To = the amount of the expressed contents, including residue. (For calculation of the latter compare p. 401.)

F = the fat-content of the flour soup in per cent.

f = the fat-content of the expressed contents in per cent.

Su = the amount of soup contained in the expressed contents.

$$\text{Then,} \quad \frac{Su}{To} = \frac{f}{F}$$

$$Su = \frac{f \,.\, To}{F}$$

From this value the amount of gastric juice may be obtained from the formula $Ma = To - Su$.

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The determination of the acidity of the pure secretion may be calculated as follows:

Let a = the acidity of the expressed gastric contents in per cent.
A = the acidity of the pure secretion in per cent.
To = the amount of the expressed contents (including the residue).
Ma = the amount of secretion contained in the expressed contents.

$$\text{Then,} \quad \frac{a}{A} = \frac{M}{To} \text{ and } A = \frac{a\,To}{Ma}$$

By a similar formula the enzyme content of the pure secretion can be calculated quantitatively.

The Objections to this Method and the Improvements in the Procedure.

Almost immediately upon the publication of the method just described, its accuracy was attacked by various authors who had not sufficiently tested it. The chief objection made was that the mixture of the test meal and gastric juice separated into layers within the stomach, so that a homogeneous mixture of the fats and other contents did not occur, and that consequently the fat in the expressed contents could not be taken as an indicator for the test meal left in the stomach. This objection was based upon the observation that if the test meal be expressed in two portions, one corresponding to the upper, and the other to the lower, layer, there will frequently be a difference in the amount of fat contained in the two cases. This observation was founded upon fact. A complete homogeneity of the gastric contents is not usually present after the introduction of the test meal. At the writer's suggestion this question has been carefully studied by Dr. Seiler, who published his results in a recent article.[1] Dr. Seiler found that these differences were not great (0.15–0.25 per cent. fat as an average), and that they were not due to a separation in layers by the influence of gravity, but to the fact that the gastric mucus never mixes homogeneously with the gastric contents, and that the gastric juice does not do so immediately. The amount of fat in the expressed contents will consequently depend upon the location of the tip of the stomach tube. On the other hand, even a moderate gastric peristalsis seems able to prevent a separation into layers by the action of gravity. The basis for these statements is given in Seiler's article, to which the reader is referred. The same work also shows that these differences in the quantities of fat contained in different portions of the gastric contents may be diminished, if we make use of gravity, by causing the patient to change his position every five minutes during the length of time the test meal remains in the stomach.

From a consideration of these facts, the following modifications in the practical application of the method are advised in order to secure the most accurate results:

1. Every five minutes during the time of the test the patient must change his position, varying between the sitting, the dorsal, and the left lateral position. By this means the gastric peristalsis is markedly aided in producing a homogeneous mixture. The right lateral position is to be avoided, since it might mechanically aid the escape of some of the gastric contents.

2. In order to gain an idea of the homogeneity of the gastric contents, it should be expressed in two portions, the first being obtained before a deep introduction of the tube, and representing the upper layer, and the second corresponding to the lower layer. The first portion should be smaller in volume, so that any existing difference between its composition and that of the lower layer will be the more striking.

3. Only those portions of each layer which are free from mucus should be

[1] *Deutsch. Arch. f. klin. Med.*, 1904–05.

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employed for butyrometry. If collections of mucus are visible, they should be removed with forceps.

4. As a rule, a mean may be taken from the results of the two tests. This will be the more accurate the less the difference between the fat-percentages obtained in the two tests.

5. If the butyrometric results of the two tests vary so much that they are open to suspicion, the results should be interpreted only like those of the older and less accurate tests.

6. In reference to the accuracy of taking the mean of the two tests, the following fact should be borne in mind: The same difference in the fat-percentages of the two layers has quite a different significance in the calculation, according to whether the expressed contents contain a high or a low fat-percentage. For example, if the two layers contain 2.3 and 2.6 per cent. of fat, a difference of 0.3 per cent., this is so slight (one-eighth to one-ninth of the total amount of fat) that the average may be taken without question. Should we find fat-percentages of 0.9 and 1.2, however, we again have a difference of 0.3 per cent., but this time it amounts to one-third to one-half of the total amount of fat, and the average percentage is subject to criticism.

Normal Results with the Use of This Method as a Foundation for Interpretation of Pathologic Results. Examples for Diagnostic Use and the Value of the Method.

As normal results, the following figures found by Dr. Seiler by experiments upon normal individuals may be given:

Number.	Amount of soup introduced.	Total stomach contents, including the residue, after 1 hour. *To.*	Content of soup in the stomach contents. *Su.*	Contents of gastric juice in the stomach contents. *Ma.*	Acidity of the pure juice. Per cent. HCl.
1.	300	124	72	52	3.5
2.	300	158	83	75	4.4
3.	300	57	30	27	3.5
Average	. . . .	113	62	51	3.6
4.	250	113	44	69	3.2
5.	250	108	56	52	4.2
Average	. . . .	110	50	60	3.7

If we consider 300 c.c. of flour soup as the standard amount ingested, then we can calculate from these and other experiments the average value of *To* after the expression in one hour to be a little more than 100 c.c. This volume is divided normally into approximately equal parts of secretion and soup. Nevertheless it is the common experience with the ordinary test meal that the amount of the expressed contents varies greatly even under apparently physiologic conditions, so that only considerable variations from the normal are to be considered pathologic. It is to be noticed that the relation of the amount of secretion to that of soup is far more constant under normal conditions than the amount of *To*. Free hydrochloric acid should, as already mentioned, always be present. The acidity for the normal pure gastric juice varies between 3.2 and 4.4 per cent. These figures stand between those which Pawlow obtained for the normal gastric juice of the dog (5 per cent.) and those

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which Schüle and Troller found in human gastric juice, which they caused to be secreted reflexly by chewing certain substances (1.8 to 3.6 per cent.). That the values of Troller and Schüle are upon the whole less than the author's is explained by the fact that the mere chewing of substances which cannot be considered as food (citron rind, mustard) cause a slighter secretion than an actual meal.

It is perhaps best to make the diagnosis of *hyper-* or *hypo-acidity* depend, not upon the acidity of the total expressed gastric contents, but upon the hydrochloric acid content of the pure gastric juice. Values which vary greatly from normal figures may then be considered as expressions of a hyper- or hypo-acidity. It may perhaps also be best to speak of *hyper- and hyposecretion* if the volume of gastric juice amounts to considerably more or less than that given above as normal. As a basis for this estimate we should not take the absolute amount of gastric juice found in the stomach after an hour, but, better, the amount of gastric juice in proportion to that of the soup still present in the stomach. It is this relative amount which is of value for judging the secretory power of the stomach, because, while at every moment a mixture of gastric juice and soup is being removed into the intestine, still the relative amount of soup and gastric juice expression is not influenced by the motility. Seiler is quite right to call this relation of the volume of secretion to that of the flour soup remaining in the stomach the secretion quotient. This is normally not far from 1. Considerable variations above 1 would argue for the diagnosis of hypersecretion; below 1, for the diagnosis of hyposecretion.

The motility of the stomach may then be said to be normal if, after the ingestion of 300 c.c. of meal soup, not more than 100 to 150 c.c. can be expressed at the close of one hour, of which about one-half consists of soup. Therefore, the ratio of the amount of juice to that of soup recovered, as expressed by the secretion quotient, must be considered in the estimation of the motility. For example, if the amount of soup remaining is absolutely normal, but a relatively greater amount of juice is found, this amount of soup points not to a normal, but rather to a hypernormal motility. So a normal amount of soup with diminished secretion points to a decreased motility. The ratio of the amount of soup removed during one hour from the stomach into the intestine to the amount of soup ingested, $\frac{300 - Su}{300}$, may be called the motility quotient. This varies normally between three-quarters and nine-tenths, and must be interpreted, as has been said, by the consideration of the secretion quotient.

It is evident from the above that it is possible to discover by this method a number of conditions which must have completely escaped the older methods of investigation. By it, it is possible to decide whether an increase in the amount of the expressed stomach contents points to a hypersecretion or to a motor disturbance. It is also possible to discriminate more exactly than before between hypersecretion and hyperacidity, since the volume of secretion, as well as the acidity of the pure

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gastric juice, can be calculated. Experience with this method has showed us already that stomach disturbances are decidedly more complicated than had heretofore been imagined. The more exact differentiations it enables us to make will in the future increase our therapeutic possibilities. It has shown us, for example, that there are cases of hypersecretion with hypo-acidity, and cases of hyposecretion with hyperacidity. This condition is possibly due to an abnormal watery osmosis, to which we have previously referred, and which we have traced to an insufficient absorption of the soluble products of digestion (see p. 406, footnote). These conditions by the old methods gave apparently normal results, because in such cases the amount of secretion and the acidity of the gastric juice in mixed gastric contents completely neutralized each other in the determination of the total acidity. To these belong in greater part those cases diagnosed usually as sensory neuroses, in which, in spite of disturbances of digestion, the examination of the stomach contents by means of the usual methods produces completely normal results. A case which appeared remarkable to the writer gave the following results: With otherwise normal conditions (motility and the amount of secretion), the acidity calculated for the pure gastric juice was abnormally low, while the contents expressed from a fasting stomach showed a large amount of decidedly hyperacid gastric juice. The condition here can evidently be explained only by the fact that the introduction of food into the stomach caused an inhibition of the secretion instead of stimulation. That the present simple and insecurely founded classification of the symptomatology of the diseases of the stomach into increased or decreased secretion and excessive or diminished motility must suffer decided alteration may be shown by the following scheme of some functional diagnoses which we are in the position to make by means of the new method:

A. Cases with Sufficient Motility.

1. Hypersecretion with hyperacidity and hypermotility.
2. Hypersecretion with hypo-acidity and normal motility.
3. Normal amount of secretion with hypo-acidity and hypermotility.
4. Hyposecretion, hyperacidity, and normal motility.
5. Hyposecretion, hypo-acidity, and normal or increased motility.

B. Cases with Insufficient Motility.

6. Diminished motility, hypo-acidity, and hyposecretion.
7. Diminished motility, nearly anacidity, and hypersecretion.

More extended experience will certainly tend to give still more combinations.

From the preceding recapitulation it is evident how poorly founded is the attempt of certain authors to ascribe the conditions of hyperacidity and hypersecretion to primary motor disturbances, even narrowing the cause to pyloric stenosis, a view which has often led to gastroenterostomy in cases where such an operation was not at all justifiable. These more exact functional diagnoses in stomach disturbances are

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important also in a therapeutic connection. Above all, they indicate clearly whether it is best to influence the motility (laxatives, regular expression of stomach contents) or to affect the secretion (belladonna preparations, alkalies, by employment of means for stimulating the secretion directly by means of the appetite, according to Pawlow [meat extract, bitters]).

Further Value of the Butyrometric Method of Examination as Applied to the Testing of Amylolysis, Absorption of Carbohydrates, Proteolysis, and Absorption of Proteids by the Stomach.

What has already been said concerning the advantages of the butyrometric method of the examination of the stomach contents by means of the flour-soup meal has not exhausted the possibilities of this method. It is well suited, for instance, to the quantitative testing of the digestion of starch. In fact, the following method is much more exact than those previously employed, although it must be confessed that it has not been as yet practically worked out. The expressed stomach contents, as well as the undigested flour soup which was retained as a control, are examined butyrometrically for their fat-content. Then equal volumes of both of these fluids are measured off, each thrown upon a separate filter, and washed with water so long as the filtrate still shows a starch or sugar reaction. This is to remove the soluble products of the starch digestion. In the residue on the filter in each case the starch content relatively to the content of the insoluble carbohydrate is determined, and the difference in the two amounts calculated for the unit of the fat-content shows how much of the starch has been transformed in the stomach into soluble carbohydrates—*i. e.*, has been digested.

The soup meal can again be employed for the determination of the power of absorption of the gastric mucosa for soluble carbohydrates. Equal amounts of the expressed and retained flour soup are hydrolyzed, and in each the sugar tested quantitatively. The amount of fat in the two fluids must also be determined butyrometrically. The content of sugar which is found in the expressed contents serves as a measure of the total soluble and insoluble carbohydrates which were contained in the stomach and not absorbed. If we therefore subtract this amount, calculated for the unit of fat-content, from the sugar of the undigested flour soup, expressed in the same way, there will be obtained the amount of the absorbed carbohydrate in terms of the unit of fat-content.

In order to understand this value correctly it must be emphasized that the carbohydrates which are introduced by this method into the stomach do not for the most part occur in a preformed absorbable form, so that the power of absorption, as found, appears at the same time as a function of the digestion of carbohydrates. If one wishes to determine the amount of the absorption by itself, as by the v. Mering method of determination, it must be considered how much soluble carbohydrate is contained in the ingested flour soup and how much of carbohydrate was transformed into a soluble condition during the digestion.

The interpretation of the relations of carbohydrate digestion and absorption may be shown in the following way :

Let C_1 = the carbohydrate of the undigested flour soup.

Let C_2 = the carbohydrate of the undigested flour soup minus the soluble carbohydrate.

Let C_3 = the carbohydrate of the expressed-meal soup.

Let C_4 = the carbohydrate of the expressed-meal soup minus the soluble carbohydrate. Then $C_2 - C_4$ = a measure of the carbohydrate that has been digested—*i. e.*, rendered soluble.

And $C_1 - C_3$ = a measure of the absorbed carbohydrate.

$$\frac{C_1 - C_3}{(C_1 - C_2) + (C_2 - C_4)} = \frac{\text{carbohydrate absorbed}}{\text{carbohydrate originally soluble and that dissolved}} =$$

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the absorbed fractions of the carbohydrate originally soluble and dissolved during the digestion.

This latter fraction, in an analogous manner to the secretion and motility quotient, may be called the absorption quotient of the carbohydrate. It will give us data analogous to those obtained by the method of v. Mering. It has the advantage over his method that the observation takes place under physiologic conditions—namely, with a test meal physiologically constituted and palatable, while by his method the examination occurs under conditions which are somewhat abnormal.

Finally, the butyrometric determination can be used to test the digestion and absorption of proteids under natural conditions. Here also the method must be more thoroughly worked out technically, but the plan of the examination appears to be clearly outlined. The process is entirely analogous to that for testing the carbohydrate digestion. After the fat in the undigested flour soup, as well as in the expressed stomach contents, has been determined, each fluid is filtered, and washed free from soluble proteids with water. In all four tests the nitrogen content as determined by the Kjeldahl method serves as a measure of the total proteids, including proteoses and peptones. All of these values must be calculated for the unit of fat-content.

Let N_1 = the nitrogen content of the undigested flour soup.

Let N_2 = the nitrogen content of the undigested flour soup minus the nitrogen of the soluble proteids.

Let N_3 = the nitrogen content of the expressed stomach contests.

Let N_4 = the nitrogen content of the expressed stomach contents minus the nitrogen of soluble proteids.

Just as in the calculation of the carbohydrate digestion, $N_2 - N_4$ = a measure of the proteids digested—*i. e.*, of the nitrogen which has become soluble.

$N_1 - N_3$ = a measure of the proteid—*i. e.*, nitrogen absorbed.

$$\frac{N_1 - N_3}{(N_1 - N_2) + (N_3 - N_4)} = \frac{\text{absorbed } N}{N \text{ soluble and that dissolved}} =$$

the measure of that part of the soluble nitrogen, both that preformed and that made soluble by digestion, which was absorbed. This may be called the *absorption quotient* of the proteids.

Concerning the Technic of the Necessary Determination of Carbohydrate in These Examinations.—This determination can be carried out accurately only if the carbohydrate is completely transformed into dextrose and as such determined. For this purpose, according to the method of Pollitz, 20 c.c. of the stomach contents or flour soup are treated with dilute sulphuric acid until the appearance of the positive Congo-red reaction, then further diluted with 40 to 50 c.c. of dilute sulphuric acid (5 c.c. of dilute sulphuric acid, specific gravity 1160, diluted up to 1 liter with water), and the mixture heated for eight hours at 108° to 110° C. in a Lintner pressure flask upon a paraffin bath, until a color is no longer obtained when iodin is added to a small portion.

The task of determining sugar in a fluid of this kind is not very simple, on account of the content of proteid and albumose. In order to obtain an exact determination the proteids must be completely removed; this is true of the polarization test (p. 515) or of the Soxhlet-Allihn methods with modifications. The polarimetric method is inaccurate, because the proteids cause rotation to the left; and the reduction tests, because the proteids hinder the filtration of the suboxid formed. We usually have not enough material for the areometric fermentation test. After many experiments the writer has found that the removal of the proteids, as well as the albumoses and peptones, can be best accomplished by the filtration of the mixture, thorough washing with water, and final treatment of the filtrate and washings with a solution of phosphotungstic acid[1] until no further precipitation occurs. The solution must not contain hydrochloric acid. We

[1] One part of phosphotungstic acid diluted with 20 parts of water and acidified with sulphuric acid.

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cannot, therefore, employ hydrochloric acid either for the hydrolysis or for the acidification of the phosphotungstic acid solution.

After the precipitation of the proteids, the phosphotungstic acid must be removed from the filtrate.[1] This is best accomplished by adding barium carbonate so long as an effervescence takes place. The filtrate from this precipitate should be neutral and free from proteids, and can be employed for the determinations of sugar according to Soxhlet-Allihn, colorimetric, or iodometric methods.

Where it is simply a question of the determination of the insoluble starch in the expressed meal in comparison with that in the ingested flour meal, the starch can be approximately determined colorimetrically as iodostarch according to the usual method of Ambuhl.[2] But since the stomach contents always contain erythrodextrin in soluble condition, and since this substance, when iodin is added to the expressed meal, causes a dirty brownish-violet color instead of a pure blue, the colorimetric determination of the starch must be undertaken after the soluble starch derivatives, especially the erythrodextrin, have been removed. This can best be accomplished by repeatedly washing the expressed contents with small amounts of water in the centrifuge. It must be mentioned that the exact separation of the solid from the soluble constituents by means of the centrifuge is successful only when the expressed contents have been extracted with five volumes of ether for the separation of the fat (by means of the separatory funnel, p. 375). One cubic centimeter of the undigested and of the expressed flour soup are exactly measured off by means of a pipet divided into $\frac{1}{100}$ c.c., washed with water, and then with ether until a test of the substance remaining shows a pure blue color. The remaining part is boiled for ten minutes with about 500 c.c. of water, during which the greater part of the starch passes into solution. After cooling, diluted Lugol's solution is added drop by drop to both tests until the blue color does not become more intense. The shades of color of the two fluids are then compared colorimetrically, and it is determined in a measuring cylinder how much the darker fluid must be diluted in order to correspond exactly to the shade of the other. The rates of dilution necessary for this purpose gives directly the ratio of the amount of starch in the ingested to that in the expressed meal.

Special Examination of the Stomach for "Raw Motility."

Those cases in which the ability of the stomach to empty itself is impaired, and where pyloric stenosis is not present, are frequently spoken of as insufficiency or motor weakness of the stomach. We have learned from v. Mering and Marbaix that the emptying of the stomach is regulated by the intestine rather than by the stomach itself, since nutritive substances reaching the intestine effect a reflex closure of the pylorus (v. Mering's reflex) until the intestine has completed its work. It consequently seems to the writer to be inadmissible to regard the emptying of the stomach as a pure question of strength, as would be suggested by the term insufficiency or motor weakness of the stomach, since even a well-developed and efficient stomach does not empty itself if it is opposed by the intestine. Actual weakness of the stomach nevertheless does occur, and in order to differentiate it from the more frequent forms of disturbed motility which proceed from the intestine, a special method of examination is necessary, which the writer will now give, and which he has carefully tested in recent years. The motor activity of the stomach must be examined under conditions in which v. Mering's reflex does not occur. As v. Mering and Marbaix have shown, such a condition is present when the stomach contains water alone, since, under physiologic conditions without obstruction, the water is emptied into the intestine within a short time. We must consequently determine the length of time required by the stomach to empty itself of a definite quantity of water—a half liter, for example.

Since we have to do with stomachs which do not empty themselves well, the

[1] It is only in the determination by means of the polariscope, which for this purpose is hardly accurate enough, that the phosphotungstic acid need not be separated, since it has no effect on the plane of polarized light.

[2] *Cf. Schweiz, Lebensmittelbuch*, Bern, Verlag v. Neukomm and Zimmermann, 1899.

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test must be preceded by a thorough cleansing of the organ. The patient then drinks the prescribed quantity of water, the gastric contents are then expressed in a half-hour, and the quantity passed onward into the intestine is thus determined. Since it is usually impossible to empty the stomach completely, we must first determine how much water is left behind from the cleansing irrigation, and add this to the quantity drunk by the patient, if correct results are to be obtained. After the expression of the gastric fluid we must also determine how much has escaped expression. These facts are best determined colorimetrically in the following way: After the cleansing irrigation, about 50 c.c. of a distinctly colored solution of methylene-blue are introduced into the stomach through the tube, the stomach is gently massaged, and a small portion of the contents are expressed. The mixture removed will be pale blue in color. Another 50 c.c. of the same solution are now diluted with water until the same shade of pale blue is obtained; the amount of water added must obviously be the same as that left in the stomach by the cleansing irrigation. A half-hour after the introduction of the 50 c.c. the fluid is expressed, and the residue determined as before. After we have expressed all the fluid possible (fluid *a*), 100 c.c. of water are introduced into the stomach, the stomach massaged slightly, and the water again expressed (fluid *b*). We now determine how much of the fluid *a* must be added to 100 c.c. of water in order to obtain the same shade as that of the fluid last expressed (fluid *b*). The required amount of the fluid *a* will equal the quantity of the residue in the stomach which could not be expressed. The entire amount in the stomach at the end of the half-hour will be found by adding this residue to the expressed fluid *a*. In case the expressed fluid *a* is no longer colored, the residue escaping expression may be directly determined, as in the first instance, by adding some of the original solution of methylene-blue.

This test for "raw motility" must be carried out with the patient in the upright position, since gravity interferes too much with the expression if the patient be lying down.

Special Examination of the Stomach for Pyloric Stenosis.

Pyloric stenosis is diagnosed much too frequently nowadays when the stomach fails to empty itself properly. In reality the case may be simply a functional disturbance of v. Mering's reflex or a condition of motor weakness. Moritz has shown that the stomach is an excellent sedimenting apparatus, and that in all forms of impaired motility firm, indigestible substances (fruit kernels and other vegetable constituents) may remain in it for days, since they are never elevated to the level of the pylorus on account of their high specific gravity. We are consequently not justified in diagnosing a pyloric stenosis offhand from the presence of such old substances in the retained gastric contents. A little reflection will also show that the retention of indigestible substances of a specific gravity low enough to enable them to float may be utilized to diagnose a pyloric stenosis the caliber of which is not sufficient to allow of their passage. This is the basis of the procedure the writer recommends, by which it is possible to diagnose the existence and caliber of a pyloric stenosis by the administration of little balls of cork. The patient to be examined for pyloric stenosis is made to swallow a ball of cork 1 cm. in diameter. If the cork ball be found in the stool the following day, the pylorus must be large enough to have allowed it to pass. On account of its low specific gravity, the cork ball may easily be found, since it readily floats when water is added to the stool. Certain precautions must be observed, however, in carrying out this test. After swallowing the cork the patient should lie in bed for a half-day, preferably upon the left side, so that the ball may be floated upward into the neighborhood of the pylorus. If the stomach is markedly loop-like, it may be sufficient to have the patient lie upon the left side for a short time, under the supposition that the cork ball is immediately floated toward the pylorus and remains there. The writer has not yet employed cork balls larger than 1 cm. in diameter, for fear they might stick in the esophagus. To what extent the caliber of the ball may be increased remains a subject for further study.

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EXAMINATION OF THE INTESTINE AND FECES.

INSPECTION, palpation, auscultation, and percussion of the abdomen, and their help in the examination of the intestine, have already been considered upon pages 301 *et seq.*, 304 *et seq.*, 286 *et seq.*, 196 *et seq.*, and 215 *et seq.*

Hence, we will now discuss only the local examination of the rectum (digital and speculum examination), the testing of the intestinal functions, and the examination of the feces.

LOCAL EXAMINATION OF THE RECTUM.

DIGITAL EXAMINATION OF THE RECTUM.

The examiner ordinarily employs the index finger, well lubricated, and, of course, with a carefully cut nail. The patient may be in the dorsal decubitus with the legs widely separated, resting upon the small of the back (the latter is advisable, so that the examiner's elbow shall not prevent his palpating the anterior rectal wall). If, as is sometimes more convenient, we examine with the patient in the lateral position, the knees should be well drawn up to allow room for the examiner's arm in all directions. The knee-chest position, which has recently been so frequently employed by the gynecologists, is also to be recommended, since it displaces the intestinal coils upward and facilitates the palpation of the pelvic viscera. For different purposes and under different conditions these positions can be varied and the examiner's hand changed; the important thing is that the entire circumference of the rectum must be palpated, and we must be able to reach up with the finger as far as possible. The anal opening should be inspected before performing the digital examination, as we can thus often recognize the presence of hemorrhoids, prolapsus, fistula in ano, or fissures.

The finger should be introduced slowly and carefully with a gentle screw-like motion, so as not to cause pain or injury. Beginners often make the mistake of not following the axis of the rectum after introducing the finger, especially when they try to palpate as high up as possible. They will then fail to reach up very far, and so may even believe that they have found a stenosis, though none is present. The anatomic position of the rectum should be remembered and this error will be prevented. The direction is first a little forward from the anal opening, then backward into the hollow of the sacrum, and finally to the left toward the sigmoid flexure. While advancing the finger we must remember to keep it always in the middle of the funnel formed by the mucous membrane. If feces are present they will be the best guide as to the direction in which to advance the finger. The height to which the palpating finger may reach depends upon various factors; thick, short fingers on the part of the examining physician, or much fat on the part of the patient, are the chief obstacles. Sometimes the pain of the

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examination or the spasmodic contraction of the sphincter will prevent an effective examination. Only in exceptional cases is it allowable to introduce two fingers. The introduction of the whole hand under anesthesia (after Simon) has, so far as we know, been discarded as too dangerous a method.

Digital examination reveals first of all the more crude anatomic changes—carcinoma of the rectum, other tumors and ulcerations of the rectum, rectal polyps which are not situated too high up, an invagination which has reached down as far as the rectum. Large internal hemorrhoidal masses may also be felt; but unless thrombosed it requires considerable dexterity to differentiate them with certainty from the normal inequalities of the rectal mucous membrane. Superficial ulcerative processes are quite as difficult to appreciate by palpation.

In a case of intestinal obstruction, *considerable distention of the rectum* points strongly to fecal rather than some other type of intestinal obstruction. If fecal masses have pushed down into the rectum and the general symptoms of obstruction have improved, our prognosis immediately becomes more favorable, even before any feces have been evacuated. An extremely painful digital examination would suggest inflammatory changes of the rectum. If slimy, purulent, or bloody masses adhere to the finger when withdrawn, this diagnosis is still more strongly suggested. A microscopic examination of the material adhering to the withdrawn finger will sometimes aid in the diagnosis of tubercular or dysenteric changes, and perhaps be more serviceable than the examination of the feces, especially if the rectum has been just cleaned of fecal matter by an enema (pus, tubercle bacilli, amebæ). (Compare Examination of Feces, p. 427.)

A careful rectal examination is important in diseases of the nervous system. The amount of distention of the rectum, the sphincter tonus, and the sensitiveness of the rectal mucous membrane tell of the condition of the rectal activity (function). This information is the more important, as the character of defecation and the shape of the stools depend not only upon the rectum, but upon the influence of the bowel above.

In examining the rectum we must also try to obtain information regarding the condition of the neighboring organs—the prostate and seminal vesicles in the male, the uterus, tubes, and ovaries in the female. A careful rectal examination will generally obviate the necessity of a vaginal examination in a virgin.

EXAMINATION WITH THE SPECULUM.

With the aid of a so-called rectal speculum the examiner can see the inner surface of the rectum directly. Rectal specula are made quite like vaginal specula—tubular, bivalve or duck-bill-shaped, or many valved. They may also consist of two separate spoons. The speculum should first be warmed and well oiled, and then carefully introduced into the axis of the rectum. With an ordinary head mirror a good light can be reflected, and the rectal mucous membrane at the end of the speculum carefully examined. The position of the patient will be varied according to the part of the rectum to be examined, either the lateral, the usual dorsal gynecologic position, or sometimes the knee-chest position. The advantage of the knee-chest position is that the rectum is relieved by the intes-

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tines dropping forward, and so is more accessible to examination. In difficult cases the speculum examination should always be tried in this position. The speculum should be introduced very carefully and slowly. The many-valved specula must be furnished with an obturator before being introduced, for otherwise they easily pinch and injure the mucous membrane.

Examination with a speculum is difficult on account of the limitation of the field of vision, and the fact that the parts are usually in an abnormal position and under abnormal tension. The results are therefore rather meager. Peristalsis of the rectum is often caused by the irritation of the speculum, especially in inflammatory affections, so that it is very difficult to adjust the instrument. Palpation reveals many of the changes, and others (inflammatory, superficial ulcers) are just the most difficult to recognize with a speculum, for the pressure of the instrument causes considerable congestion, altering the color, and in the case of ulcers oftentimes brings on hemorrhage, which covers the field of vision again and again even if sponged off continuously. The bivalve duck-billed speculum usually affords a better view, but its employment always requires the aid of an assistant.

A detailed description of the possibilities of speculum examination of the rectum by means of a complicated "Rectoscope" provided with electric illuminating appliances may be found in the recent monograph of J. Schreiber, *Die Rectoromanoskopie*, Berlin, Hirschwald, 1903.

INSUFFLATION OF THE RECTUM.

Insufflation of the rectum is best accomplished, as described on page 306, with a Davidson syringe. It aids in determining the position of the sigmoid flexure and of the colon. When inflated they can be seen to stand out against the anterior abdominal wall, separate from the surrounding parts, and may be mapped out by percussion and palpation. The position of abnormal tumors, especially in relation to their origin in the colon, the sigmoid flexure, or the kidney (compare p. 310), can often be determined by the aid of this method. It does not tell very much about the condition of the rectum itself, even for the diagnosis of stenosis of the rectum. For moderate stenoses—which are just the ones so difficult to diagnose—offer no obstruction to insufflation, while very pronounced or impermeable stenoses can be easily enough diagnosed without insufflation. This method is of little value even for determining the seat of such obstruction. If it is impossible to introduce any considerable quantity of air into the rectum, a low position of the obstruction has generally been assumed; but if the stenosis is pronounced, there will be so much abdominal distention that it will prevent the distention of the rectum even if the obstruction is high up.

INJECTIONS OF WATER INTO THE RECTUM AND RECTAL LAVAGE.

Introducing fluids to determine the rectal capacity or the seat of a stenosis is even less valuable than inflation because the rectum is generally less tolerant of fluids than of air. By irrigating the rectum and then picking out any suspicious fragments from the wash water to be examined microscopically, we can sometimes find cells from a high-seated carcinoma. It is best to cleanse the rectum thoroughly with an enema and to administer a mild laxative the day before.

SOUNDING OF THE RECTUM.

If an obstruction is situated low down, a rectal bougie will give no more satisfactory results than an ordinary digital examination. If seated higher up, we can never tell accurately whether the bougie is impeded by a pathologic stenosis or by some physiologic obstruction, as

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is very often the case. The elastic metal intestinal tubes constructed with a spiral spring have been advocated by F. Kuhn[1] for rectal work. Their practical value has hardly been proved.

EXAMINATION OF THE INTESTINAL FUNCTIONS.

EXAMINATION OF THE MOTILITY OF THE INTESTINES.

Inspection and auscultation of the abdomen will tell something about the motility of the intestines (see p. 302 *et seq.* and p. 286). We must also depend as well upon the examination of the feces (see 422 *et seq.*). Of course, conclusions in regard to the intestinal motility are given by the passage of flatus as well as by the passage of feces. The intestinal motility is certainly not directly proportional to the frequency of passing flatus, for this depends to a far greater degree upon the production of gas in the intestine; yet, at the same time, a free and abundant passage of gas indicates good intestinal motility, while a complete inability to evacuate any flatus, which is so annoying to the patient on account of the associated distention, proves some impairment in intestinal motility. This latter condition (see p. 422) is important in the types of ileus where solid or fluid evacuations still take place (fecal matter coming from below the obstruction), but where the absence of evacuated gas presents such a contrast as to furnish a very significant symptom of obstruction.

To obtain more information about the intestinal motility, certain substances, such as charcoal, milk, or lycopodium, are ingested at a definite time, and the stools watched to determine the interval required for these substances to appear. Charcoal will be recognized by the black color, milk by the light-yellow color, and lycopodium by the characteristic tetrad shapes of its spores under the microscope. The charcoal and the lycopodium should be administered directly after eating, but the milk between meals, as far apart as possible. Of course, the character of the gastric motility must be taken into account in judging of the results of this test.

EXAMINATION OF THE CHEMISTRY OF THE INTESTINE AND OF ABSORPTION IN THE INTESTINE.

The careful examination of the feces will aid in determining the intestinal chemistry, especially with reference to the utilization of the food (see p. 438); but even so, we are not able to estimate the influence of the length of time the food remains in the intestine. Examining the feces for any digestive enzymes will be of no assistance in judging the intestinal chemistry, because most of the enzymes are destroyed in the intestinal canal, as is explained on p. 445. The examination of the chemical changes brought about by intestinal bacteria is as yet impracticable. Even the tiresome process of a complete metabolic investigation (see the fundamental examinations of v. Noorden[2]) does not always allow of but one deduction in judging of intestinal

[1] See *Berlin. klin. Woch.*, 1898, No. 2, p. 27.

[2] *Grundriss einer Methodik des Stoffwechsels*, Berlin, 1892; *Lehrbuch der Path. des Stoffwechsels*, Berlin, 1893; *Zeits. f. klin. Med.*, 1890, p. 137.

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digestion, because it furnishes results both of stomach and intestinal digestion. The same difficulty also applies to the conclusions which may be drawn regarding metabolic changes, since these also are affected by variations in the absorption and motility of the whole gastro-intestinal tract. Neither are we justified in deducing from the amount of urea eliminated the absolute degree of metabolism, nor the sum total of the digestive processes taking place in the organism.

EXAMINATION OF THE INTESTINAL DIGESTION BY MEANS OF THE GLUTOID CAPSULES.

For a long time the writer has been interested in the problem of obtaining some *direct* evidence in regard to the chemical activity of the intestines, especially with reference to proteid digestion. For the small intestine he has employed the glutoid capsules, which are made from gelatin (glutoid) hardened with formaldehyd. They either do not dissolve in the gastric juice at all, or only after a considerable time, but they are rather quickly soluble in the intestinal juices.[1] They take the place of the pills coated with keratin to prevent the action of the gastric juice, which formerly were employed but which have not proved successful. These glutoid capsules may be utilized to diagnose the condition of the intestinal digestion—*i. e.*, the pancreatic function. They are filled with some substance which will not diffuse through the capsule-wall, and whose absorption may be studied from an examination of the saliva or the urine. Substances containing iodin are best adapted for this purpose. Iodoform has given the author the best results. It is absorbed both from the stomach and the intestines in a very short time, within from one-quarter to one and one-quarter hours. After the ingestion of 0.15 gm. of iodoform, a marked iodin reaction can be obtained in the saliva or in the urine (especially well in the former) with chloroform and nitric acid (see p. 501). Iodoform has another great advantage in that it cannot penetrate the glutoid capsules.

Salol answers very well for the same purpose, for, within one to one and one-half hours after the ingestion of 0.5 gm. of salol salicylic acid, it can be demonstrated in the urine with ferric chlorid as salicyluric acid (see p. 502). So that normally after the glutoid capsule has been dissolved by the pancreatic juice, the iodin reaction will be demonstrable in the saliva at the latest within one and one-quarter hours if 0.15 gm. of iodoform has been administered, and the salicyluric reaction can be demonstrated in the urine within one and one-half hours after the administration of 0.5 gm. of salol. For diagnostic purposes glutoid capsules with 0.15 gm. of iodoform and 2.3 degrees of hardening are the best adapted. For children, capsules containing 0.15 gm. of iodoform may be used, or 0.05 gm. of iodoform and 0.25 gm. of salol, so as to test for the salicyluric reaction in the urine. Adults should take 2 or 3 of these capsules in order to obtain sufficient salol. The following remarks refer to capsules of the above-mentioned degree of hardening.

In order to make the conditions as constant as possible in regard to the length of time that the capsules remain in the stomach and in regard to the degree of digestive stimulation, it is advisable to administer them with an Ewald test breakfast (p. 367). Experience has shown that normally, under the best conditions possible—*i. e.*, normal gastric motility, normal intestinal digestion, and normal intestinal absorption—the iodin reaction may be expected to appear in the saliva, and the salicyluric reaction in the urine, in from four to six hours. If one wishes to test the accuracy of the capsules obtained from the shops, he must prove that the reactions are obtained within this length of time in healthy individuals. Only rough, approximate differences in the time of the reaction are of diagnostic importance, so that it is sufficient to examine the saliva after six,

[1] *Deutsch. med. Woch.*, 1897, No. 1; *Correspondenzbl. f. Schweizer Aerzte*, 1898, No. 10; *Deutsch. Arch. f. klin. Med.*, vol. lxi., 1898.

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eight, ten, and twenty-four hours. If we wish to make the test as accurate as possible, it is perhaps advisable to administer the capsules in the morning, upon an empty stomach, and then four hours later allow the patient to eat as usual. The specimens of saliva and urine may, of course, be saved and all examined afterward.

Before drawing conclusions in regard to the intensity of the pancreatic digestion—*i. e.*, the rapidity with which the contents of the capsule are absorbed—we must, of course, be sure that within six hours the capsules are dissolved neither by the gastric juice nor by any chemical agency in the intestine other than the pancreatic secretion. This condition obtains, for it has been proved that these capsules can resist a strong gastric digestion for at least seven to eight hours, and the putrefactive changes in the intestine for twenty-four hours. When they are introduced, together with an Ewald test breakfast, into a stomach with normal motility, they do not remain in the stomach longer than one hour, but swell up in the gastric contents and are readily floated through the pylorus. Before we can obtain trustworthy data in reference to intestinal digestion, we must necessarily know that the stomach empties itself within the normal period of time. A delayed reaction can never be accurately referred to impaired intestinal digestion unless the function of the stomach has been previously tested carefully by means of the stomach tube. In this connection no method of gastric examination is admissible in which the stomach tube is not employed. If there is motor insufficiency of the stomach, a retarded reaction is to be ascribed to the delay of the capsule within the stomach, whether it has been dissolved there or, later on, in the intestine.

Individual differences in the rapidity of the excretion of the iodin and salicylic acid are not responsible for any particular error in the experiment, because as soon as these substances get into the circulation the reaction appears in the saliva very quickly. Another factor in the experiment which is important is the rapidity with which the iodoform is absorbed after the capsule has been dissolved by the pancreatic juice. The glutoid-capsule test gives us, therefore, the resultant of the power of the pancreatic digestion and the absorptive power of the intestine. If we wish to determine the latter alone, we can repeat the test on another day, when we are sure that the saliva no longer gives an iodin reaction, administering the iodoform plain, not enclosed in a glutoid capsule, but with a glass of water, on an empty stomach. From v. Mehring's experiments we know that the result will largely depend on the absorbing power of the intestine. Absorption is usually rapid, provided the stomach is normal, so that when the hardened glutoid capsules are used the result depends almost entirely on the intensity of the pancreatic digestion.

The results obtained from this method of examination are interesting. Even when the stomach contents contained no free hydrochloric acid or pepsin, the reaction was not delayed so long as the gastric motility was good. The remarkably good state of nutrition of such patients, and the result of such a test, prove that the intestinal digestion may perform vicariously the entire gastric function. In cases of diarrhea due only to an increased peristalsis, without any marked disturbance of intestinal digestion, the reaction is either normal or even somewhat hastened. In other types of diarrhea characterized by an involvement of the intestinal chemistry or intestinal absorption, the reaction is either absent or distinctly delayed. In the former case the undigested capsules are often found in the feces, together with microscopically visible undigested food remnants. This method also aids in differentiating an icterus due to occlusion of the ductus choledochus at its point of entrance into the intestine from one where the obstruction to the bile flow is higher up near the liver. In the former case the digestion of the capsules may be interfered with because the pancreatic duct is simultaneously plugged. Of course, the pancreas may have a separate exit of its own, besides the one in common with the choledochus. The test also aids in the diagnosis of pancreatic affections—*e. g.*, in several cases of carcinoma of the pancreas which we have observed, a negative result has supported a presumptive diagnosis of such a position for the tumor. Circumscribed carcinomata of the pancreas do

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not necessarily occlude the duct, however, so that, of course, a positive result of the glutoid reaction will not exclude cancer of the pancreas.

BOAS' METHOD OF OBTAINING INTESTINAL JUICE.[1]

Boas has shown that it is often possible, both in healthy and sick persons, to obtain enough of the secretion in the upper part of the small intestine for an examination of the pancreatic juice, bile, and succus entericus. This method depends upon the fact that, as Boas and others have found, the pyloric sphincter can often be overcome easily by a very moderate straining, so that the duodenal contents enter the stomach. As the duodenum, like the empty stomach, often contains preformed secretion, it is sometimes possible to force this into the stomach by the patient straining gently. The examiner can help a little by massage of the duodenal region. The technic of the procedure is as follows: The stomach tube is first introduced and the stomach washed with a 1 per cent. solution of sodium carbonate. With the patient in the dorsal decubitus position, the region under the right costal arch, between the mammary and parasternal lines, is massaged from right to left for several minutes, and then, after the tube has been introduced, the patient expels the gastric contents in the ordinary way by the help of coughing or straining. With such a method we can often obtain 40 to 50 c.c. of a neutral, alkaline, or weakly acid fluid. The acid reaction is due partly to the irritation of the tube and partly to the presence of preformed gastric juice. The fluid is usually stained strongly with bile. We can prove that it is duodenal by demonstrating a typical proteolytic, lipolytic, and diastatic action. The presence of trypsin can be very easily proved; but the demonstration of the lipolytic and of the diastatic enzymes has not been universally successful. For examination the fluid must, if necessary, be rendered distinctly alkaline as soon as possible by the addition of a weak sodium carbonate solution (so as to prevent any of the trypsin being destroyed by pepsin). Trypsin can be most easily demonstrated by digesting in the alkaline fluid, at incubator temperature, a flake of fibrin stained with Magdala-red. The fibrin, which is dissolved by the typical digestion process, colors the fluid red.

Arthus and Huber have announced another method[2] for demonstrating the action of trypsin. Fresh fibrin is prepared by whipping freshly drawn horse blood, and then washing the coagulum with water until it becomes colorless. It is finally completely covered and allowed to stand for twenty-four hours, at 40° C., in a 2 per cent. solution of sodium fluorid, then filtered. We thus obtain a solution of fibrin in sodium fluorid which will keep for months. The fluid which is to be examined for trypsin is first diluted with an equal volume of a 2 per cent. solution of sodium fluorid, and one volume of this dilution is added to two to three volumes of the fibrin solution, and the whole mixture digested for a considerable time at 40° C. If trypsin is present in the fluid, crystals or crusts of tyrosin will form on the walls of the vessels. The crystals may often be recognized by the naked eye. In addition to their shape (see Fig. 173), they are characterized by their appearing light on a dark background in the polarizing microscope (with crossed nicols). One advantage of this procedure is that the digestive mixture remains sterile for an unlimited length of time, owing to the antiseptic qualities of the sodium fluorid.

Grützner-Gamgee recommended the following method to demonstrate lipolytic activity: An emulsion of 10 parts of oil, 5 parts of gum, and 35 parts of water is prepared; also a neutral solution of litmus, which, in test tubes of 12 mm. diameter, appears violet against white paper. Ten cubic centimeters of such a litmus solution and 5 drops of the emulsion are placed in several of these tubes; and increasing quantities—*e. g.*, 2, 4, 8, 16, and 32 drops—of the fluid to be

[1] Boas' "Ueber Darmsaftgewinnung beim Menschen," *Centralbl. f. klin. Med.*, vol. x., 1889, No. 6, p. 97. The same, *Zeits. f. klin. Med.*, 1890, vol. xvii., p. 155; also Tschelenoff, "Ueber Darmsaftgewinnung beim Menschen," *Correspondenzbl. f. Schweizer Aerzte* 1889, No. 6, p. 161.

[2] *Arch. de Physiol.*, 1894, p. 622.

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examined are added to the successive test tubes. They are then immediately set in a water bath at 37° C. After a few moments the different tubes are compared. If any fat-splitting ferment is present, the color of the fluid will have turned redder the larger the amount of solution added.

To demonstrate diastatic action, increasing amounts of the fluid to be examined are added to a thin starch paste, and the whole kept at 38° C. If a minimal amount of iodin is then added to this mixture and no violet color appears (see p. 371), we obtain evidence that a diastatic action has taken place. Again, the presence of dextrose, as shown by Trommer's test, will prove the same thing (see p. 482 *et seq.*).

Although it is an interesting fact that intestinal juice may be obtained in this manner, the procedure is not sufficiently developed to enable us to study intestinal digestion in this way, and it seems improbable that it ever will be, since the results obtained are dependent upon entirely too many factors which are difficult of estimation. If a juice containing active trypsin be obtained, the most that can be said is that it demonstrates a patulous condition of the pancreatic duct, just as does the reaction with glutoid capsules (p. 419).

EXAMINATION OF THE FECES.[1]

FREQUENCY OF MOVEMENTS; DIARRHEA; CONSTIPATION; AMOUNT OF FECES.

There is considerable variation in the *frequency of the bowel movements* within the bounds of health. Some absolutely healthy people have a movement of the bowels only once in two or three days, others have several every day. There is no sharp line of distinction in either direction between the pathologic and physiologic. The term constipation is usually restricted to a condition of infrequent movements which is associated with certain other difficulties or which does not bear a proper relation to the quantity of food taken; in the same way, the term diarrhea is employed only when the movements are not only frequent, but also liquid. Infants normally have two or three movements.

Other things being equal, the frequency of the movements depends upon the amount of fluid ingested. In starving individuals the number is reduced to a minimum; and in many and most varied diseases a more or less developed state of starvation may be developed. For instance, an individual who vomits everything is practically starving. In the same way, a gastric case or that of any other severely ill individual, who takes little on account of lack of appetite or because he cannot retain anything, is to be considered as a starvation case.

Diseases which lead to *constipation* are: gastric and intestinal catarrh (especially chronic), gastric dilatation, intestinal obstruction of various sorts, peritonitis, meningitis and other diseases which increase the cerebral pressure. Sometimes constipation becomes a more or less independent disease (chronic constipation), the nature of which has not yet been explained.

A complete absence of movements is generally characteristic of some kind of intestinal obstruction. Not infrequently, however, fecal material may be evacuated from the section of the intestine below the obstruction. Continuous diarrhea is

[1] Consult J. Schmidt and J. Strassburger, *Die Fäces des Menschen*, Berlin, Hirschwald, 1901.

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characteristic of some types of obstruction, especially in invaginations, in complete axis rotation, or in case something is jammed in the gut. But these movements are really not fecal in character; they consist of serous or, often, bloody masses from the lower intestinal segment, due to the venous congestion at the seat of lesion. The character of the movements, and especially the absence of the passage of gas, should be sufficient to justify the diagnosis of obstruction.

Diarrhea occurs in acute and chronic gastric and intestinal catarrh, in certain types of chronic peritonitis, in intestinal tuberculosis, intestinal amyloid degeneration, cirrhosis of the liver, cholera, typhoid fever, dysentery, many other infectious diseases, and uremia.

The daily quantity of the feces passed, other things being equal, is directly proportional to the amount of food ingested. The volume of the individual movements is generally inversely proportional to the number.

There are, however, numerous exceptions to the first of these statements—*e. g.*, in patients who vomit often, much less is evacuated in the movements than is ingested. Again, in severe forms of diarrhea, especially cholera, much more is evacuated than is ingested, because in addition to the food residue there are the secretions and exfoliation of the intestinal mucous membrane. The admixture of blood may also increase the quantity of the feces.

In diarrhea the individual movements vary in volume and frequency according to whether the disease is localized in the upper or lower intestine. In diseases of the lower part of the colon (dysentery is a type) the individual evacuations are not very voluminous, but they are very frequent, on account of the continuous reflex tenesmus. In diseases of the upper intestine each evacuation is more profuse, but they do not occur so frequently—*e. g.*, typhoid—because there is not the same persistent desire to empty the rectum. These, so to speak, "profuse" diarrheas may be found in involvement of the upper portion of the colon as well as of the small intestine.

After a period of marked constipation quite incredible quantities of feces are often passed.

CONSISTENCE AND SHAPE OF FECES; STRATIFICATION OF LIQUID MOVEMENTS.

The normal consistence and shape of feces is well known. The consistence is hard in constipation, fluid in diarrhea. Between these extremes are all sorts of intermediate steps—*e. g.*, small fecal balls like sheep dung, which occur in intense constipation, from the tightly packed fecal matter becoming friable. On the other hand, dry fecal masses may be of very unusual volume in constipation if a large quantity of feces stagnate in the rectum and distend it mechanically. In intestinal stenosis but a short way above the anus the fecal masses have a diminished transverse diameter; but if the stenosis is high up the fecal matter changes its shape again below the obstruction. Leichtenstern has justly called attention to the fact that a small caliber of the fecal masses may be found also in inanition (starvation feces) and in rectal tenesmus.

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Very liquid diarrheal movements often stratify themselves with the liquid constituents in an upper layer, and the solid food residue in the lower. Frequently, however, the formation of layers is due simply to an admixture with urine.

COLOR AND GENERAL APPEARANCE OF THE STOOLS.

The normal color of the stools of an adult is dark brown. This color is not due to biliary pigment, but to its secondary products (urobilin, etc.). Infants' stools are normally light yellow or golden yellow, because they contain unaltered bilirubin.

The color of the feces varies more or less according to the nature of the food. A milk diet gives a light color to the stools; abundant consumption of red wine, blueberries, blackberries, or black cherries, on the other hand, gives them a dark color. Food rich in chlorophyll (vegetables) generally produces a green or olive shade in the stool. Drugs may also change the color of the feces. Extractum ligni Campechiani produces a reddish-brown color in the stools. Calomel is apt to color them greenish. According to Wassilieff and Hoppe-Seyler, this coloration is not due to the formation of sulphur compounds of mercury, as was formerly believed, but to the antiseptic action of the calomel, which prevents the transformation of the bile pigments into urobilin, and also to the sublimate derived from the calomel, which changes the bilirubin to biliverdin. After the use of bismuth the stools are generally of a blackish color. Quincke[1] has proved that this color is not due to the formation of metallic sulphur, as was formerly thought to be the case, but rather to a reduction of the bismuth salt to bismuth suboxid, as in Nylander's test for sugar. Not infrequently bismuth stools are colored green, very much like those after calomel, a phenomenon which Quincke explains by assuming that the bismuth also checks the transformation of the bile pigments to urobilin. He claims that, contrary to the general belief, the stools after the use of iron are not black from ferrous sulphate. He maintains that immediately after evacuation they present no abnormal color, but become grayish brown to grayish black only after standing and upon the surface being exposed to the air. He believes that this phenomenon is due to the oxidation of organic iron combinations when exposed to the air. Patients to whom we administer iron are usually the ones in whose dark-colored stools there is often an admixture of blood, so that we should remember Quincke's reasoning. The character of the stools after the administration of methylene-blue gives evidence of the processes of reduction going on in the intestinal canal itself. When freely evacuated they are usually of the ordinary color, due to the reduction of the methylene-blue, but within a few moments they become bluish green on the surface. This coloration gradually penetrates the mass. If the stool is preserved under oil no such color appears.

Under pathologic conditions the stool may be abnormally colored by the addition of blood. The stool is abnormally light in color in *acholia* (deficient production of bile with absence of icterus) and in retention of bile due to occlusion of the biliary passages (in icterus). Such stools present a peculiar grayish-white appearance (clay-colored). This is due not only to the deficiency in amount of bile, but also to an abundance of unabsorbed fat.

Nothnagel[2] distinguishes uncolored feces from acholic stools. The former occur not only without any icterus, but, in contradistinction to the latter, without

[1] *Münch. med. Woch.*, 1896, No. 36.
[2] *Die Erkrankungen des Darmes und Peritoneums*, p. 18.

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any demonstrable disturbance of fat-absorption. Nothnagel and v. Jaksch suspect that in these cases, instead of urobilin, colorless reduction products are formed in the intestine from bilirubin (leucourobilin of Nencki). This subject has not as yet been very closely studied from a chemical standpoint. Nevertheless this possibility is certainly suggested, because v. Jaksch succeeded in extracting with acid alcohol very considerable quantities of urobilin from such so-called acholic stools, and because such stools often darken considerably when exposed to the air, an effect evidently to be ascribed to oxidation. Quincke claims that the normal color of such stools is reproduced when calomel is administered, merely because the latter inhibits the reduction of the urobilin, which is ordinarily caused by putrefactive processes in the intestine.

In diarrhea the movements are, generally speaking, light colored because the pigment is distributed through a much larger volume, but sometimes (especially in intestinal catarrhs) they may contain the bile pigment and vary in color from green to yellow. (See p. 445 for the demonstration of bile pigment in the stools.) The presence of unchanged bile pigments in the stools is always pathologic, except in infants (see above).

We sometimes find undigested food particles in the stools. This is of no diagnostic significance so far as indigestible constituents are concerned, such as seeds, stones, and the skins of fruits. But if the stools contain considerable macroscopic evidence of substances which are ordinarily so changed by the intestinal digestion as not to be recognized, —*e. g.*, bits of meat, casein flocks, etc.—we naturally attribute this peculiarity to some digestive disturbance. This condition, found in stomach and intestinal disorders, was formerly described as "lientery." If there is a fistulous communication between the stomach and intestine or between a loop of the upper and a loop of the lower intestine, more or less undigested food will appear in the stools. (See p. 438 *et seq.* for the microscopic examination of the movement with reference to the utilization of the food.) If excessive decomposition processes take place in the intestine, the diarrhea movements will exhibit a peculiar foamy consistence. In infantile diarrhea the stools are often of a greenish color, due to the abnormal decomposition of the bile pigment; or if there are many flocks of casein, they may have a crumby or lumpy appearance instead of their normal smooth character.

With reference to the admixture of mucus, blood, and pus in the stools, see the following pages.

ODOR OF THE STOOLS.

The odor of normal human feces is well known. It is due chiefly to indol and skatol, and perhaps also to methyl mercaptan. The normal stool of an infant emits only a very faint odor, which is not peculiarly fecal. In intestinal catarrh, however, the odor of infants' stools may be very foul. The odor of diarrheal movements varies greatly, sometimes abnormally strong, sometimes noticeably slight. Nearly odorless stools very frequently follow the use of strong cathartics. (Compare the characteristics of certain types of movements on p. 443.)

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EVIDENT ADMIXTURES OF MUCUS IN THE STOOLS.

A noticeable admixture of mucus in the stool will be recognized here, as in other excreta, usually by its peculiar consistence and transparency. In a doubtful case the addition of dilute acetic acid will cloud the portions containing mucus. The mucus in the feces is a true mucin.

Even a normal stool contains some mucus, and sometimes in small masses which can be recognized macroscopically. Greater amounts of mucus indicate some catarrhal condition of the intestinal mucus membrane.

In catarrh of the small intestine the mucus is evenly distributed in the solid or semisolid feces, and may be recognized by the slimy consistence of the whole stool or by the presence of small, evenly distributed shreds or tiny transparent lumps. We must, however, be careful not to confuse bits of swollen vegetable tissue with lumps of mucus. The former are frequently found in a normal stool, and can be easily recognized microscopically by their cellular structure (see Fig. 141). Large shreds of mucus in a watery stool are found especially in catarrh of the large intestine. The large lumps of mucus which sometimes coat solid fecal masses are also derived from the large intestine. Mucus passages are also observed in catarrh of the large intestine between normal movements (colico mucosa, enteritis membranacea). These peculiar whitish ribbon- or tube-like formations are passed frequently and with rather violent pains, probably due to the violent peristalsis needed to tear them from the wall of the large intestine. The laity often mistake these formations for tapeworm. Admixtures of small kernels or tapes of mucus are observed in the watery stools of cholera (rice-water stools, see p. 443). With reference to the chemical test for mucin, see p. 446).

VISIBLE ADMIXTURE OF BLOOD IN THE STOOL.

The blood contained in the stool may exhibit itself in different ways. Sometimes a microscope is required to demonstrate it; at others it may be recognized macroscopically by a red or blackish color.

In the latter event, various deductions in regard to the seat of the hemorrhage may be made from the color and arrangement of the blood admixture. For instance, solid feces coated externally with blood would indicate a hemorrhage only in the lower portion of the intestine, where the feces are already solid and shaped (hemorrhoids). Conversely, solid feces evenly tinged throughout would point toward a hemorrhage in the stomach or the upper part of the intestine. The admixture of blood even in liquid stools is usually more evenly distributed if the hemorrhage is in the upper part of the intestine. But the liquid movements from the large intestine (dysenteric meat-water stools, see p. 444), may also contain evenly mixed blood. The color of the blood in liquid stools sometimes forms a good criterion for determining the seat of hemorrhage. The higher the hemorrhage is, the more altered is the original blood color, due to intestinal decomposition and intestinal digestion. In hemorrhage from the stomach, gastric digestion may also exert some change. Profuse hemorrhage from the stomach does not always

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cause hematemesis, so that we may first suspect the condition from the appearance of the stools, which may be nearly black and of a tar-like consistence. Profuse typhoid hemorrhages sometimes show an alteration of the blood; usually, however, it is still distinctly red, because it generally comes from the lower portion of the small intestine, and a stool follows a hemorrhage quickly. The bloody, serous diarrheal stools without true fecal matter are of great diagnostic importance in certain types of obstruction, especially invagination (see p. 423). In doubtful cases a microscopic examination may determine the presence of blood in the feces; but the red blood-corpuscles are often found considerably changed and hard to recognize. Sometimes they may be destroyed completely, and then the question as to whether blood is present or not must be decided by a chemical or spectroscopic examination.

PUS IN THE STOOL.

A very considerable admixture of pus or pure pus stools are always due to perforating abscesses. A slight amount of pus in the stool, which often can be demonstrated only microscopically, and which is frequently associated with blood or mucus, may be due to catarrhal changes, but is generally due to ulcerative processes of the mucous membrane (tuberculosis, dysentery). Lumps of pus (even when microscopic) immediately suggest an ulcerative process. It may be very difficult to demonstrate pus in the stool, because the pus corpuscles are sometimes destroyed by the digestion and the intestinal decomposition. Experiments bearing directly on this point have shown us that intestinal decomposition may destroy pus corpuscles within a comparatively short time, so that it is impossible to recognize them. The nuclei may resist the decomposition for a considerable length of time, but they are difficult to recognize in this isolated condition, especially as they are polymorphous[1] and differ in no way from the cell nuclei of any animal food, which may persist after digestion. Moreover, even in normal stools individual white blood-corpuscles may be found as a result of the normal transmigration through the mucous membrane. The demonstration of pus is sometimes difficult even in perforation of perityphlitic abscesses into the intestine, because decomposition in the abscess has already begun to change the character of the pus, and this change may be complete in the intestine.

The undigested lumps of casein found in the diarrheal movements accompanying a milk diet must not be confounded with bits of pus. Although impregnated with fecal pigment, the former sometimes assume a pus-like appearance; they may easily be recognized microscopically by the fat-drops which they contain.

NEOPLASTIC FRAGMENTS IN THE STOOL.

Larger or smaller pieces of tumor are not infrequently cast off in carcinoma of the rectum or of the intestine higher up. These fragments attract attention chiefly in a liquid stool by their grayish-red color and solid consistence.[2]

[1] Pus cells have polymorphous nuclei, and are called polynuclear leukocytes. (See Examination of Blood.)

[2] A bit may be teased apart or frozen and sectioned and then examined microscopically, or the fragments may be placed in a 4 per cent. watery solution of formaldehyd

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As it is often impossible to recognize the finer details, the essential point is to detect the numerous nuclei arranged according to the character of the cell constituents. Fragments of adenomatous polyps are sometimes found in the stools. They may occur independently or they may be located in the neighborhood of carcinomatous and tuberculous ulcers of the intestines.

GALL-STONES, PSEUDOGALL-STONES, BILIARY GRAVEL, PANCREATIC STONES, INTESTINAL CONCRETIONS, INTESTINAL SAND IN THE STOOLS.

Cholelithiasis, an attack of biliary colic, may be followed by the appearance of gall-stones from time to time in the stools, or they may appear without any previous biliary colic. In searching for them the stool should be thoroughly mixed with water and washed and rubbed through a sieve. To be certain, we should examine the movements during at least fourteen days after the cessation of the attack of colic.

Gall-stones are concretions which form in the biliary passages, from the size of a pinhead to that of a pigeon's egg, or even larger. They consist chiefly of cholesterin and calcium bilirubin, in varying proportions. Besides these there are present subordinate amounts of biliverdin, bilicyanin, bilifuscin, bilihemin, and calcium carbonate. Cholesterin gives a light, and calcium bilirubin a dark, color to the concretion. The color varies, according to the predominance of one or the other of these constituents, between white and dark brown or dark olive green. Sometimes they are soft enough to be readily cut or crushed, but often, on the contrary, are very hard. The cross-section usually shows distinct concentric layers of crystalline consistence, sometimes of different colors. In other cases the surface may present very beautiful facet formations, so that tetrads and rhombic or multiangular shapes result. Sometimes the surface is irregularly granular, a point of diagnostic importance. If facets are distinctly shaped we may be sure of the presence of multiple concretions, and, in all probability, of their origin in the gall-bladder. Round stones occur singly and also in numbers. Very large stones (larger than hazelnuts) hardly ever reach the intestine *per vias naturales*, but probably always by perforation of the bile passages.

FIG. 147.—Wood cell from the seed of a pear (after Bizzozero).

We must be careful not to confound other solid constituents of the feces with gall-stones—*e. g.*, woody bits of plants, especially from the cores of pears. These similar formations have been termed *pseudogall-stones*.[1] The microscopic examination of a small fragment scratched off with a knife from such a formation shows characteristic wood cells (Fig. 147). The chemical examination (see below) would also prevent any mistake. Besides, the wood-like particles are much harder than even the hardest gall-stones.

So-called "*biliary sand*," in the majority of cases, consists of these small pseudogall-stones. The existence of biliary sand—*i. e.*, large numbers of the smallest gall-stones—in the feces has not been accurately proved as yet. Naunyn considers it impossible, because his investigations show that such small concretions easily dissolve in the intestine, and because such small concretions would not be passed in any such large quantities at one time, but gradually, as they were formed.

Another kind of pseudogall-stones is the concretion which consists of fats and fatty soaps that are not easily melted. They are found in the stools after the admin-

(℞ Formaldehyd solul. venale (33 per cent.) 120, aq. dest. ad 1000). Bits of from ½ to 1 in. in thickness are sufficiently hardened after one-half to one hour; but it does no harm to leave them in the solution up to eight days. The pieces are then frozen in with the formalin solution itself or with clear water and cut; the sections are placed in 50 per cent. alcohol and stained with aqueous anilin dyes. With this method the sections are said to be much better preserved than with the ordinary freezing process.

[1] Compare Fürbringer, *Verhandl. d. XI. Congr. f. inn. Med.*, 1892, p. 313.

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istration of large amounts of olive oil in treating cholelithiasis. Hence, the over-*estimation* of the therapeutic value of this oil. It is difficult to understand this error. These artificially produced concretions are characterized usually by their transparency and soft, greasy consistence, and, if the biliary passages are not occluded, by their greenish color.

Even a careful and prolonged search may fail to find the concretion in certain cases of *gall-stone disease*, sometimes because the stone, which was jammed in the neck of the gall-bladder and so caused the colic, has returned to the bladder itself. In other cases the stones which are at fault have become stuck in the ductus choledochus, while the patency of the biliary passage has been re-established alongside of the stone. Finally, the concretion may have fallen to pieces in the intestine. Naunyn's experimental investigations seem to prove that the latter is very often the case. He believes that ordinarily only the more solid gall-stones—for instance, those with a solid cholesterin shell—are found in the feces. A further explanation of the cases where no gall-stone is found in the feces after a typical attack of colic is the fact that typical attacks of colic may be due to inflammation with or without gall-stone.

For chemical examination gall-stones are first dried, then powdered, and then extracted with alcoholic ether to dissolve the cholesterin. This may be easily recognized by allowing the solution to evaporate slowly in a watch glass. The characteristic glistening cholesterin crystals separate, and are easily recognized under the microscope by their sharp, linear, rhombic outline (see Fig. 211). After extraction with alcohol and ether, the residue is treated while cold with very dilute potassium hydroxid. If the powder contains calcium bilirubin a yellow solution will be obtained, from which Gmelin's reaction may be obtained (p. 472). Naunyn claims that some concretions contain only bilirubin, in which case the green in Gmelin's reaction will be absent and only the blue ring will be found.

The much rarer *pancreatic concretions* differ from gall-stones in not containing bile coloring matter, and, chemically, in consisting chiefly of calcium carbonate, which dissolves with effervescence in hydrochloric acid. They seldom have facets.

Intestinal Stones or Fecal Concretions; Incrustations of Food Particles with Organic Salts.—These play an important part in human pathology in exciting appendicitis. They appear very rarely in the stools. They consist almost exclusively of ammonium and magnesium phosphate, and should be examined in the same way as urinary calculi (see p. 563). Eichhorst and Deetz [1] report cases, which were not sufficiently explained pathologically, in which large quantities of intestinal sand, consisting of finely granular mineral concretions, were found. Perhaps these cases have at times been erroneously considered of biliary origin.

ANIMAL PARASITES IN THE STOOLS.

PROTOZOA.

Of the protozoa there have been observed in the human intestinal contents amebæ (Fig. 150), sporozoa of the species Coccidium (psorosperms), flagella of the species Megastomum (Fig. 149), cercomonas (Fig. 148) and trichomonas (Fig. 151) and infusoria of the species Balantidium. The parasites of the species Ameba have become of considerable importance in the past two decades, because they have been found to be the cause of some dysenteric disturbances (Fig. 150). Amebæ are organisms with streaming, creeping (so-called ameboid) movements. They consist of a protoplasmic substance containing granules and vacuoles, which changes its shape in an irregular way while moving. Each ameba contains in its

[1] *Arch. f. klin. Med.*, vol. lxx., parts 3 and 4, p. 365.

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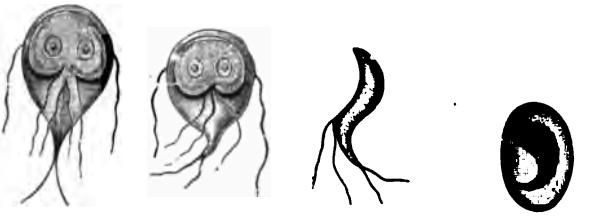
interior a nucleus, which is, however, not always easy to see. Propagation takes place by division; the formation of spores has not been definitely proved. Some amebæ have permanent types (permanent cysts, encysted amebæ). They are

FIG. 148.—Cercomonas hominis: Length, 9 to 11 μ; width, 5 μ (Davaine) (after Roos).

small, round, with a sharp, often double, contour, and are immobile. The death of an ameba is indicated by the cessation of its ameboid movements (Fig. 150), and it then disintegrates so rapidly that it is not easily to be recognized. Quincke



Seen on flat. Seen on side. Encysted form.

FIG. 149.—Megastoma entericum: Length, 15 to 17 μ; breadth, 9 to 11 μ (after Roos). *Deutsch. Arch. f. klin. Med.*, vol. li., p. 506.

claims that the permanent types, on the contrary, may remain visible in the stool for twenty days.[1] As yet no one has been able to cultivate amebæ, so that it is

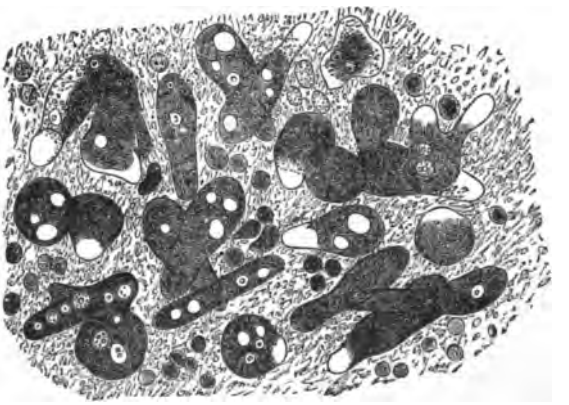


FIG. 150.—Amebæ of dysentery (after Leuckart) (Lösch).

difficult to obtain any light on the species of the individuals found. Amebæ sometimes occur in normal intestinal contents. Large numbers are found quite regu-

[1] Quincke and Roos, *Berlin. klin. Woch.*, 1893, No. 45; Janowsky, *Zeits. f. klin. Med.*, 1897, xxxii., 5 and 6; Roos, *Arch. f. klin. Med.*, vol. li.

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larly in tropic dysentery. Pure cultures of dysenteric amebæ have not as yet been grown ; but dogs and cats have been infected with stools containing amebæ introduced per os and per rectum. Streptococci are also very frequently found in dysenteric stools along with the amebæ, so that some cases are due to mixed infection. The amebæ penetrate the base of intestinal ulcers, and are also found in the pus of dysenteric abscesses, which also argues in favor of their etiologic significance. With Lösch, who discovered it, Quincke suggested calling the ameba

FIG. 151.—Trichomonas intestinalis: Length, without tail, 11 to 15 μ; width 5.5 to 8 μ (Marchand) (after Roos).

which has sometimes been found in the stools of healthy individuals *Amœba intestinalis vulgaris*—the true dysenteric ameba. *Amœba coli* (Fig. 150) and the ameba of the German dysenteric disorders he names *Amœba coli mitis*, placing it halfway between the others. We do not really know whether these are different species or whether they are varieties of varying virulence.

The organisms can be most readily recognized by their ameboid motion, so that it is very important to examine the stool when freshly voided and contained in a

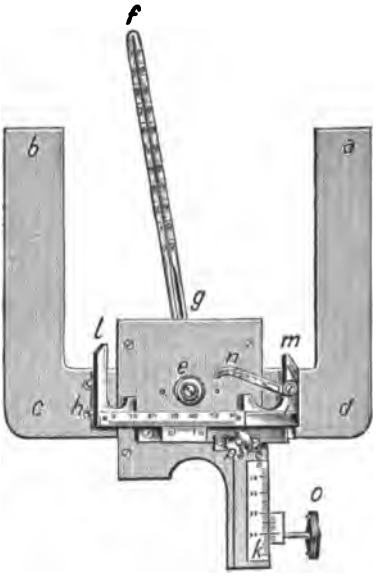

FIG. 152.—A movable stage which can be heated.

vessel which has been warmed to 40° C. Bloody or purulent flakes are selected from the stool and examined microscopically. More solid masses must first be diluted with warm physiologic salt solution. Dry preparations may be stained, and the amebæ are seen to be much paler than the deeply stained bacteria. Their motility can be preserved by employing a warm stage. The warm movable stage, constructed according to the author's directions by Leitz, in Wetzlar, is illustrated in Fig. 152. It is a combination of Schulze's warm stage with a superimposed movable stage. The apparatus is attached to the stage of the microscope by two

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screws, which are not visible in the figure, and the stage is heated by two small burners beneath the points *a* and *b*.

ENTOZOA.

Discovering the eggs of one of the intestinal worms is quite as important from the diagnostic point of view as discovering the organism itself. A low power is the best to employ at first, and then a higher one. The stools should either be liquid or washed with water. It may be necessary to scratch off particles of feces from the edge of the anal opening with a microscopic spatula, or to obtain some from the rectum by means of the introduced finger. The Oxyuris vermicularis is not infrequently observed in this way. Larger quantities of feces may be obtained by introducing a thick glass tube with blunt edges. If the eggs cannot be demonstrated upon the first examination, administering some cathartic (castor oil) will often prove of service, as the liquid intestinal contents will thus be more evenly mixed. (For the characteristic peculiarities of the eggs of the different species, see below.) If neither eggs nor bits of the parasite are found after the administration of an ordinary cathartic, the next procedure is to give the patient some anthelmintic—*e. g.*, santonin. In case of ascarides this will serve the double purpose of diagnosis and treatment; but for tapeworm the treatment is more prolonged and complicated. For diagnostic purposes it would be sufficient to administer about one-third of the usual therapeutic dose—*e. g.*, 3 gm. ext. filicis.

Leichtenstern[1] has drawn attention to the diagnostic importance of Charcot's crystals in the stool (see Fig. 211) for the recognition of intestinal worms. These crystals occur in the feces with every variety of entozoa, and often in very large amounts. They appear to bear some relation to the metabolism of the worms.

See Blood Examination (p. 650) with reference to the almost constant occurrence of Charcot's crystals and eosinophilic leukocytosis in the various types of helminthiasis.

Round Worms.

Nematodes Ascarides.—The only ascaris which occurs frequently in the human intestine is the Ascaris lumbricoides (Fig. 153). The diagnosis of ascaris is generally very easily made, either from the eggs or from the worms themselves. The latter are 10 to 15 cm. long, white to dirty brown in color. They are evacuated with the feces from time to time, and sometimes may be vomited. The ascarides eggs are passed in so great numbers with the feces that the examination of bits of feces from about the anal orifice is usually sufficient. They differ from all other entozoa eggs in their peculiar, irregularly wavy albuminous shell (see Fig. 153, *c*). This envelope may, however, be absent. The largest diameter of the eggs is about 0.05 to 0.06 mm.

Oxyuris vermicularis (Fig. 154).—This small intestinal worm inhabits chiefly (but not exclusively) the large intestine. It is 3 to 12 mm. long. It causes very annoying itching in the anal region, and should be searched for in almost any case of pruritus ani. Either the small thread-like worms are found in the feces or on the skin in the vicinity of the anus, or else the eggs are found microscopically in the bits of feces which adhere to the anal orifice. The eggs are 0.05 mm. long, and some-

[1] *Deutsch. med. Woch.*, 1892, p. 582.

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what symmetric. Leichtenstern has proved that oxyuris never lay eggs in the intestine, but always migrate through the anal orifice for that purpose. Hence, for diagnostic purposes, it is of no use to examine the stools for the eggs. Even the eggs found at the anal orifice are more or less accidental. In the majority of cases the only way to demonstrate the presence of oxyuris is to find the worm itself.

Ankylostomum Duodenale.—If viewed with the naked eye, this worm (Fig. 155) closely resembles oxyuris. It inhabits the small intestine, and is 6 to

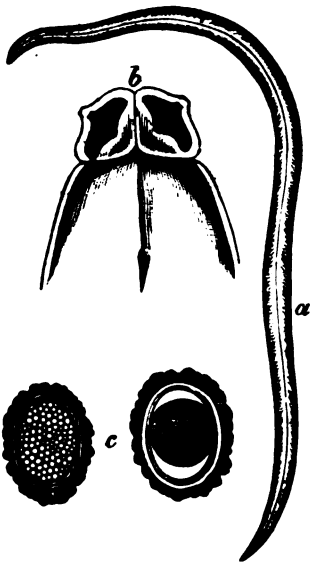

FIG. 153.—Ascaris lumbricoides: *a*, Body: *b*, head; *c*, eggs (after v. Jaksch).

18 mm. long (Heller). The male is always smaller than the female. The eggs are oval; 0.05 mm. long, 0.023 mm. broad, and, unlike those of oxyuris, are evacuated in the feces. Ankylostoma, as found in the stool, are usually of a reddish tinge, due to the coloring-matter of the blood sucked from their host. They are hooked so firmly to the intestinal wall that they rarely appear in the stools unless some anthelmintic has been administered. For this reason the diagnosis must be based upon the history, upon the appearance of severe consecutive anemias, and

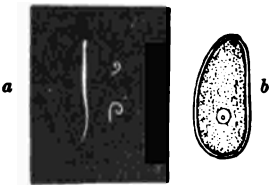

FIG. 154.—Oxyuris vermicularis and egg: *a*, Natural size; *b*, egg (after Heller).

principally upon the demonstration of the eggs in the stools. Ankylostomum induces a very severe anemia. It occurs in certain vicinities among miners, workmen in tunnels, and brickmakers. In the tropics it is called tropic or Egyptian chlorosis.

[Since the publication of Stiles' masterly monograph,[1] the importance of this

[1] Hygienic Laboratory, *Bulletin No. 10*, Feb., 1903. (*Report upon the Prevalence and Geographic Distribution of Hook-worm Disease* (Uncinariasis or Ankylostomiasis).

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parasite in the Southern States has been attested by numerous observers. No clinician here should fail to familiarize himself with the general features of the

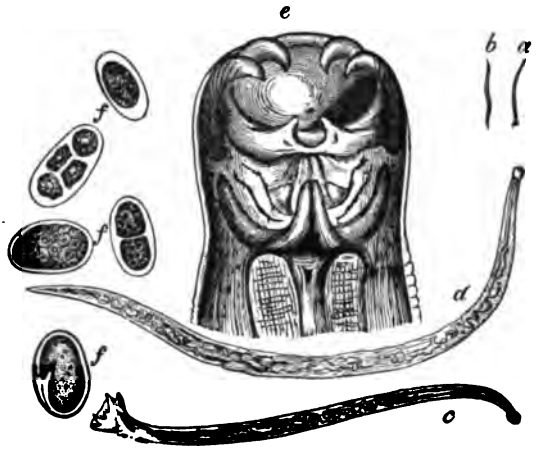

FIG. 155.—Ankylostomum duodenale: *a*, Male (natural size); *b*, female (natural size); *c*, male (enlarged); *d*, female (enlarged); *e*, head; *f*, eggs (after v. Jaksch).

worm, its eggs, and embryos. The appended diagrams (Figs. 156 and 157) are copied from the above-mentioned source.—ED.]

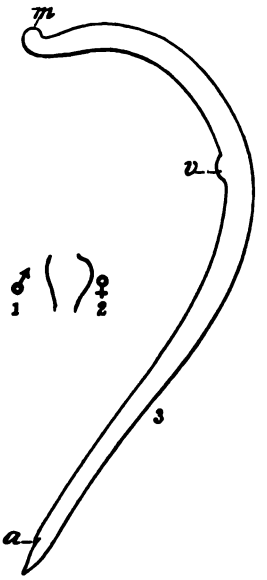

FIG. 156.—New World hook-worm (*Uncinaria Americana*); natural size: 1, Male; 2, female; 3, the same enlarged to show the position of the anus (*a*), the vulva (*v*), and the mouth (*m*) (after Stiles).

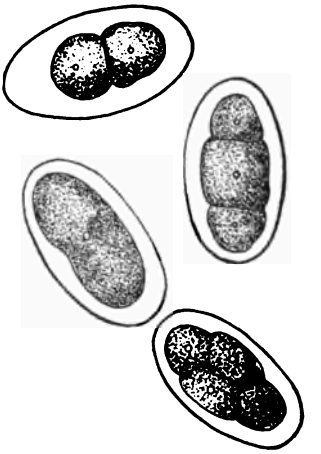

FIG. 157.—Four eggs of the New World hook worm (*Uncinaria Americana*), in the one-, two-, and four-cell stages. The egg showing three cells is a lateral view of a four-cell stage. These eggs are found in the feces of patients, and give a positive diagnosis of infection. Greatly enlarged (after Stiles).

Tricocephalus Dispar (Fig. 158).—This is a round worm, 4 to 5 cm. long, which inhabits the cecum and colon. The eggs are easily recognized and fre-

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quently found in the stools. Ordinarily it is a very harmless parasite, but if present in very large numbers it may produce very grave, even fatal, enteritis, as has been shown by Leichtenstern and Moosbrugger.

Trichina Spiralis (Fig. 159).—In the first stages of trichinosis the adult or embryo trichina may sometimes, though very rarely, be evacuated in the stools, and so be utilized in establishing the diagnosis. The full-grown male is 1.5 mm. or less in length, and the female 4 mm. or less.

Anguillula Intestinalis and Stercoralis (Fig. 160).—These worms, 0.8 to 2.2 mm. long, are found in the intestinal contents of patients with Cochin-China

FIG. 158.—Trichocephalus dispar: Parasite (natural size) and egg (after Küchenmeister).

diarrhea. Leichtenstern found this worm among brickmakers under similar conditions as ankylostomum, sometimes occurring with the latter parasite and sometimes alone. Both Perroncito and the author found it in the workmen employed in the St. Gothard tunnel. We do not know whether it has any pathologic significance. Grassi and Leichtenstern [1] believe that Anguillula intestinalis and stercoralis are only different stages of development of one and the same parasite, which develops in the feces outside of the human body in the sexually ripe stages as Rhabditis stercoralis.

Trematodes (Flukes).

Distomum lanceolatum and **hepaticum** are found, although very rarely, in the biliary passages. Their eggs have been found several times in the intes-

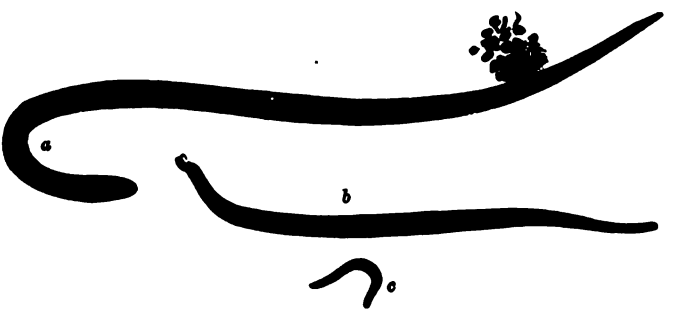

FIG. 159.—Trichina spiralis (greatly enlaged): *a*, Female; *b*, male; *c*, embryo (after Heller).

tinal .contents. They closely resemble the eggs of the Distomum pulmonale (Fig. 212).

Cestodes (Tapeworms).

The tapeworms which are important, so far as the human body is concerned, are almost exclusively the Tænia solium, Tænia mediocanellata (saginata), and the Bothriocephalus latus. They all inhabit the small intestine. As is well known, tapeworms consist of different generative forms united to each other, the so-called head, and the sexual segments or proglottides, which develop from and are attached to the head, and in which the eggs develop hemaphroditely. These segments and free eggs are from time to time evacuated with the patient's stools. The diagnosis of tapeworm infection should never be made from the general symptomatology, but only from the occurrence of proglottides or of eggs in the

[1] "Ueber Anguillula Intestinalis," *Deutsch. med. Woch.*, 1898, No. 8.

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feces (see above, p. 432). The color of the individual proglottides, or of a series of them as they appear, is whitish. Shreds of mucus or strings of vegetable or animal fiber are often mistaken by the laity for tapeworm, mistakes which merely emphasize the importance of the physician observing the suspected material himself.

FIG. 160.—Anguillula intestinalis and stercoralis: 1, Larva (Anguillula intestinalis); 2, male Anguillula stercoralis; 3, female Anguillula stercoralis (after Perroncito).

After the tapeworm has been passed, it is especially important to know whether the head has been secured. If not, the treatment is unsuccessful and will have to be repeated at some future time. If the small end of the tapeworm has been passed without the head being attached, we must search carefully for the latter. This is best done by gently mixing the feces with a large quantity of water and straining through a fine sieve, or by allowing the mixture to settle, and then pouring off the upper layers and searching for the head at the bottom. A cathartic should always precede the anthelmintic, to render the search easier. If the head has remained behind, fresh segments or other eggs will be passed within three months.

Tænia.—Tæniæ differ from the bothriocephalus in two points. In the first place, the sexual orifice is situated at the margin of the proglottides; sometimes on one side, sometimes on the other; looking to the naked eye like a slight excavation in a blunt projection. In the second place, the head has round sucking disks.

Tænia Solium (Fig. 161).—In its early stages this tapeworm inhabits (as swine measles, Cysticercus cellulosæ) the intermuscular connective tissue of the hog. Man becomes infected by eating raw or insufficiently cooked pork. The worm in man may attain a length of 3 m. The head is rounded and small, the size of a pinhead (1.3 mm. in diameter), and has a wreath of hooklets in the middle between 4 sucking disks. Continuous with the head is a thread-like non-segmented portion about 3 cm. long, to which the proglottides are attached; toward the end of the worm the latter are large and broader. Each segment is a little narrower at the upper than at the lower end. Full-grown segments in a relaxed condition are 9 to 10 mm. long, 4 to 5 mm. broad, and when fresh are somewhat contracted and wavy. Each proglottis contains a uterus with comparatively coarse dendritic branches.

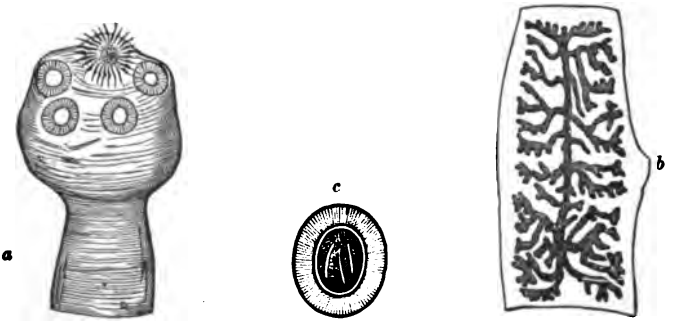

FIG. 161.—Tænia solium: *a*, Head (enlarged); *b*, proglottis (magnified six times); *c*, egg (after Heller).

These details can be seen best with the naked eye by compressing a mature proglottis between two slides. The eggs are round, about 0.035 mm. in diameter. They have a thick covering with radiating stripes. Tænia solium in man may lead to an auto-infection with Cysticercus cellulosæ.

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Tænia Mediocanellata (*Saginata*) (Fig. 162.)—The cysticercus stage of this tænia is found in cattle, probably also in goats and sheep. It is comparatively rarely observed in meat, because it occurs in no such great numbers as the Cysticercus cellulosæ of the hog. This fact probably has some connection with the different modes of life of the two parasites.

The adult segments *Tænia mediocanellata*, when relaxed, are 16 to 20 mm.

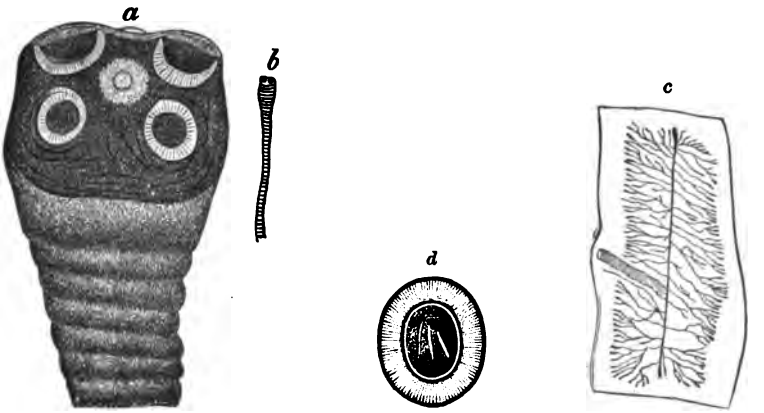

FIG. 162.—Tænia mediocanellata: *a*, Head (enlarged); *b*, head (natural size); *c*, proglottis (magnified six times); *d*, egg (after Heller).

long, 5 to 7 mm. broad. The uterus has five subdivisions, and the branches are much more numerous than in Tænia solium. The entire tænia reaches a much larger size than Tænia solium (up to 6 m. long) (saginata—"stuffed"). The head is also much larger, 2.5 mm. broad. It has no hooklets, and was therefore formerly called Tænia inermis. The eggs do not differ much from those of

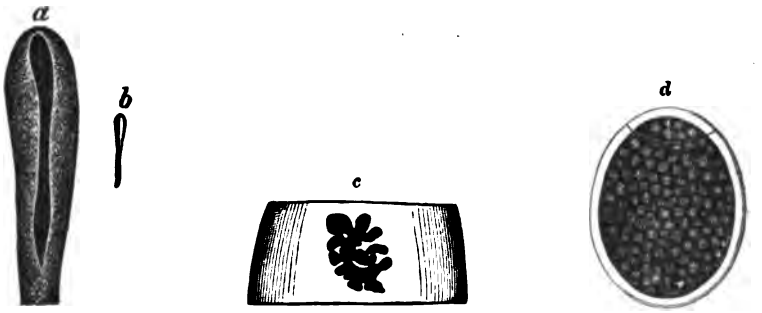

FIG. 163.—Bothriocephalus latus: *a*, Head (enlarged); *b*, head (natural size); *c*, ripe segment (magnified); *d*, egg (after Heller).

Tænia solium. Once in a while single proglotti creep out of the anus. Auto-infection with the cysticercus of Tænia mediocanellata has been observed but once.

Bothriocephalus Latus (Fig. 163).

This is the largest of all human tapeworms, attaining sometimes a length of 8 m. The mature segments are quadrilateral and nearly square (about 5 mm.); the growing segments are three to four times as broad as they are long, 10 to 15 mm. broad by 1 to 4 mm. long. The head is flat and oblong, 1 mm. broad, 2 mm. long, with a slit-like sucking groove at the corner on each side. The sexual

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opening in the proglottides is not at the margin, as in the teniæ, but in the middle of the surface. The uterus is situated around this opening, in the shape of a roset. The eggs have a thin shell, with coarse, granular, mulberry-like contents, and a lid at the top, and can thus be easily distinguished from the tenia eggs. They are also larger than the latter (0.07 mm. long and 0.045 mm. broad). In the young ova it is not always easy to recognize the lid, as it is either undeveloped or occupies only the extreme pole. With bothriocephalus we usually find longer pieces and not individual segments in the stools. The segments do not occur as often as with the teniæ. Hence, searching for the ova is of especial diagnostic importance. As certain cases of pernicious anemia are due to the action of this parasite, the stools should be carefully examined in every case of severe anemia whose etiology is not plain.

MICROSCOPIC EXAMINATION OF THE STOOLS WITH REFERENCE TO THE UTILIZATION OF THE FOOD.

UTILIZATION AND SPLITTING OF FAT.

An incomplete utilization of the fats sometimes becomes apparent even in a microscopic examination of the stool. Stools which contain an abnormal quantity of fat appear gray, often glistening. The fat consists partly of a microscopic admixture and partly of larger particles, whose form depends upon the manner in which it was administered. Under the microscope the fat is seen to consist of fat-drops and of needle-like crystals. (See Fat-crystals of the Sputum, Fig. 211, *a*.) Their appearance depends upon the melting-point of the respective fats, and upon whether the fats are found in the stool unchanged, split up, or saponified. Neutral fats may appear either as drops or as needles. Fatty acid soaps usually appear as thick, short needles or cakes; free fatty acids, as needles.

Normal stools contain a moderate number of *fat-needles*, which represent the fats that are not so easily melted, and are therefore less readily absorbed. Fat-drops are not so common except after abundant ingestion of milk or easily melted fats. They usually signify an insufficient utilization of the fats, as does also an abnormal prominence of fat-needles.

The stools may be abnormally fatty in disease of the pancreas, and in cases when the bile is shut off from the intestine. But in both cases emulsified fat, such as is contained in milk, seems to be well absorbed. (See Chemical Examination of Feces, p. 446.)

UTILIZATION OF STARCH.

The microscopic appearance of starch is well known (p. 359, Fig. 141). Well-preserved starch granules are rarely found in the normal feces of an adult, but they may occur in the movements of infants improperly fed with starches.

An abundance of starch granules in the stools of adults is pathologic, and is usually a sequence of diarrhea or of gastric hyperacidity. The absence of pancreatic juice does not seem to produce pathologic quantities of starch in the feces, because the starches, along with the other carbohydrates, are utilized very extensively by the intestinal bacteria for the formation of acids. Deficiency of bile in the intestinal contents is not responsible for any increase of starch in the stool, as the bile contains only traces of a diastatic enzyme, which are of no practical importance.

UTILIZATION OF THE MUSCLE FIBERS AND OF THE OTHER PROTEIDS OF THE FOODS.

Meat fibers in the movements are easily recognized microscopically by the more or less well-preserved transverse striation. (Fig 164, *aa*.) The more thorough the digestion, the less distinctly this appears, and the more are the fragments rounded off. Undigested muscle fibers are constantly found in the feces of a person upon a mixed diet. The amount becomes pathologically increased in diarrhea, in fever, and in other disturbances of the digestive mechanism.

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To discover connective tissue the stools should be stirred with water and examined over some dark substance (black plate). (See Examination of Sputum.) The connective-tissue fragments may then be recognized as white fibers.

Ad. Schmidt[1] claims that the macroscopic appearance of muscle fibers in the stools of a patient who has ingested a test meal of 100 gm. of very slightly roasted chopped meat demonstrates some severe damage to the intestinal digestion. On the other hand, the occurrence of macroscopically visible fragments of connective tissue is indicative of insufficient gastric digestion, because connective tissue, unless it is cooked to pieces, is dissolved only by pepsin.

Coagulated proteids and casein may be found in amorphous masses in normal

FIG. 164.—Microscopic elements of normal feces: *a*, Muscle fibers; *b*, connective tissues; *c*, epithelial cells; *d*, white blood-corpuscles; *e*, spiral vessels of plants; *f*—*h*, vegetable cells; *i*, plant hairs; *k*, triple phosphate crystals; *l*, stone cells. Scattered among these elements are micro-organisms and débris (after v. Jaksch).

stools, and more especially in stools denoting insufficiently utilized food. The coarse, crumbling casein lumps are important diagnostically, because they compose the major part of an infant's poorly digested stool. An infant's normal stool should be homogeneous and even.

INDIGESTIBLE FOOD RESIDUE.

In every normal stool the constituents of the food which are indigestible reappear. All cellulose-like substances are not attacked by the digestive ferments, and only very incompletely by the bacteria of the intestine (plant hairs and spiral vessels, vegetable cell formations, etc.). (Fig. 164, *e.*, *i.*, *l.*) Various parts of a meat diet also remain undigested; for instance, larger strands of connective tissue and fragments of elastic fibers (Fig. 164).

FERMENTATION OF THE FECES AFTER EVACUATION.

Ad. Schmidt and his pupils have recently endeavored to draw conclusions in relation to intestinal function from the manner in which the feces ferment after evacuation—*i. e.*, from the gases which are thus formed. The most important of the original communications is to be found in the *Deutsches Archiv für klinische Medizin.*, vol. lxi.

BACTERIA OF THE FECES.

Feces consist to a large extent of the body substance of micro-organisms. Before Koch's culture methods became known, it seemed to be quite impossible to isolate the bacterial admixture of a normal or pathologic stool. But since then, by the isolation of individual varieties, some light has been thrown on

[1] *Deutsch. med. Woch.*, 1899, No. 49, p. 811.

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this subject. Bienstock was the first to study the question exactly. He decided that only four kinds of bacilli were to be found in the intestine, one of which was the specific decomposer of the proteids; and that there were no micrococci, because, being non-sporebearing, they were destroyed upon entering the digestive tract by the hydrochloric acid of the stomach. Later investigators, who worked with anaërobic culture methods and varied culture media, could not corroborate these results; they showed that the number of varieties was much larger. Although at the present time no practical clinical methods seem available, except for the demonstration of specific pathologic varieties, it does appear probable that sooner or later these investigations will aid in our understanding of some of the complicated problems of digestion.

Mannaberg's comprehensive description may be consulted for the better known species of normal intestinal bacteria and fungi.[1] By far the majority of the species of bacteria in the feces belong to the Bacterium coli group. Among the best known varieties are the Bacterium lactis aërogenes, the Bacillus subtilis, Proteus vulgaris, Bacillus butyricus (Bacillus amylobacter), and others. Bacteria which elsewhere we know as the cause of disease, such as the staphylococci, are found in abundance in normal feces. Only when these bacteria are so very abundant that they can be demonstrated without culture do they become important from a diagnostic standpoint.

We can obtain an approximate notion of the bacteriologic condition of the feces by examining with an oil-immersion lens a small particle unstained (if too hard, mix with water). The number, the shape, and the individual movements of the bacteria should be noted. For more accurate microscopic examination a dry specimen should be stained with carbolfuchsin or after Gram's method (p. 596). The ordinary plate culture media are used, if necessary, to grow the individual varieties—*e. g.*, bouillon, gelatin, and agar-agar. Especially for the intestinal bacteria, faintly acid beer-wort, in the form of agar plates, is recommended. Several varieties are said to grow in this medium which do not thrive upon the ordinary culture media. Here we cannot go into the technic of these nor of the anaërobic cultures.

Escherich's investigations on the intestinal bacteria of infants are of interest. He found that immediately after birth the meconium was free from bacteria. Four to seven hours after birth was the earliest that he found any bacteria. The feces of milk generally contain but two species of bacteria—the Bacillus coli communis, which chiefly inhabits the colon and the lower segment of the small intestine, and is much in excess so far as numbers go, and the Bacillus lactis aërogenes, which grows in the upper part of the small intestine. In the stools of more than half of the infants suffering from acute gastric catarrh he also found a spirillum, extremely fine and hard to stain, to which he ascribes a grave prognostic significance.

PATHOGENIC BACTERIA OF THE FECES WHICH ARE IMPORTANT FROM A DIAGNOSTIC STANDPOINT.

Bacilli of Tuberculosis (Fig. 213).—In intestinal tuberculosis these bacilli are found in the feces, and are, therefore, of diagnostic importance. The stools may, however, contain these bacilli, even though there is no intestinal tuberculosis (if the patients swallow their sputum). Searching the stools has even been recommended for the diagnosis of lung tuberculosis in cases of irresponsible persons who swallow the sputum. Although in the examination of sputum previous treatment with dilute potassium hydroxid or digestive enzymes is often successful it is rarely of use in the examination of feces (see p. 596), because the feces consist chiefly of indigestible substances, which are insoluble in potassium hydroxid. But it may be serviceable in the examination of mucopurulent particles of the movement which have been isolated from the mass of feces. We do not know whether under certain conditions decomposition may destroy tubercle bacilli in the intestine. At

[1] In the introduction to Nothnagel: *Diseases of the Intestines and Peritoneum.* It contains a complete index of the literature on the subject.

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any rate, we cannot always demonstrate tubercle bacilli in the stools even when there is undoubted intestinal tuberculosis. Perhaps this is on account of the dilution of the content of tubercle bacilli by the abundant particles of food. Tubercle bacilli are most readily found in the purulent or bloody pieces of diarrheal stools. Generally speaking, direct searching through solid stools is less promising. Nevertheless they may quite frequently be demonstrated in solid movements if, as Hamburger recommends, we mix a piece of feces the size of a pea with a few centimeters of water, then centrifuge gently to remove the coarser pieces, dilute the supernatant cloudy fluid with a double volume of alcohol, centrifuge once more, and then after drying examine the remaining precipitate, which will consist almost exclusively of bacteria. As tubercle bacilli in the feces may be due to swallowed sputum, we can diagnose intestinal tuberculosis if bacilli are found in the feces only, when at the same time attacks of diarrhea occur with pus and blood in the stool. The tubercle bacillus must be carefully distinguished from the smegma bacillus, which is said to occur at the anal orifice and might have become mixed with the feces.

Bacillus of Cholera (Comma Bacillus) (Fig. 165).—The demonstration of cholera bacilli is of great importance in the early diagnosis of the first cases of a cholera epidemic. The bacilli are constantly present in the stools of cholera

FIG. 165.—Cholera bacilli (× 1000) (after Weichselbaum).

patients, and at times in very great quantities. By demonstrating their presence the differential diagnosis between cholera nostras and asiatica, formerly so very difficult, may be made with certainty in an isolated case and without first waiting for an epidemic. A dry specimen should be prepared from one of the little mucus flakes suspended in the movement, and then stained in the ordinary way with fuchsin-gentian-violet or methylene-blue. (See Examination of Sputum.)

A microscopic examination for comma bacilli is not enough to make a diagnosis absolutely sure, because there are other comma-shaped bacilli, as, for example, those of Finkler and Prior, found in cholera nostras. Moreover, a negative microscopic result without any culture tests would not permit the absolute exclusion of cholera. For this reason gelatin plate cultures must be prepared from the suspected cholera stool in order to render the diagnosis certain. This is done in the ordinary way described in any text-book on bacteriologic technic. After twenty-four to thirty-six hours numerous liquefying colonies will be found, from which gelatin-streak and potato cultures are to be made. The gelatin-streak cultures are characterized by the fact that the gelatin on the surface is liquefied very rapidly, whereas in the interior the liquefaction occurs only as a slender canal along the line of inoculation. On potato the comma bacilli grow rather slowly at 20° C. as a thin gray-green coating. If the comma bacilli are cultivated in a sterilized solution of 1 gm. of peptone and 0.5 gm. of NaCl to 100 gm. of water,

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and concentrated sulphuric acid is then allowed to flow underneath, a red discoloration will occur at the line of junction of the two fluids, due to the indol contained in the culture. This pigment used to be called cholera-red, but this name is not quite correct, because the same indol reaction occurs also with other bacteria. If the culture is not a pure one, the reaction is of no diagnostic importance. Cholera bacilli are motile in a hanging drop.

The comma bacilli may sometimes be found in the vomitus.

Competent data relative to the bacteriologic demonstration of cholera bacilli are to be found in Pfeiffer's comprehensive article in *Deutsche medicinische Wochenschrift*, 1892, No. 36.

In regard to the differences between the Finkler-Prior (see p. 361) and other comma bacilli found in certain cases of cholera nostras, and the true comma bacillus of Koch, bacteriologic manuals should be consulted.

Various difficulties in making a bacteriologic diagnosis of cholera have recently arisen on account of the discovery of numerous varieties of cholera bacilli. To solve these difficulties Pfeiffer requires for diagnosis a biologic cholera reaction of the suspected culture. This is accomplished by injecting into the peritoneal cavity of young guinea-pigs a lethal amount of the bacilli in question simultaneously with an injection of a definite amount of the serum of animals immune to cholera. If they are true cholera bacilli they will be dissolved within a short time in the peritoneal cavity. Pfeiffer[1] claims that other bacteria, no matter how much they may resemble cholera bacilli, do not show this purely specific reaction.

If serum from animals immunized against cholera be added to a liquid culture of cholera bacilli, the latter becomes non-motile and agglutinates in lumps, very much as do typhoid bacilli when acted on by typhoid serum. (See Widal's Serum Diagnosis, p. 675, *ff.*) This peculiarity may be utilized in making a differential diagnosis of cholera.

Typhoid Bacilli.—Typhoid bacilli have been demonstrated in the stools of typhoid patients from the second or third week on. Culture methods (isolation and plate cultures) are, of course, necessary for each demonstration, since the typhoid bacilli do not possess any typical morphologic peculiarities. This demonstration is, however, too complicated and takes too long to be of any practical importance in the diagnosis of typhoid fever. Elsner[2] has announced a method of isolating typhoid bacilli more easily from the stools, and also differentiating them from colon bacilli. He employs a faintly acid culture medium containing potassium iodid. Piorkowski[3] has attempted to achieve the same result by the employment of plate cultures upon a medium to which alkaline urine has been added.

To overcome the difficulties of a bacteriologic diagnosis of typhoid bacilli in contradistinction to certain varieties of colon bacilli, Pfeiffer[4] tried to characterize the typhoid bacilli by some biologic reaction with the serum of animals immune to typhoid. This reaction as described by Pfeiffer is performed upon animals in exactly the same way as the cholera reaction (see above). It may now be replaced by the convenient Gruber-Widal agglutination reaction of typhoid. (See later, Blood-examination, p. 675.) To be sure, the utility of this procedure for recognizing typhoid bacilli has of late been questioned, while the value of the serum reaction as a diagnostic measure is now hardly ever disputed.

Kruse's Bacillus of Dysentery.—While tropic dysentery seems to be an amebic disease (see p. 429), Kruse[5] has recently described a peculiar bacillus as the exciting cause of non-tropic epidemic dysentery. These bacilli are found as plump rods in the purulent portions of the stool, and grow readily upon artificial culture media, forming delicate leaf-like colonies upon gelatin plates within twenty-four to forty-eight hours. According to Kruse, they are found in such large

[1] *Zeits. f. Hyg.*, 1895, vol. xix., p. 75.

[2] *Zeits. f. Hyg. u. Infectionskrankh.*, 1895, vol. xxi.; and Brieger, *Deutsch. med. Woch.* 1895, No. 50, p. 835.

[3] *Berlin. klin. Woch.*, 1889, No. 7, p. 149.

[4] R. Pfeiffer and W. Kolle, "Ueber die specifische Immunitätsreaction der Typhusbacillen," *Zeits. f. Hyg.*, 1896, vol. xxi., part 2.

[5] *Deutsch. med. Woch.*, 1900, p. 637.

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numbers in the fresh stools of dysentery that in plate cultures their colonies greatly exceed those of the colon bacilli. Upon glucose agar they grow like typhoid bacilli, without producing gas either superficially or in the depths of the culture medium. Upon potato there is a yellowish growth along the line of inoculation, surrounded by a clear area. They may be differentiated from typhoid bacilli by their plumpness and also by their lack of motility and the absence of cilia. They do not stain by Gram's method. They sometimes exhibit distinct polar granules. Kruse believes that the relation of these micro-organisms to dysentery is shown by the fact that they are agglutinated by the blood-serum of a dysenteric convalescent in a dilution of 1 : 50, and sometimes by a dilution of even 1 : 1000. Under certain conditions the agglutinative power of the serum of a dysenteric convalescent may last for a year. In reference to the diagnostic value of the agglutinative reaction, the reader is referred to Lentz's[1] article.

Streptococci.—A number of serious diseases have recently been attributed[2] to an invasion of the digestive tract by streptococci. Their course is like that of typhoid, cholera, or finally, of an acute enteritis with strepticopyemic localization (peritonitis, endocarditis, nephritis, etc.). These cases can be easily diagnosed from a microscopic examination of the diarrheal stools, which always contain very extraordinary quantities of streptococci. They may be found in a dry preparation stained with carbolfuchsin or by Gram's method (see p. 596). The vomitus and the urine may sometimes contain abundant streptococci. The prognosis in these cases is usually fatal, except in those resembling typhoid, which generally recover. (See pictures of streptococci, Fig. 218).

Anthrax Bacilli.—Anthrax can sometimes be diagnosed by the presence of anthrax bacilli in the loose, frequently bloody, stools. (See Fig. 235).

CHARACTERISTIC NATURE OF THE STOOLS IN SOME DISEASES.

Stools of Typhoid Fever.—Typhoid fever is generally accompanied by loose yellow stools with a consistence like pea soup. They usually settle in layers with a thick crumbling sediment and a supernatant, cloudy, watery menstrum. The odor is generally very strong and offensive, and possibly diagnostic. The reaction is usually strongly alkaline. In fact, the stools usually contain abundant microscopic crystals of triple phosphates (ammoniomagnesium phosphate; see Figs. 189 and 190). Simple microscopic examination does not disclose any special bacteriologic characteristic. The stools are frequently stained with blood. A slight tinge not infrequently precedes a profuse intestinal hemorrhage and, hence, is to be carefully watched for. Pus in quantities which can be demonstrated microscopically is only contained in the stools of cases with severe and extensive ulcerations. The diarrheal stools of typhoid are usually very copious. Solid movements are not infrequently found, not only in the beginning, but even throughout the entire duration of the disease.

Stools of Asiatic Cholera and Cholera Nostras.—In mild attacks of Asiatic cholera and cholera nostras the stools may present the character of an ordinary diarrhea. In severe cases they are no longer fecal in color, but are thin and nearly colorless or grayish, like water in which tiny cloudy flakes are suspended (rice-water stools). The absence of any brown coloration is probably due to oligocholia or acholia. The stools no longer emit a fecal, but a sour semen-like odor. The reaction is alkaline or neutral. In the mucous flakes we see, under the microscope, intestinal epithelium arranged in layers, more or less abundant cholera bacilli (Fig. 165), and sometimes drops of fat. The stools may also be slightly tinged with blood. Cholera stools contain only about 2 per cent. of solids, and of this a large amount is sodium chlorid, but very little proteids. The stools are usually very profuse and voided without any especial effort (not like

[1] *Zeits. f. Hyg. u. Infectionskrankh.*, vol. xli., 1902, part 3, p. 559.

[2] Compare, *e. g.*, "Contribution à l'étude du streptocoque et de l'enterite streptococcique. Quatre mémoires par MM. de Cérenville, Tavel, Eguet et Krummbein," *Ann. Suisses de méd.*, Series II., 1895. C. Sallmann, Basel.

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dysentery). As the general condition improves they gradually resume a fecal appearance. As is well known, cases of cholera do occur without diarrhea (cholera) sicca).

The stools of cholera nostras are much like those in true cholera, except that true cholera bacilli are absent, and that, corresponding to the milder nature of the disease, they are more or less stained by bile. Both the stools and vomitus in cholera nostras contain very varied micro-organisms (Finkler-Prior bacilli, streptococci, and others, see pp. 442 and 443).

Stools of Dysentery and Carcinoma of the Rectum.—Dysentery stools present the character of rectal diarrhea (see p. 423)—*i. e.*, they are not profuse, but voided so much the oftener. Soon after the attack begins they lose their fecal character, and are at first slimy, then mucopurulent, and finally sanguinopurulent or seropurulent (meat-water stools, white flux, red flux). We may often see peculiar, solid, reddish or white fragments, which consist partly of blood-stained mucus (carunculæ of older writers) and partly of macroscopically visible bits of exfoliated mucus. The stools in dysentery may be practically odorless, but in grave cases (gangrenous flux) they emit a noticeable cadaverous odor. The stools of carcinoma of the rectum sometimes very closely resemble those of dysentery.

Stools in Diseases of the Pancreas.—Considering the predominance of the pancreas in the digestion of fats, it is easily understood that in some of the diseases of this organ which lead to its destruction or to the occlusion of its ducts, so-called "*fat-stools*" are observed. The latter are characterized microscopically and chemically by their abnormal amount of fat (steatorrhea). We must, however, be rather cautious in making use of this symptom for diagnosis, because, on the one hand, the stools may show an abnormal amount of fat in any case of marked icterus, and, on the other hand, cases of almost total destruction of the pancreas have been observed when the stools at the time showed no abnormal amount of fat. This is because emulsified fat, even without any pancreatic juice, can be very readily absorbed, as showed on p. 438, while evidently the fat not in emulsion was taken care of by the vicarious action of the bile. Hence, fatty stools are diagnostic of pancreatic disorders only where there is no jaundice, and hence also the absence of fatty stools does not exclude destructive pancreatic disturbance. In making a diagnosis of pancreatic disturbance from the appearance of the stools we must also remember that, if the pancreatic juice is absent, chemical examination shows only insignificant quantities of soaps in the fatty stools (p. 446), the movements show but slight signs of putrefaction, and the urine contains only a slight amount of indican (see p. 475). In pancreatic disease where there is no flow of pancreatic juice into the intestines, well-hardened glutoid capsules (see p. 419) are found undissolved in the intestinal contents. The iodoform-glutoid reaction is therefore absent.

CHEMICAL EXAMINATION OF THE FECES.

REACTION OF THE STOOLS.

Under normal conditions the reaction of the feces may be neutral, faintly acid, or faintly alkaline. Gamgee considers a faintly alkaline reaction the most frequent. The reaction on the surface of formed feces often differs from that in the interior. The reaction may also change upon prolonged standing. An admixture of urine will very soon produce an alkaline reaction. Pathologically the reaction may become either strongly acid or strongly alkaline, according to the kind of decomposition processes which are occurring in the intestinal canal. Typhoid and cholera stools are usually alkaline. The stools accompanying a milk or a starchy diet are usually, but not always, acid in reaction.

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PIGMENT OF THE FECES.

The color of the feces of normal adults is never due to unchanged bile pigment (bilirubin), because the latter is partly transformed into urobilin in the intestine, and partly reabsorbed and used over again in the organism (probably to help form bile and to produce urinary pigment). Therefore the presence of bilirubin in the feces always indicates some abnormality in the intestine, either some disturbance in absorption or in the chemical processes, or else some increased peristalsis. Bile pigment often appears in diarrheal stools. It can then sometimes be recognized by its intense yellowish or greenish shade. Chemically, it can be very easily demonstrated by Gmelin's test (p. 472)—*i. e.*, by dropping a little nitric acid directly upon the feces and noting the green, red, and violet rings around the drops of acid. The green ring is the most characteristic.

The normal pigment of stools is *urobilin* or *hydrobilirubin*. These substances may be easily extracted from the feces by means of alcohol containing hydrochloric acid, and then demonstrated in solution spectroscopically or chemically with zinc chlorid. (See Examination of the Urine, p. 478.) Ad. Schmidt recently announced a simple method of demonstrating urobilin in the feces directly without extraction.[1] He adds a little concentrated mercuric chlorid solution to a small amount of feces in a porcelain dish. If urobilin is present the feces will turn red. The reaction can be completed in about a quarter of an hour. The same test can be employed to demonstrate the presence of bilirubin, which will turn green in the presence of the mercuric chlorid solution. In the same specimen of feces we can therefore recognize by the contrast of color the particles which contain bilirubin and those which contain urobilin. Schmidt believes that the formation of urobilin begins in the large intestine, or perhaps very low down in the small intestine. He was unable to produce the change of bilirubin to urobilin by bacterial cultures; but the fresh intestinal wall itself in an incubator did cause this change to take place. Hence, it would seem that the formation of urobilin is a function of the intestinal wall.

BILE ACIDS IN THE FECES.

Normally, the bile acids are almost completely reabsorbed from the intestinal canal, so that in the feces only small quantities of cholic and choloidinic acid are to be found. Very little definite is known about the presence of bile acids in the feces under pathologic conditions. Their detection is a complicated process. (See Hoppe-Seyler, *Physiologisch-und pathologisch-chemische Analyse*, 1893, p. 47.) If a large quantity of the bile acids is present in the feces they may be detected, in all probability, directly by extracting with dilute sodium carbonate solution and then performing Pettenkofer's test (see p. 475).

DIGESTIVE ENZYMES IN THE FECES.

The enzymes may be extracted from the feces by means of glycerin or of bits of fibrin, which absorb the enzymes physically. This requires the addition of antiseptic substances to prevent putrefaction. Thymol, oleum menthæ piperitæ, and oleum sinapis volatile are well adapted for this purpose. The presence of the enzyme can then be demonstrated by an artificial digestive test. To some digestive mixture we add a little of the glycerin extract, or of the fibrin containing the enzymes. In the latter case the fibrin itself may serve as the object to be digested, or disks of coagulated egg albumin (see p. 391) may be used. These digestive tests also require the presence of small amounts of antiseptic substances, so as to exclude the action of bacteria. At the conclusion of a digestive experiment we must examine microscopically to exclude any bacterial action. The action of pepsin is to be tested in a 0.2 per cent. solution of HCl, that of trypsin in 0.3 to 0.4 per cent. solution of Na_2CO_3. Arthus' method of testing for trypsin may be employed (p. 421). Both pepsin and trypsin seem normally to be destroyed in the intestine.

v. Jaksch generally found diastase and an inverting ferment in children's

[1] *Verhandl. des Cong. f. inn. Med.*, 1895.

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feces. Leo found the same in adults'. Neither author was able to demonstrate the presence of trypsin in most cases, but Leo demonstrated all three ferments in cases of diarrhea.

MUCIN IN THE FECES.

Stools normally contain considerable mucin, which is increased in catarrhal conditions of the intestines. Sometimes the appearance and consistence of the feces (see p. 426) are enough to show this. Chemically, mucin can be demonstrated by mixing the feces with water, adding an equal volume of calcium hydroxid, in which mucin is soluble, and then adding dilute acetic acid to the filtrate. Cloudiness indicates mucin (v. Jaksch method).

PROTEIN AND PEPTONE OR PROTEOSES IN THE FECES.

The detection of these substances by means of the biuret reaction must be performed with special caution, because the urobilin which is contained in the feces also reacts to this test (extracting urobilin with alcohol; see p. 467). Normal feces are free from any soluble proteids, peptones, and proteoses, but they can be demonstrated under pathologic conditions, especially diarrhea.

CARBOHYDRATES IN THE FECES.

Unaltered starch is best demonstrated microscopically (see p. 438 and Fig. 141, *l*, *n*, p. 359).

To show the presence of sugar, feces are boiled with water, then filtered, and the filtrate tested by Trommer's test or by the phenylhydrazin test (pp. 482 and 487).

QUANTITATIVE AND QUALITATIVE TESTS FOR FATS, FATTY ACIDS, AND SOAPS IN THE FECES.

Even a microscopic examination of the stool will enable one to form some estimate of the content of fat (p. 438).

To estimate the amount of fat quantitatively[1] and accurately a weighed amount of the stool is dried at 100° C., then mixed with double the quantity of sand, which has been treated for several days previously with water, alcohol, and HCl, and then again with water. The feces-sand mixture is now extracted with ether, by means of Soxhlet's apparatus, until no more fat can be removed. This usually requires between eight and ten hours. The ether residue after thorough washing with warm water indicates the amount of neutral fats and fatty acids which are present. The content of fatty acids is determined by dissolving a weighed amount of the ethereal extract in alcohol and ether and then titrating with alcoholic potassium hydroxid, with phenolphthalein as an indicator.

To determine the amount of the soaps, the feces are boiled with acidulated (HCl) alcohol, after they have been first extracted with ether in the manner mentioned above; they are then dried again and extracted once more with ether. The hydrochloric acid will free the fatty acids from the soaps, and the latter may then be titrated in the second ether extract as described above. The amount of soap present may now be calculated from the result. F. Müller has suggested a simpler method when we wish to determine only the neutral fats and the fatty acids, including the soaps. He advises boiling the dried specimen directly with acidulated alcohol in order to liberate the fatty acids from the soaps. The feces will then contain only fatty acids and neutral fats. The former can be titrated as above in a definite portion of the ether extract.

With these quantitative estimates as a basis, it is easy to determine the quantita-

[1] *Cf.* Fried. Müller, "Untersuchungen über den Icterus," *Zeits. f. klin. Med.*, vol. xii., p. 51, 1887; Deucher, *Correspondenzbl. f. Schweizer Aerzte*, 1898, No. 11.

Volhard's work upon the lipolytic ferment of the stomach should be consulted for the method of estimating the neutral fats and the free fatty acids separately (*Zeits. f. klin. Med.*, 1901, vol. xliii., p. 417).

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tive variations of the free fatty acids and soaps as compared with the neutral fats. In healthy individuals, and even in cases of jaundice, provided the pancreatic juice flows into the intestine, by far the greater part (84.3 per cent., F. Müller) of the fat is split up into fatty acids or soaps. If the pancreatic duct is occluded, Müller found only 39 per cent. of the fats split up, as against Deucher, who found 80 per cent. According to Deucher, the 80 per cent. is composed almost exclusively of free fatty acids, not soaps.

CHEMICAL AND SPECTROSCOPIC TESTS FOR BLOOD IN THE FECES.

Blood undergoes a variety of chemical changes during its passage through the digestive tract. The most important derivatives which originate from the hemoglobin are methemoglobin and hematin.

Teichmann's hemin test and *Schönbein-Almén's turpentine-guaiac test* are the most useful for detecting chemically the hemoglobin derivatives in the feces. These methods often give positive results even when the microscopic examination for blood-corpuscles fails.

One difficulty in utilizing the turpentine-guaiac test (p. 471) in such complicated mixtures as the feces and the gastric contents (p. 360) is that, according to Weber,[1] other substances can produce the bluish coloration of the reagent; for instance, potato and other vegetable substances, preparations of iron, bile, saliva, milk, and pus. By employing the acidulated ethereal extract, Weber claims to avoid this difficulty: A sample specimen of the feces or stomach contents is well stirred with one-third its volume of glacial acetic acid. The mixture is then shaken with ether. A little alcohol will hasten the clarifying of this ether extract. A few cubic centimeters are poured off, and 10 drops of a freshly prepared tincture of guaiac and 20 to 30 drops of oil of turpentine are added. If

FIG. 166.—Direct-vision hand spectroscope.

blood is present, the mixture will turn violet blue; if not, it turns red brown, with often a slightly greenish tinge. The reaction is more marked if we add some water and then extract the blue pigment with chloroform. In healthy individuals the feces never react positively to this test. Ewald[2] was able to demonstrate an admixture of blood with this method even in gastric contents which were not colored, especially in cancer of the stomach. Weber found the test sensitive enough to demonstrate blood-pigment in the daily movement of a healthy individual after the ingestion of only 3 centimeters of blood.

According to Boas,[3] the guaiac test for blood does not always yield decisive results in the examination of the feces, since the blue color is often veiled by the brown shades and rendered indistinct. For this reason Boas recommends a control test with aloin, as suggested by Klunge, Schär, and Rossel. According to Rossel,[4] the test is performed as follows: 5 c.c. of the stool are extracted with 20 c.c. of ether, in order to remove the fat, which would subsequently interfere with the test by the formation of emulsions. After the removal of the ether, 3 to 5 c.c. of acetic acid are added to the feces, and the mixture is again extracted with ether in a test-tube. The acid ethereal extract obtained in this manner is then employed for the investigation. A solution of aloin is prepared by dissolving as much aloin as can be placed upon the point of a small knife in from 3 to 5 c.c.

[1] *Berlin. klin. Woch.*, 1893, No. 19.
[2] "Ueber occulte Magenblutungen," *Deutsch. med. Woch.*, 1901, No. 20.
[3] *Ibid.*, 1903, No. 47.
[4] *Arch. f. klin. Med.*, vol. lxxvi., p. 505, 1903.

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of 60 to 70 per cent. alcohol. To the acetic acid ethereal extract is first added 20 to 30 drops of a resinous oil of turpentine, and then 10 to 15 drops of the solution of aloin; if the stool contain blood the resulting mixture soon becomes bright red, and upon standing for a time assumes a cherry color. If no blood be present the aloin solution remains yellow for at least one to two hours, and then acquires a slightly reddish tinge. According to Boas, the aloin reaction may be markedly accelerated by the addition of a few drops of chloroform. With this modification,

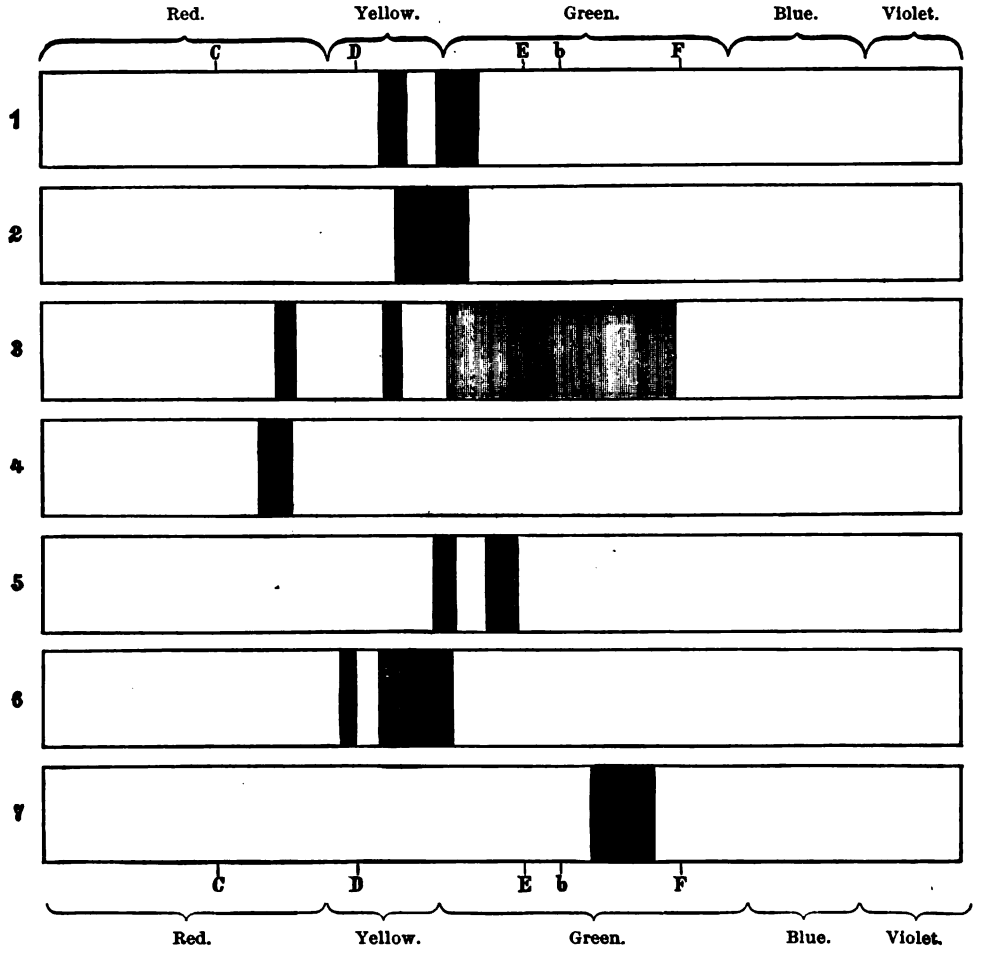

FIG. 167.—Important clinical spectra: 1, Oxyhemoglobin; 2, reduced hemoglobin; 3, methemoglobin; 4, hematin in acid alcoholic solution; 5, reduced hematin in alkaline solution; 6, hematoporphyrin in acid solution; 7, urobilin (after Salkowski).

agitation of the mixture results in the formation of reddish droplets which settle in the bottom of the tube as an intense red precipitate. Under certain conditions Boas regards the aloin test as more sensitive than the guaiac test. According to Brandberg, the oil of turpentine may be replaced by a dilute solution of hydrogen peroxid.

An ordinary hand spectroscope (direct-vision, after Browning, Fig. 166) is sufficient for the *spectroscopic detection* of the derivatives of the blood-pigment. The substance must be well diluted with water and examined in the sunlight. The

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spectroscope should first be adjusted by moving the inner tube so that Fraunhofer's lines can be easily seen if the instrument is held toward a white surface or the sky. In order to avoid side lights and to make an examination with an ordinary test tube we employ the apparatus "*B*" (see figure), made of brass and blackened on the inside. This can be shoved over the prism end of the spectroscope, and fastened by means of the screw, *s*. The test tube, with the substance to be examined, is placed in the cross-tube *C*, and the light is admitted through *B*.

The normal pigments of the feces, even when considerably diluted, frequently mask the characteristic spectrum of the hemoglobin derivatives by their diffuse absorption of light; hence a mere watery dilution of the feces is not to be recommended in doubtful cases. The following method is more desirable: Several cubic centimeters of the stool to be examined are mixed with water and acidulated with several drops of sulphuric acid until the Congo-red reaction is masked (see p. 372). The mixture is then filtered, and the filtrate extracted with ether. The extraction may be hastened by adding a few drops of alcohol. If the feces contain blood the ether turns reddish brown and shows spectroscopically the characteristic bands of acid hematin (see Fig. 167).

If the fluid to be examined is alkaline, we need only to acidulate with a little acetic acid to obtain the spectrum of the acid solution. Conversely, the spectrum of the alkaline solution can be easily obtained from the acid by adding soda solution. Of course, the hemoglobin of muscle fiber gives the same reaction and the same spectrum as the hemoglobin of blood, so that we must be careful to exclude any mistake by seeing that the patient does not ingest any large amount of raw or half-cooked meat.

URINARY EXAMINATION.

AMOUNT OF URINE.

THE daily volume of urine excreted by a healthy adult varies under average conditions between 1500 and 2000 c.c.[1] An abundant ingestion of water will considerably increase, and a diminished ingestion will correspondingly diminish, the amount. The amount will vary in inverse ratio to the insensible perspiration; hot weather diminishes and cold weather increases the amount. Profuse perspiration, diarrhea, and vomiting all decidedly diminish the amount. In children or abnormally small individuals the amount of urine is correspondingly less. The normal average for such a person can be determined from the following formula, supposing the average weight of a healthy adult to be 75 kilos (165 pounds):

$$x : 1500\text{–}2000 :: a : 75,$$

where x represents the daily amount of urine sought and a the weight of the individual in kilos. Children, and especially nursing infants, on account of the preponderance of liquid in their food, excrete proportionally a larger amount of urine than is represented by the formula.

[1] [In this country the average quantity eliminated in twenty-four hours would fall considerably below this. The figures usually given are 1000 to 1500 c.c.—ED.]

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According to Martin and Ruge,[1] 67 per cent. of all newborn infants excrete urine the first day of life, but generally only after the lapse of twelve or more hours. The remaining 33 per cent. do not void urine until the second or the beginning of the third day. The daily amount of urine of the newborn varies between 150 and 200 c.c. Rancke, Bischoff, and others estimate the excretion per diem from the third to the fifth year as about 700 c.c.

As a rule, less urine is voided during the night than during the day (from one-fourth to one-half as much).

According to Quincke,[2] this condition is reversed—*i. e.*, more urine is excreted during the night than during the day in heart and kidney diseases, in the aged with arteriosclerosis, in cachexia, and in diabetes insipidus. Under these conditions the amount voided at night may be doubled that voided during the day. Not only the watery, but the solid constituents as well, are thus increased. Quincke regards this phenomenon as the result of a nocturnal recuperation of the damaged organs (heart and kidneys).

Polyuria means an increase, *oliguria*, a diminution, in the quantity of urine.

The quantity of urine in pathologic conditions depends, first, upon the condition of the secreting renal parenchyma, and, second, upon the rapidity of the blood-current in the kidneys (Heidenhain). It will therefore be affected by a general circulatory disturbance, as well as by disease of the kidneys alone. To alter it appreciably both kidneys must be diseased, for otherwise the healthy kidney would assume vicariously the total function, as regularly occurs in unilateral nephrectomy. The more acute the nephritis, the more the amount of urine falls below the normal; the more chronic the course of a nephritis, the more the amount exceeds the normal. Although as yet we have no definite explanation of the latter phenomenon, it is presumably due to a compensatory process.[3] This increase reaches a maximum in the true contracted kidney, where the volume has been found to be as high as 12 liters per day (Bartels). In regard to the amount of secretion, the so-called chronic parenchymatous nephritis resembles sometimes acute nephritis and sometimes the contracted kidney. A similar variation in amount is observed in amyloid kidney. Diseases of the heart and lungs, leading to passive congestion, are associated with a diminution in the amount of urinary excretion, which clearly depends upon a slowing of the renal circulation. In such disturbances the estimation of the amount of urine is therefore of great diagnostic and prognostic significance.

The increased excretion following convulsions (especially in hysteria—the so-called *urina spastica*) and after attacks of angina pectoris is probably to be attributed to some vasomotor influence. Many other alterations in the volume of the urine depend upon quantitative and qualitative variations in the substances thus eliminated, and are primarily induced by disturbances of metabolism—*e. g.*, the polyuria in diabetes mellitus. We do not know the cause of the polyuria in diabetes insipidus.

[1] Vierordt, *Daten und Tabellen*, 1888.

[2] *Arch. f. exp. Path. u. Pharm.*, vol. xxxii., parts 3 and 4.

[3] The increase of blood-pressure is frequently assumed as an explanation of the polyuria. This assumption seems to the writer unwarranted, since he has observed cases with long-continued polyuria and typical contracted kidneys in which the autopsy revealed absolutely no trace of cardiac change.

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FREQUENCY OF URINATION.

Although varying decidedly in different individuals, the frequency of urination corresponds in general to the amount of urine excreted. It is also influenced, however, by any inflammatory affection of the urinary tract or by any disturbance of innervation of the bladder. The increased frequency of urination from inflammatory conditions of the urinary tract depends upon a stimulation of the bladder reflexes (pollakiuria, bladder tenesmus). Although observed chiefly in affections of the bladder, and especially of its neck or of the urethra, this frequent voiding of urine may depend also upon kidney disease, resulting either from a pathologic reflex emanating from the kidney, or from the irritation of an abnormal urine upon the mucous membrane of the bladder. Diseases of the spinal cord which either increase the irritability of the bladder or weaken the sphincter are also responsible for an increased frequency of urination. An abnormally infrequent urination depends upon some mechanical or nervous obstacle to the emptying of the bladder. (See later, Examination of the Nervous System, Innervation of the bladder.)

SPECIFIC GRAVITY OF THE URINE.

For the sake of simplicity the specific gravity of the urine is ordinarily expressed in four figures, taking the specific gravity of distilled water as 1000 instead of 1. Special aërometers called urinometers are employed for this purpose. Their scale is marked from 1000 to 1050. To insure a correct reading the subdivisions should be sufficiently wide apart. Many urinometers are not very accurately constructed, so that it is always well to compare a new instrument with a reliable one; at least, it can be ascertained whether it reads 1000 in distilled water at the ordinary room temperature, 15° C. (60° F.). Although a very large urinometer is more accurate, a smaller instrument requires less urine, which sometimes is a distinct advantage.

In the determination of the specific gravity the urine is poured into a tall glass cylinder (a urinometer and an appropriate glass cylinder are generally obtained together), the urinometer is immersed in the urine, and then with the eye at the level of the surface of the urine the subdivision is read off which corresponds to the lowest part of the curve of the meniscus. For the sake of accuracy the cylinder should be sufficiently large to allow the urinometer free motion. The bulb of the urinometer should not be allowed to stick against the side of the vessel, all bubbles or froth should be removed from the surface of the urine by means of filter paper, and the urine should be at the ordinary room temperature. If the urine is colder than 15° C., one-third of the urinometer unit should be subtracted for every degree of Centigrade below the ordinary temperature; if warmer, the proportionate amount should be added. Since the specific gravity of individual specimens of urine varies so decidedly during the day, it is imperative that the specific gravity of a mixed sample from the total twenty-four-hour secretion be obtained if any definite conclusions are to be drawn from the results.

If the specimen is too small to work with in the ordinary way, it

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is a simple matter to dilute it with distilled water in a known proportion, to estimate the specific gravity of the mixture, and then to calculate the specific gravity of the urine at least approximately.

The specific gravity of a twenty-four-hour specimen of urine varies normally from 1015 to 1020. Copious beer- or water-drinking may bring it as low as 1002; excessive perspiration as high as 1040. The ingestion of food and liquids, or the loss of water from the skin or from the lungs, is responsible for the marked variations which occur in single specimens, as well as in the twenty-four-hour specimen.

Pathologically, the specific gravity is influenced by the secreting parenchyma of the kidney, by the velocity of the renal circulation, and by abnormalities of metabolism. In acute nephritis the urine is concentrated and of a high specific gravity; in chronic nephritis due to a true contracted kidney, the urine is profuse and of a low gravity. The specific gravity usually varies inversely with the amount of urine. Both under physiologic and pathologic conditions, therefore, a scanty urine is more concentrated than a more profuse one. Diabetes mellitus forms an exception to the rule, since the excretion of sugar produces a high specific gravity despite the excessive amount of urine; this is so characteristic that a diagnosis is often possible on this ground alone. Some types of nephritis are also exceptions to the above rule, such as those showing a tendency to uremia and concomitant diminution in the excretion of solids; these cases present a low specific gravity with a small amount of urine. Again, in some cachectic conditions, even without nephritis, diminished metabolism and the lessened ingestion of food as well as of water produce a fall in volume of urine and, at the same time, a low specific gravity.

The specific gravity is a pretty accurate indication of the amount of solids excreted in the urine. The solids excreted in 1 liter of urine can be approximately represented in grams by multiplying the last two figures of the specific gravity by 2.2337.[1] A urine of high specific gravity is called *concentrated*. The urea is mainly responsible for the specific gravity. For this reason alone, apart from the diminished secretion of water, a fever urine is always concentrated.

In reference to the value of the determination of the specific gravity of the urine as a substitute for the cryoscopic urinary examination, see p. 551.

TRANSPARENCY OF THE URINE.

Freshly voided normal urine is absolutely clear and transparent; but after it has been standing for some time an indistinct cloud of so-called mucin, "the nubecula," appears (see p. 468). Various other insoluble substances may cloud pathologic specimens of urine. (See the section on the Sediment and Turbidity of the Urine, p. 553 *et seq.*)

[1] Vierordt, *Daten und Tabellen zum Gebrauch für Mediciner*, 1888.

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COLOR OF THE URINE.

NORMAL URINARY PIGMENT.

The color of the urine varies normally within different shades of yellow, the depth of color increasing in direct proportion to the specific gravity. The abundant urine of a drinker or of a person with contracted kidneys may be as pale as water, as contrasted with the scanty urine of acute nephritis, of passive congestion, or of fever, which is almost always dark. In diabetes mellitus, despite the high specific gravity, the urine is exceptionally pale. This is of similar diagnostic importance as the great increase in amount. The urine in anemic individuals is always paler than normal, except in pernicious anemia. There the urine is quite dark, because this disease is associated with a rapid destruction of the red corpuscles, and, as is well known, the urinary pigments are derived from the hemoglobin.

The only method of designating the color of the urine is to compare it with certain fixed shades on a chart. In Neubauer and Vogel's book,[1] unfortunately, only a few different shades are represented. Radde's[2] scale can be used for other purposes as well, and is more highly recommended. Naturally, to obtain uniformity of results, a certain uniform thickness of the urine must be observed in each case, and the specimen must be held against a white background. For the qualitative determination of the normal urinary pigment (urochrome) see p. 504.

COLORING OF THE URINE BY PATHOLOGIC URINARY PIGMENTS.

Hemoglobinuria; Hematuria.—Urine which contains blood or hemoglobin presents a red, brownish-red, or sometimes even a greenish-black or black color. These variations depend partly upon the amount of admixed blood, and partly upon the degree of alteration of the blood-pigment in the urine. Urine containing hemoglobin proper is reddish, while that containing methemoglobin is more brownish. (See Spectroscopic Demonstration of Blood-pigment, p. 471 *et seq.*) Such urines are usually cloudy on account of the blood-corpuscles and other organic mixtures, and in hemoglobinuria on account of flakes of hemoglobin. Upon standing, these elements all settle in the form of a sediment.

Hematoporphyrinuria (see p. 471).—Urine which contains hematoporphyrin is sometimes excreted after persistent use of sulphonal, trional, or tetronal. Its color, if the layer of urine is thick, is a very dark brown or almost a black; and if thin a yellowish red to violet.

With **jaundice** the urine contains *bile pigments*, and its color varies from a dark yellow or green to a brown or black, depending upon the concentration of the urine and the amount of bile pigment, or upon its

[1] Anleitung zur qualitativen und quantitativen Analyse des Harnes, older editions. In the more recent editions, edited by Hüppert and Thomas, that plate is no longer included.

[2] *Stenochromatische Anstalt*, Hamburg, 1877.

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chemical modifications. When such a urine, especially if strongly acid, is allowed to stand in the cold for some time, needle-like crystals of bilirubin separate out and settle with the rest of the sediment. (See p. 472, Demonstration of Biliary Coloring Matter; also p. 561 *et seq.*) The urine of patients suffering with **melanotic tumor,** a very rare condition, contains melanin (phymatorrhusin). It becomes dark brown to black after prolonged exposure to the air, the melanin having separated from the "melanogen" of the urine. As a rule, the urine is perfectly clear, although it may rarely become cloudy from a granular precipitate of the melanin. (See Demonstration, p. 477 *et seq.*)

The large amount of **urobilin** in the urine of some varieties of icterus (cirrhosis) shows a similar dark-brown color. (See Reaction, p. 478 *et seq.*)

Various **aromatic products,** such as result from decomposition by putrefactive processes (peritonitis, suppuration, etc.), are often responsible for a remarkably dark-colored urine. This color is sometimes seen even in a freshly voided specimen, but more commonly appears after exposure to the oxidation of the air. Either *pyrocatechin*, *alkapton*, or *indican* may give rise to a dark-colored urine. If much indican is present there is sometimes a conspicuous precipitation of indigo (see pp. 475 and 561). Although the bluish tinge of the indigo may be masked by the color of the urine, a bluish-black scum, due to the separation of the indigo, generally a rises to the surface. (See Demonstration of Indigo and Indican, p. 475 *et seq.*)

Indigo may be formed from indican even before the urine is voided. The author once obtained a specimen of grass-green urine from an apparently healthy boy without being able to determine the precise condition. The chloroform extract showed that the color depended upon the admixture of the blue of the indigo with the yellow of the urine.

MEDICINAL PIGMENTS WHICH COLOR THE URINE.

There are so many drugs which may be responsible for an abnormal color of the urine that it is possible to mention here only the most important. At the same time the writer wishes to emphasize the fact that, if the urine shows any peculiar or extraordinary color not otherwise explainable, we should consider the possibility of attributing it to the administration of some drug.

Carbolic acid, coal-tar products, hydroquinone, resorcin, pyrocatechin, naphthalene, salol, the *arbutin* derived from the leaves of *Uva ursi,* and many other aromatic substances, when administered internally, and sometimes even when employed externally, produce a dark olive-green to black urine. The dark color frequently becomes apparent only after considerable exposure to the air or after the urine becomes alkaline. It depends upon the formation of colored compounds by the oxidation of the excreted drugs. Hydroquinone and pyrocatechin are especially prone to result from such oxidations, and they cause the dark color of urines containing phenol, salol, and arbutin.

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The appearance of this color in the urine should occasion no alarm unless one of these drugs is being used externally; as, for instance, in the old Lister antiseptic employment of carbolic acid. Then the drug is evidently being absorbed in considerable and uncontrollable amount, although not intended to be absorbed at all. As a product of excretion simply, the dark color is of no particular significance. The dread of it is largely due to the frequency with which it was met after the use of the carbolic spray of Lister's method. Often quite moderate doses of salol (30 gr. (2 gm.) a day) will be accompanied by the excretion of a black urine without any ill effects, while doses large enough to cause toxic symptoms may cause no discoloration. These variations depend to a considerable extent upon the degree of acidity of the urine, as well as upon the length of time it is exposed to the air.

The administration of *chrysarobin, rhubarb, senna* or *cascara* may produce a yellow or reddish-brown urine, which will become distinctly red if the reaction is alkaline. (This is due to the presence of chrysophanic acid; see p. 503.) *Santonin* is apt to produce a saffron-yellow or greenish color, which changes to red if the urine is rendered alkaline. (See Test for Santonin, p. 503.) The pigment of *madder*, of *beets*, and of *huckleberries* may under certain conditions be excreted in the urine (Gorup-Besanez).

ODOR OF THE URINE.

Normal urine possesses a peculiar faint odor which is not particularly unpleasant. The disagreeable so-called urinous odor depends upon bacterial decomposition taking place either in the urinary tract or after the voiding of the urine. It is often called ammoniacal, since ammonia can usually be demonstrated in such a specimen. There are, however, other odorous substances concerned besides ammonia, as one can appreciate by the sense of smell—*e. g.*, aromatic decomposition products (phenol). Decomposing albuminous urine presents an odor so characteristic as to justify a definite diagnosis of the presence of proteid.

Decomposition in the urinary tract, or the absorption of hydrogen sulphid from putrefactive areas in the body, will sometimes produce an odor of sulphuretted hydrogen (hydrothionuria),

Various odorous substances if introduced into the body appear directly as such in the urine—*e. g.*, the odorous substances of valerian, leek, castor, crocus (saffron), asafetida, meat, bouillon, and coffee. Other substances form characteristic odorous compounds in the body about which little is understood, but which are excreted in urine—*e. g.*, balsams, especially the balsam of copaiba, cubebs, saffron, and the oil of turpentine. Even tiny doses of the last impart a very distinct odor of violets. The peculiar odor of the urine after eating asparagus is due to methylmerkaptan (Nencki).

REACTION OF THE URINE.

Normal urine has an acid reaction, due not to free acid, but to acid salts, especially dihydrogen sodium phosphate, H_2NaPo_4. At times the reaction may be amphoteric—*i. e.*, the urine may turn blue litmus paper red, and red paper a faint blue. This is due to the presence of the acid dihydrogen phosphate and the alkaline disodium phosphate in a definite relationship of such a nature that each salt is capable of exerting its influence upon the indicator without interference with the other. This

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is only possible provided the acid salt is not present in too great an excess.

Specimens of urine voided at different times of the day react differently, a variation depending largely upon the phenomena of digestion. During digestion the secretion of hydrochloric acid by the stomach diminishes the acidity of the urine, which may even become alkaline. This diminution of acidity is shown by a more ready precipitation of the earthy phosphates and carbonates, so that the urine is cloudy when first voided or when heated. The more hydrochloric acid secreted by the stomach, the more decidedly alkaline the urine simultaneously excreted, so that such a turbidity after eating oftentimes suggests a diagnosis of pathologic gastric hyperacidity. It is incorrect to consider the precipitation *per se* a manifestation of perverted metabolism inducing phosphaturia. If the acid contents of the stomach are vomited or removed by gastric lavage, the acidity may be still further reduced or the urine even rendered decidedly alkaline, although oftentimes without vomiting or lavage, the degree of hypersecretion alone is sufficient to cause quite a marked permanent phosphaturia and an alkaline urine. Perhaps the acid is excreted in the feces; but thus far we have no direct investigation of this point.

The nature of the food ingested will also alter the reaction of the urine. A vegetable diet or a liberal consumption of wine or of fruit may cause the excretion of an alkaline urine; the alkalinity is occasioned by the fact that the organic acids become oxidized in the body to alkaline carbonates. On the other hand, a diet rich in proteid or meat will intensify the acid reaction, because acids, especially sulphuric acid, result from the oxidation of proteid in the organism.

Alkaline drugs render the urine alkaline. All acids not oxidized to carbonic acid—*e. g.*, the mineral and aromatic acids—intensify its acidity. Most of the fatty series are oxidized to carbonic acid, and so, in moderate doses, do not usually increase the acidity. Alkaline bacterial fermentation of the urine outside the urinary tract is often responsible for an alkaline reaction, especially when the urine is kept in a warm place.

The following pathologic conditions either diminish the acidity of the urine or render it alkaline:

1. Abnormal conditions of gastric digestion.
2. The admixture of alkaline secretions from exudations of the urinary tract (cystitis, gonorrhea, rupture of abscess into the tract).
3. The rapid absorption of transudations or exudations, the alkaline salts of which are excreted in the urine.
4. The alkaline fermentation of the urine within the urinary tract.

To understand the significance of an alkaline reaction of the urine, it is important to determine whether this depends upon free ammonia or upon a fixed alkali. If red litmus paper placed near the surface of the urine is turned blue without being immersed, or if, when a glass rod dipped in dilute hydrochloric acid is held over the surface of the urine, white fumes of ammonium chlorid are produced, or if we note a strong ammoniacal odor, the reaction is caused by a volatile alkali, probably

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ammonia. Moistened red litmus paper suspended for a sufficiently long time, one-quarter to one-half hour, over a normal acid urine will become blue, showing that a certain amount of ammonia, even without any apparent fermentation, is given off by a freshly voided acid urine; but this blue color will disappear when the paper is dried. It is characteristic of urines which owe their alkalinity to fixed alkalies that only upon the immersion of red litmus paper in the urine will the paper become blue, and that this color is permanent even upon the application of heat.

Whenever the reaction of the urine is alkaline from free ammonia, it is safe to assume that the condition is the result of the decomposition of the urine or ammoniacal fermentation. To decide whether this has taken place within or outside the urinary tract, it is necessary to test the urine directly after it has been voided.[1]

The alkaline fermentation of the urine brought about by the action of micro-organisms is characterized by the change of urea, $CO<^{NH_2}_{NH_2}$, into $NH_2\,COONH_4$ and $(NH_4)_2\,CO_3$, with the taking on of one or two molecules of water, as the case may be. These latter two substances, in consequence of their instability, easily break down and give off ammonia. The alkaline reaction due to fixed alkalies is never caused by decomposition of the urine, but by other conditions already mentioned.

The urine of dogs has been rendered alkaline (without the aid of bacteria) by administering an excessive amount of milk of lime. The normal acidity of the urine may be pathologically increased by an increased decomposition of the body proteids, especially in fever. The cause of the acid in this case is the same as in the ingestion of food rich in proteid.

It is not yet definitely determined whether patients with the so-called uric acid diathesis excrete an abnormally acid urine. (See 541 *et seq.* for the Quantitative Estimation of the Reaction of the Urine; Acidimetry; Alkalimetry).

SEPARATION OF THE URINES OF THE TWO KIDNEYS.

Modern renal surgery frequently demands the separate examination of the urine from each kidney. This requirement is most exactly met by ureteral catheterization by means of the cystoscope, for the details of which the reader is referred to the text-books of cystoscopy.

This method requires special technical training, and may be replaced to a certain extent by the use of the so-called urinary separators which have recently been devised. All these instruments depend upon a similar principle: the bladder is emptied, a sagittal septum is introduced into the viscus, and the separate urines are withdrawn by catheters appropriately attached to the sagittal septum.

[1] When it is not possible to examine the urine perfectly fresh, the addition of about one-third its volume of chloroform water (1:200), or of a few pieces of coarsely granulated camphor or thymol crystals, will preserve the specimen.

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The best instrument of this type seems to be that of Luys (Fig. 168), which Garré[1] describes as follows:

"It consists of two metallic grooves which may be united to form a catheter of about the caliber of 22 (French), and having the usual curve. Since these lateral metallic grooves embrace a third flat piece they form a two-way catheter, each side of which possesses two eyes at one extremity and a lateral discharge tube at the other. The middle piece is enclosed in a condom which may be made tense by a screw, so that it fills the concavity of the curve. The instrument is introduced closed, the urine in the bladder is drawn, the septum is made tense by turning the screw, and, since the bladder is divided sagittally into halves, the urine from each kidney may be obtained separately. Handy accessory appliances make it possible to attach separate vessels to receive the urines from the discharge tubes. The instrument may be employed in the female with extraordinary ease, and its use in the male is not attended with great difficulty or much annoyance to the patient. In male subjects with enlarged prostates a space may be left between the gland and the catheter, which is not drained upon the introduction of the instrument, and this mixed urine may consequently interfere with the accuracy of the method. This source of error may be avoided by introducing the finger into the rectum and pressing the base of the bladder against the catheter. When the screw is reversed the tension chain and the condom are accommodated between the grooved halves, and the instrument may be easily withdrawn. The apparatus works best with the patient in a sitting or semirecumbent position."

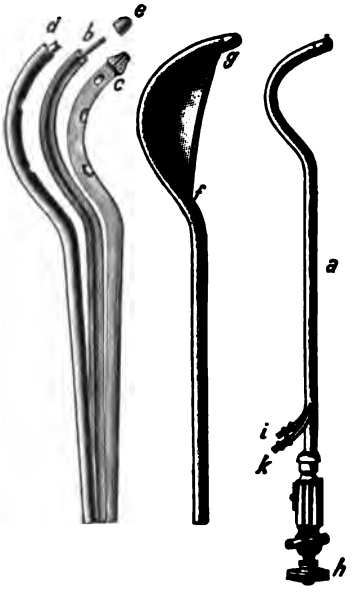

FIG. 168.—*a*, The composite instrument ready for introduction; *i* and *k*, discharge tubes; *h*, screw to regulate the tension of the membrane; *b*, flat middle piece; *c* and *d*, grooved lateral portions; *e*, tip uniting the parts; *g f*, rubber membrane, tense. The chain is not visible in the figure.

[We have found Cathelin's instrument more reliable. The sound is, however, larger, and hence more difficult to insert through a narrow meatus.—ED.]

Opinions differ as to the reliability of the urinary separation. Practice possibly plays a large rôle. The results are certain, however, when one discharge tube furnishes are entirely normal, and the other a pathologic, urine. If, on the contrary, both urines are abnormal, the possibility of an incomplete separation must be considered and cystoscopy finally employed.

QUALITATIVE CHEMICAL EXAMINATION OF THE URINE.

EXAMINATION FOR PATHOLOGIC CONSTITUENTS.

ALBUMINURIA.

The clinical expression *albuminuria* applies merely to urines which contain albumin or globulin (coagulable proteids) and does not include nucleo-albumin, proteoses, nor peptone.[2]

[1] *Therapeutische Monatshefte*, 1903, No. 1.

[2] It is customary to designate as albumin any proteid-like substance which appears in the urine and which responds to the ordinary "tests for albumin." On this account the term albumin, although it signifies a definite class of proteids, has become synono-

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Albuminuria may be subdivided under two heads: (1) The *true* or *renal albuminuria*, in which the albumin is excreted by the kidneys themselves; and (2) *false* or *accidental albuminuria*, in which the albumin comes from the admixture of some albuminous exudate, blood, or lymph with the urine in its course through the urinary tract. To distinguish between these two forms, it is necessary to examine the urinary sediment under the microscope and to consider carefully the entire clinical picture.

Renal albuminuria always depends upon an abnormal permeability of the epithelium, especially of the glomeruli, which permits the proteids of the blood to escape into the urine. The vessels themselves cannot be responsible, because physiologically they permit the transudation of albuminous liquid in the formation of lymph. Whether the passage of the proteid through the renal epithelium is always pathologic, or, in other words, whether a so-called physiologic albuminuria can actually exist, has created considerable discussion, and some misunderstanding of terms has complicated the question. Albuminuria is usually considered the most important symptom of the diffuse kidney disorders called Bright's disease. Hence the term "physiologic" has been applied to cases of transitory albuminuria which are associated with no other symptoms, and whose subsequent development proves the absence of any real nephritis or other disturbances. Examples of this form are the albuminuria observed in apparently healthy individuals, and the so-called "cyclic albuminuria" found at certain hours of the day (generally after rising), especially in the young. As a matter of fact, the term "physiologic" means merely that this type of albuminuria is not at all serious, but it is hardly an appropriate term. Just because a biologic phenomenon is harmless, it is not necessarily physiologic. It seems to the writer that we are quite as justified in designating as physiologic a catarrhal process which causes an individual no discomfort, as we are in assuming such an excretion of albumin to be physiologic. It is not physiologic, but rather due to some slight affection of the renal epithelium which causes a harmless albuminuria in otherwise healthy people, unassociated with any grave anatomic renal change.[1] This conception is of considerable importance from a practical standpoint, because the kidney of an individual with physiologic albuminuria would naturally be regarded as rather sensitive or even predisposed to actual disease, and so suggest to a careful physician appropriate prophylaxis. Perhaps it holds the same relation to actual nephritis as the alimentary glycosuria bears to actual diabetes mellitus.

Definite pathologic forms of albuminuria must certainly depend upon some affection of the renal epithelium, which is most pronounced in

mous with proteids, and the word is so employed in the text. As is well known, however, the proteids in the urine may consist of globulins, proteoses, etc., to the exclusion of albumins. For further characterization of these proteid bodies see Cohnheim ("Chemie der Eiweisskörper," *Text-book of Physiologic Chemistry*) and Hammarsten.

[1] According to recent researches the minute traces of proteid found quite commonly in the urine of healthy people are neither albumin nor globulin, but nucleo-albumin, which has certain reactions in common with the former (see p. 468 *et seq.*).

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inflammatory and circulatory disturbances of the kidneys (nephritis and in amyloid disease of the kidneys. But the renal epithelium also may be damaged sufficiently to permit the transudation of albumin in *local* or *general anemia, cachexia, venous congestion, transitory renal ischemia, temporary occlusion of a ureter, prolonged retention of the urine in diseases of the bladder or of the spinal cord, the circulatory disturbances accompanying attacks of epilepsy, temporary compression of the thorax* and *prolonged cold baths*. To the same category the so-called febrile albuminuria observed in various fevers belongs.

Nephritic albuminuria varies decidedly with the type of the disease. In acute nephritis a large amount of albumin, sometimes 2 per cent. or over (though generally not more than 1 per cent.), is excreted; in chronic nephritis the amount varies. The more the disease approaches the type of a true contracted kidney, despite the severity of the disease the smaller is the amount of albumin excreted; and in an extreme chronic case it may even disappear temporarily.

The same variability and temporary disappearance may characterize *amyloid disease*.

The amount of proteid in *passive congestion* is in most cases not very great. There are, however, exceptions, in which the amount is large. In these cases the amount of proteid corresponds closely to the diminution in the volume of the urine resulting from the congestion, whereas in nephritis, on the contrary, the degree of albuminuria does not correspond in any well-defined way to the quantity of urine. A large amount of albumin and a relatively large volume of urine are hardly ever combined in passive congestion; but such a combination is not uncommon in nephritis, although ordinarily only in very serious conditions of disease.

Febrile albuminuria rarely occurs except in the severe infectious diseases, where the quantity of albumin may become so considerable that it is very difficult, perhaps impossible, to make a differential diagnosis between such albuminuria and actual nephritis.

DETECTION OF PROTEIDS AND RELATED SUBSTANCES IN THE URINE.

Recent opinions regarding the nature of the proteids and their derivatives have altered so materially that the reader is recommended to study the annexed table to comprehend the differences and mutual relationships of such of these substances as may occur in the urine. (See Table facing this page.)

Detection of the Ordinary Coagulable Proteids of the Urine (Serum-albumin; Serum-globulin).

(The albumin test in the usual sense of the word.)

Heat Test for Albumin.—This test depends upon the principle that in weakly acid or neutral solutions albumin will be coagulated at the boiling-point. A test tube of urine is heated to the boiling-point preferably only at the upper portion. If a precipitate (cloudiness

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of the Urine.[1]

	. Peptone (Kühne). End product of the peptic and tryptic digestion of 1, 2, 3, and 4 (appearance in the urine through newer experimentation has become doubtful).	Compound Proteids. Extremely complicated combinations, which may be decomposed by boiling with diluted acids, or by peptic degeneration into a proteid and another substance (carbohydrate, pigment, nuclein).		
		9. Hemoglobin (Blood-pigment) breaks down by boiling with diluted acids into hematin and proteid.	**10. Nucleoproteid** breaks down by peptic digestion into nuclein, and proteid contains phosphorus.	**11. Mucin.** Presence in the urine through newer experimentation has become doubtful; breaks down by boiling with diluted acids into reducing carbohydrate and proteid.
Water.	oluble.	Soluble.	*Insoluble.*	Insoluble.
Boiling	*Not coagulated.*	*Coagulated and decomposed.*	*Coagulated.*	?
Alcoho	Precipitated.	Precipitated and coagulated.	Precipitated (partly changed).	Precipitated.
5 to 1 salt	oluble.	Soluble.	?	?
Saturat with	Not precipitated.	Not precipitated.	?	?
Saturat with	Not precipitated.	Not precipitated.	*Precipitated.*	*Not precipitated.*
Saturat with	*Not precipitated.*	?	?	?
Dilute	Not precipitated.	Decomposed.	Partly soluble, partly swelling into slimy masses.	Soluble.
Dilute	Not precipitated.	Decomposed.	Easily soluble in mineral acid, very difficultly in acetic acid.	Soluble in mineral acid, not in acetic acid.
Concen	Not precipitated.	Decomposed.	Soluble.	Soluble.
Metap	Not precipitated.	Decomposed.	?	?
Nitric	Not precipitated.	Decomposed.	Precipitated, soluble in excess.	?
Potass + a	*Not precipitated.*	Decomposed.	*Precipitated.*	?
Comm	Not precipitated.	Decomposed.	Incompletely precipitated.	?
Tannic acid	Precipitated.	Decomposed.	Precipitated.	?
Trichl	Not precipitated.	Decomposed.	?	?
Phosp in m	Precipitated.	Decomposed.	Precipitated.	?

this synopsis in contrast to "precipitated," if the precipitate, produced by heating

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appears, it will consist either of albumin or of phosphates and carbonates, for the latter are also precipitated by heat (see p. 557 *et seq.*). A distinct flaky precipitate would probably indicate albumin; but to be certain the urine should be acidified drop by drop (dilute acetic acid 1 : 9). If the precipitate then dissolves, it was due to phosphates and carbonates. If it does not dissolve, but, on the contrary, becomes more flaky and heavier, it is albumin. The heat test will not always cause a precipitate in an albuminous urine which is alkaline or only very faintly acid. This is the reason why the acid is added drop by drop until the urine becomes distinctly acid. It is generally advisable to employ a dilute solution of acetic acid, because, although an alkaline urine may require considerable acid, too strong acid might allow a moderate amount of albumin to escape notice by being rapidly changed to the soluble acid albumin. Again, acidulating the urine too strongly before boiling may entirely prevent the precipitation of the albumin. In this way many cases of mild albuminuria may escape detection. If slight in amount, albumin often does not precipitate from the urine until after the lapse of some time. This is true in other tests as well as in the heat test.

The reason it is advisable to heat only the upper portion of the urine in the test tube is because in this way we can differentiate a cloudiness due to albumin even when the urine, before heating, was not perfectly clear. In case of marked turbidity, however (such as would be caused by bacteria, pus, or excess of phosphates), the urine should be filtered before an exact test is attempted. If filtration does not succeed in clarifying the urine, it is advisable to add calcined magnesia and then filter again. This powder will clog the pores of the filter mechanically, and assist in clarifying a cloudiness, even that which is caused by bacteria. Cloudiness due to a considerable excess of urates will not complicate this test, because heating the urine dissolves the urates and clarifies the urine before the albumin is coagulated.

Another method of performing the heat test is first to acidify the urine strongly with several drops of dilute acetic acid, and then add one-sixth of its volume of concentrated salt solution (30 salt to 100 water). If much albumin is present a precipitate will form even in the cold mixture and decidedly increase upon heating. The advantage of this method is that it shows the presence of albumoses, since the latter are precipitated in the cold mixture and dissolved by heating. If both albumin and albumoses are present, the latter will be precipitated by cooling the filtrate from the boiled specimen.

The nucleo-albumin in normal urine will be precipitated by either heat test. If abundant enough to produce a cloudiness upon boiling, it may be mistaken for true albumin. This confusion may readily be avoided by adding a few drops of concentrated acetic acid to a cold specimen of urine. If this acidulation produces a cloudiness of the same intensity in the cold as in the boiled urine, the precipitation in both is due to nucleo-albumin; but if the cloudiness is of less intensity,

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the precipitation must depend upon a combination of nucleo- and true albumin.

The ordinary heat test is one of the safest and most accurate methods of determining the presence of coagulable proteid. (See below in regard to its advantages.) But we must remember that not only nucleo-albumin and albumose, but also resinous acids, may be precipitated by acidifying the urine too strongly. (See below for the Nitric Acid Test Relative to the Differentiation Between Resins and Proteids.)

Tests for Albumin in the Cold.—A large variety of chemical reagents are capable of precipitating albumin even at a low temperature. Requiring no alcohol lamp, these tests are of distinct advantage to the practitioner. The best known are:

The Nitric Acid Test (*Heller's Test*).—One-half-inch of concentrated nitric acid is placed in a test tube, and filtered urine is allowed to flow down the sides of the tube and stratify itself upon the acid. Albumin, if present, causes a precipitate at the line of junction of acid and urine and forms a ring or zone of cloudiness. If, however, there is only a very small amount of albumin present, this precipitate may not separate until after the lapse of several minutes.

Under certain conditions—*e. g.*, a very concentrated urine—the uric acid or urates may be precipitated by this test; but if the zone is quite flocculent it will probably be due to albumin. To be absolutely sure the specimen should be gently heated (not boiled), when the ring, if due to urates or uric acid, will promptly disappear. Or, before testing, the specimen can be diluted with two or three times its volume of water, in which case nitric acid will precipitate only albumin. Moreover, the uric acid ring is usually broader, less sharply defined along its upper border, and often plainly situated in the urine itself above the line of junction of the two fluids.

After the internal administration of some of the balsams the nitric acid test sometimes brings down a precipitate of resinous acids, which may be confused with that of albumin. Such a precipitate, however, will be to some extent cleared up by heat. Or, if the nitric acid and urine are mixed and the liquid then sucked up from the precipitate with a pipet, the precipitate, if composed of resinous acids, will dissolve in an excess of alcohol. According to Tappeiner, it is sufficient to add two volumes of alcohol to the mixture of urine and nitric acid without first separating the precipitate.[1] Alexander has objected that certain proteids precipitable by nitric acid will under certain conditions be dissolved in alcohol, as well as resinous acids. He recommends the solution of the precipitate by the addition of ether, although a great excess of the latter will be necessary to prevent the formation of an emulsion. A precipitate soluble in ether is due only to resinous acids. The heat test also permits a differentiation, for the resinous acids are not precipitable by heat unless the urine has been very strongly acidulated with acetic acid. Alexander performs a control test by adding to the heated urine one-third its volume of nitric acid; any cloudiness must

[1] *Deutsch. med. Woch.*, 1893, No. 14, p. 323.

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then be due to albumin, since the resinous acids could not be precipitated because of the heat and the considerable excess of nitric acid. Another characteristic of the resinous acids is that the addition of a few drops of hydrochloric acid produces a cloudiness of the urine. (See p. 503, Balsams of Copaiba and Santalwood Oil.)

Albumoses and nucleo-albumin are also precipitated by the nitric acid test; but the latter is dissolved by an excess of acidity, and the former by heating.

Under certain conditions nitric acid precipitates the bile acids, and in very concentrated urine the urea, as urea nitrate. To prevent this and the other sources of error mentioned above, Hammarsten suggests a dilution of the urine to a specific gravity of 1005 or lower. Heller's test then precipitates only albumin, nucleo-albumin, and albumoses, because, unless the urine is concentrated, neither uric acid, urea, the bile acids, nor the resinous acids can be precipitated. Albumose can then be excluded if the precipitate persists after warming; nucleo-albumin can be excluded if the precipitate does not dissolve in an excess of nitric acid, or if the urine remains clear after an addition of an excess of acetic acid.

Test with Acetic Acid and Potassium Ferrocyanid.—The specimen is strongly acidified with acetic acid, and a solution of potassium ferrocyanid (1 : 10) is then added drop by drop; the slightest amount of albumin will cause the appearance of a cloudiness or the precipitation of small flocks. This reaction is one of the most trustworthy of all the tests for proteid. Nevertheless, the albumoses and nucleo-albumin will also be precipitated (the former, however, only when present in considerable concentration). In the preceding paragraph we have already detailed the method of differentiating them from the albumin.

Metaphosphoric Acid Test.—If a small particle of this substance (HPO_3) is dropped into albuminous urine the proteid will be precipitated. The test is of little value, because it applies only to fairly large amounts of proteid, 1 per cent. or more. The same precautions as before must be observed in order to distinguish urates, uric acid, and resinous acids. The only advantage this test possesses is that the metaphosphoric acid can be readily transported as a solid, although it must be kept sealed to prevent the absorption of water and a resulting change to the ordinary phosphoric acid, which does not precipitate proteid.

Picric Acid Test.—Picric acid, either in the solid substance or in solution, may be used like metaphosphoric acid. It precipitates albumoses, peptone, nucleo-albumin, resinous acids, and sometimes uric acid and urates.

Since with these cold tests so many precautions are necessary, it becomes evident that the practitioner will select the ordinary heat test for albumin (p. 460 *et seq.*) as being the most reliable and the simplest.

Appendix.—*To Remove Proteid from the Urine.*—This is often necessary before many of the following qualitative and quantitative tests can be performed. The best method is to effect an insoluble combination with it and some metallic oxid. Hofmeister adds 10 c.c. of a saturated solution of sodium acetate to half a liter of urine, and into this mixture stirs a solution of ferric chlorid, drop by drop, until the color becomes blood red. Such a mixture, which is strongly acid, is then exactly neutralized with sodium or potassium hydroxid, or at most allowed to remain very faintly acid, and then boiled, cooled, and filtered. The filtrate should be free from proteid and iron. (Test with potassium ferrocyanid.) The method is of no value for a urine containing sugar, because the oxid of iron would be kept in solution.

It is usually sufficient to boil an acid urine until the proteid is coagulated, and then to remove the latter by filtration. If the urine is alkaline or neutral it

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should be slightly acidulated with dilute acetic acid. When the proteid coagulates as a sort of cloudiness rather than in flocculi, it is advisable to add a little more acetic acid, while still boiling, until large flocks form. If coagulation by boiling does not produce coarse flocks, filtering will not completely remove the proteid. If the acid reaction is too faint, or if too much acetic acid is added, the proteid may be prevented from separating in large flocks. Unless another specimen of the urine is used, the mistake of adding too much acid may be corrected by carefully neutralizing the excess of acid by the addition of dilute sodium hydroxid. If the amount of proteid is large, it is a good plan first to dilute the urine with water before attempting to make a separation. The only way to be certain that the proteid has been completely removed is to be sure that the addition of acetic acid and potassium ferrocyanid to the filtrate does not produce any cloudiness. If any turbidity still persists it is due to the addition of too much or too little acid, and the test should be performed again, varying the acidity slightly.

Detection of Serum or Paraglobulin.

Globulin always seems to accompany serum-albumin in urine, and not to be present without it. Any diagnostic significance of the presence of globulin in urine has, therefore, not yet been found. To demonstrate globulin we add enough ammonia to make the urine neutral or faintly alkaline. This is done in order to precipitate the phosphates, which, in the subsequent treatment with ammonium sulphate, would produce a turbidity. Then we filter, and add to the filtrate an equal volume of saturated ammonium sulphate solution. After the resulting precipitate has settled well (one hour) it is collected upon a filter, and then washed with a half-saturated ammonium sulphate solution until the filtrate is proteid-free. The precipitate contains the globulin, and under certain conditions albumose as well. The albumin has not yet been precipitated, because this requires *complete* saturation with the reagent. (See Table, facing p. 460.) The precipitate is next dissolved in a little water and the filtrate heated over a water bath. This will coagulate the globulin, fibrinogen, and albumose. This precipitate is filtered, washed with water, and then digested and dissolved in a 1 per cent. solution of soda over a water bath. If necessary the resulting solution can be filtered again, and then carefully neutralized with acetic acid. If any globulin or fibrinogen was originally present, an albuminate will be precipitated which will not be dissolved by the addition of a solution of sodium chlorid. If the deposit consists of albumose, acetic acid either gives no precipitate whatever or the precipitate is dissolved by a solution of sodium chlorid. Solutions of fibrinogen differ from those containing globulins in that the latter are not coagulated by the addition of fibrin ferment (fresh blood-serum). This difference, however, is of no practical value in urinary examinations, as will be observed in the subsequent observations upon the demonstration of fibrinogen in the urine.

Detection of Fibrinogen.

Fibrinogen belongs to the globulins, and is precipitated like serum-globulin (see above). The test for it is practically very simple, however, because fibrinogen will coagulate spontaneously in the presence of lime salts and fibrin ferment. Now when fibrogen is present in the urine, these

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last two factors necessary for coagulation are never absent, so that if a coagulation occurs spontaneously when the urine is allowed to stand, the presence of fibrinogen is always indicated. This phenomenon is usually associated with a *considerable admixture of blood in the urine.* The coagulation then resembles an ordinary blood-clot. Spontaneous coagulation occurs very exceptionally in the urine without an admixture of blood. So far as we know now this has been observed only in *tropic chyluria* and in *very rare cases of nephritis.*

Detection of Fibrin.

Fibrin forms in the urine when the latter contains fibrinogen. It occurs in the form of small clots, which are easily recognized under the microscope by their shredded structure, and are oftentimes tinged with blood. These physical peculiarities and the fact that it swells in dilute acetic acid will suffice for its recognition.

Albumosuria (Propeptonuria; Peptonuria).

The terms *albumosuria, propeptonuria,* and *peptonuria* should be considered synonymous and all three included in the term *albumosuria* (better proteosuria), because the term peptonuria was applied at a time when peptones and albumoses had not been differentiated, and because so-called "*true peptone,*" which has since been distinguished from the albumoses by Kühne, has not been absolutely proved to occur in the urine. (See Table, facing p. 460). Another reason why *albumosuria* and *peptonuria* should be included under the one term is that Kühne's separation of *true peptone* from the albumoses is somewhat artificial and based upon one single reaction, the precipitation of the *albumoses* by ammonium sulphate and the non-precipitation of *true peptone.* Moreover, even this reaction does not permit of any sharp distinction between them, for certain of the albumoses are not completely precipitated.

The *albumoses* have been found in the urine both by themselves and combined with albumin *during the puerperium,* in *acute yellow atrophy of the liver,* in *phosphorus-poisoning,* in *ulceration of the stomach and intestines* (enterogenic albumosuria), in *most febrile conditions,* especially in the *infectious diseases,* particularly the *suppurating processes* (pyogenic albumosuria), in *pneumonia* during resolution, in *scurvy,* in *nephritis,* and in *multiple medullary osteosarcoma.*

The presence of the *albumoses* in the urine is of clinical importance only when the urine does not contain albumin, because in albuminuria the albumoses are almost always to be found in the urine after removal of the albumin. It then appears questionable whether they existed preformed or were formed from the albumin by the chemical processes employed for its removal.

The presence of albumoses alone in the urine is of only limited diagnostic value. But when it is very pronounced, and when there are no other reasons for albumosuria, it may lead to the recognition of *deep-seated suppuration.* This symptom must therefore be considered in the

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diagnosis of perityphlitis, in the differential diagnosis between *tubercular and purulent meningitis*, in the diagnosis of *abscess of the brain*, and of *empyema of the pleura*, etc. A decided albumosuria is most suggestive of *multiple myeloma or myelogenic osteosarcoma* (Bence-Jones Albumose).

Detection of the Primary Albumoses by Employing the Ordinary Test for Albumin. Bence-Jones Albumose.—Albumoses may sometimes be recognized in the urine when the ordinary nitric acid test or the potassium ferrocyanid and acetic acid test for albumin proteid is employed. A precipitate is formed like that of true proteid, but differing from it by disappearing when heated. In performing the heat test for coagulable proteid, a slight turbidity appears in a case of this sort upon gentle heating, or even before heating, if the sodium chlorid modification of this test, described upon p. 461, is used. This precipitate dissolves again when the boiling-point is reached. If abundant the primary, and more especially the so-called Bence-Jones albumose,[1] may be detected in this way. The latter belongs to the primary albumoses, or, at any rate, is closely related to them (Kühne). A urine which responds positively to this test for albumoses usually reacts to the biuret test (p. 467).

Späth states that the following is a characteristic reaction, particularly for the Bence-Jones albumose (which is related to hetero-albumose): The urine should be distinctly acid and contain a sufficient quantity of sodium chlorid; it may therefore be necessary to add some acetic acid and a concentrated solution of sodium chlorid. The urine is then heated to 50° C. If it contains Bence-Jones albumose it becomes milky; at 60° C. a precipitate is thrown out, which is not flocculent, but tenacious and adherent to the side of the test tube, and which subsequently forms a granular mass floating upon the surface. If the heating be continued, the cloudiness and precipitate completely disappear and a clear solution remains, which upon cooling again throws down this proteid substance. When examined microscopically the precipitate consists of globules without crystalline structure.

Bence-Jones albumose is of especial importance in the diagnosis of myelogenic osteosarcoma or myeloma. It cannot, however, be regarded as pathognomonic of these conditions, since Askanzy[2] has demonstrated it in a case of lymphatic leukemia. This author's conclusions as to the value of the reaction are as follows: Bence-Jones albumosuria always indicates a lesion of the bone-marrow, and usually multiple myeloma, but in exceptional cases it may exist in diffuse lymphatic changes, such as are observed in lymphemia.

The above-mentioned tests are not adapted to demonstrate the presence of *deutero-albumoses*, to which most albumoses found in the urine, especially those present in febrile conditions, belong. Only when they are present in very concentrated amounts, which very rarely occurs, can they be precipitated by the method above, and then only slowly and incompletely. But the following method of testing for albumoses in general is also adapted to *deutero-albumoses:*

Salkowski's[3] Test for Brücke's Peptone, Albumoses, or Deutero-albumoses.—This is a modification of Hofmeister's early method. Fifty cubic centimeters of urine free from coagulable proteid are required. If the specimen contains nucleo-albumin, the latter may be precipitated with a little neutral lead acetate. A thick floccular precipitate appears (with which, by means of filtration, the nucleo-albumin is removed). "The filtrate is then placed in a beaker with 5 c.c. of HCl, precipitated with phosphotungstic acid, and then heated over a wire gauze. In a few moments the precipitate collects in a resinous mass at the bottom of the glass. The supernatant fluid is then decanted as completely as possible, and the resinous mass is washed twice with distilled water, which, if done carefully, may

[1] Matthes, *Congr. f. inn. Med.*, 1896. Here Bence-Jones albumose is differentiated from the other albumoses, Rosin, *Berlin. klin. Woch.*, 1897, No. 48, p. 1044.

[2] *D. Arch. f. klin. Med.*, vol. lxviii., parts 1 and 2, p. 34.

[3] *Centralbl. f. d. med. Wissen.*, 32, 113, 1894, and *Praktikum der physiol. Chemie*, 2d ed., 1900.

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be accomplished with scarcely any loss. We now add to the precipitate about 8 c.c. of water and 0.5 c.c. of sodium hydroxid (of about 1160 specific gravity). The precipitate now crumbles and easily dissolves when the beaker is gently shaken. The resulting solution, usually of a deep-blue color, is heated again over the wire gauze. It usually becomes turbid and dirty-grayish yellow; sometimes it turns yellow but remains clear. If the discoloration occurs slowly, it may be hastened somewhat by the addition of a few drops of sodium hydrate. When it is completed the liquid is poured into a test tube, cooled, and the biuret reaction[1] tried. For this purpose a dilute (1 to 2 per cent.) solution of copper sulphate is added drop by drop with shaking. If peptone is present the liquid will turn a decided red, and this color will be intensified if the liquid is now filtered. The whole process does not occupy more than five minutes. Another great advantage of the method lies in the fact that, owing to the small amount of urine required, nucleo-albumin is less liable to affect the reaction."

The addition of a few drops of barium chlorid solution has seemed to the writer distinctly advantageous when the solution upon which the biuret reaction is to be performed is already colored. After filtering off the resulting precipitate, the solution will be decolorized.

Quite recently Salkowski discovered a source of error in his own method which decidedly interfered with its practicability.[2] He found that the albumose reaction might be simulated if the urine contained much urobilin, for with the biuret test urobilin gives a color very much like that obtained with the albumoses. To make the value of a positive biuret test absolute, Salkowski therefore considers that the solution must first be shown to furnish no spectroscopic evidence of urobilin (p. 478). If the urine contains much urobilin besides albumoses, we should first extract with amyl-alcohol after acidifying, in order to remove the urobilin as completely as possible. By this plan, however, some loss of albumose can hardly be prevented. Employing barium chlorid, as suggested above, will sometimes, but not always, free the urine from urobilin.

v. Aldor[3] modified Salkowski's test in order to eliminate the urobilin. He separates the phosphotungstic acid precipitate by centrifugalization instead of by heating the fluid, then washes repeatedly with alcohol, which takes up the urobilin, and centrifugalizes again until it becomes absolutely colorless. The washed precipitate is then suspended in water and dissolved in sodium hydrate, and finally the biuret test is performed. If no centrifuge is available, the precipitate may be washed on filter paper with alcohol.

Detection of Albumoses According to Schulte's[4] Method.—The urine is first filtered, and any nucleo-albumin which may be present is precipitated by dilute acetic acid and removed by filtration. Then the tests for coagulable proteid are made—the heat test, ferrocyanid test, and Heller's test. Urine containing coagulable proteid need not be examined further, because it is of no clinical interest (see p. 465). If this is absent, 20 to 30 c.c., prepared as above, are dropped into six times the quantity of absolute alcohol and then allowed to stand for twelve to twenty-four hours. The solution is then decanted and the precipitate dissolved in a slight amount of warm water. After refiltering and testing the solution again for precipitable nucleo-albumin with very much diluted acetic acid, the biuret reaction is performed. The presence of urobilin in the urine (see Salkowski's method) does not, as a rule, give rise to any difficulty in this case, because when precipitation is accomplished the urobilin remains in solution. In any case the precipitate may be washed with absolute alcohol, and so completely freed from urobilin.

[1] The *biuret reaction* is a color reaction common to all proteids in solution. It is, however, especially marked with albumoses and peptones. It is performed as follows: After an excess of sodium or potassium hydroxid has been added to the solution of proteid, a very dilute copper sulphate solution is added. It will produce a violet color, the shade varying according to the kind of proteid present; with albumins it is a blue violet, with peptones and albumoses it is a red violet.

[2] *Berlin. klin. Woch.*, 1897, No. 17, p. 353. [3] *Ibid.*, 1899, Nos. 35 and 36.

[4] *D. Arch. f. klin. Med.*, 1897.

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Detection of Substances Resembling Mucin (Now Regarded as Nucleo-albumin, but Formerly Supposed to be True Mucin).

Most of what was formerly described as mucin in the urine is now recognized to be really nucleo-albumin. The two substances resemble each other very closely in their physical and chemical characteristics, but mucin is a glycoproteid and nucleo-albumin a phosphoproteid. Mucins are free from phosphorus, and when decomposed produce proteid and carbohydrate. Nucleo-albumins, on the contrary, contain phosphorus, and when decomposed furnish proteid and a group containing phosphorus (nuclein).[1] Despite so great a difference in their composition, the two substances are difficult to distinguish. The only simple differentiating reaction which the author has been able to find in literature is the precipitation of nucleo-albumin, and the non-precipitation of true mucin by magnesium sulphate. This statement needs, however, to be corroborated.

Further investigation[2] will have to determine whether nucleo-albumin and mucin are not both concerned in what we used to call offhand the mucus of the urine. For the time being it is justifiable to call the mucin-like substance nucleo-albumin.

Nucleo-albumin occurs in urine both physiologically and pathologically, partly in solution and partly (the larger share) undissolved. The latter constitutes the "nubecula," a slight cloudiness which is usually visible only after the urine has stood for some time. On account of its lightness it remains suspended in the urine, and is concentrated as a sort of cloud in the center of the vessel. An increased amount of this undissolved portion of nucleo-albumin constitutes what has always been known as "the mucous sediment." It is, of course, pathologic.

Nucleo-albumin can be demonstrated in every urine, although often not in sufficient quantity to be weighed. It possesses certain reactions in common with serum-albumin (see Table, facing p. 460—nucleo-albumin); hence the erroneous opinion that the presence of albumin in the urine might be physiologic (see p. 459, note). The amount of nucleo-albumin is increased in all affections of the urinary tract, particularly in *inflammation of the bladder* and *pyelitis*, in *nephritis*, and in several other disorders. An abundant mucous sediment occurs only in inflammation of the urinary organs, very likely because the nucleo-albumin is mainly a product of desquamation of the mucous membrane, and perhaps of some admixture of pus. This theory agrees with the observation that these mucous sediments always contain cellular elements.

Modern chemical investigations are now concerned almost entirely with that portion of the nucleo-albumin which is held in solution in the urine. The salts contained in the urine probably hold it in solution, for it seems to be insoluble in water.

To demonstrate the soluble nucleo-albumin, an excess of glacial ace-

[1] [The author has here confused the two terms nucleoproteid and nucleo-albumin. The former, upon decompcsition, splits off nucleins, but the latter does not.—ED.]

[2] Malfatti believes that he has demonstrated both mucin and nucleo-albumin in normal urine.

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tic acid is added to the urine. If the urine becomes cloudy, particularly if it has been previously diluted, nucleo-albumin is present. Dilution serves two purposes: it diminishes the solvent action of the salts and at the same time prevents the precipitation of urates so frequently noticed in concentrated urines. The addition of an excess of acetic acid precludes the possibility of confounding the precipitate with the globulins; for, although acetic acid may also separate the latter from their combinations with the alkalies, they will be immediately dissolved again by the least excess of the acid. Both uric acid and the resinous acids are precipitated by acetic acid, so that to avoid any possibility of confusing either of them with nucleo-albumin, it is advisable to make a control test with hydrochloric acid. This acid precipitates both these substances, but not the nucleo-albumin, or, at least, if it does, the least excess of HCl will promptly redissolve the nucleo-albumin.

The general proteid tests (heat test, p. 460 *et seq.*) and cold nitric acid test (p. 462) demonstrate nucleo-albumin. Perhaps this is one of the reasons for the erroneous supposition that serum-albumin occurs constantly physiologically in the urine. The distinction is simple enough, however, because an excess of the nitric acid dissolves the nucleo-albumin (on shaking the test tube), and because nucleo-albumin is precipitated by acetic acid, while serum-albumin is not.

Nucleo-albumin differs from serum-globulin in that an acetic acid precipitate of the former will not be dissolved upon the addition of an excess of the acid.

The actual character of the substance known as nucleo-albumin has been recently questioned again, since Mörner[1] regards it as a combination of serum-albumin with chondroitin-sulphuric, nucleic, or taurocholic acid, while Rostoski[2] concludes that it is a globulin. It probably consists of a number of substances, in which case the designation used at the head of this section, "substances resembling mucin," is to be preferred.

Detection of Hemoglobin (Blood Coloring Matter) and its Nearest Derivatives: Hematuria; Hemoglobinuria.

Blood Coloring matter as it appears in the urine may arise from the admixture of blood in the kidneys or in the urinary passages, or from the transudation of dissolved hemoglobin from within the vessels. In the first case we speak of *hematuria;* in the second, of *hemoglobinuria. Hematuria* is found in all kinds of inflammation of the kidneys or of the urinary tract, in new growths which involve any portion of the organs, and after some injuries.

Hemoglobinuria is symptomatic of *certain poisonings* (potassium chlorate, truffles, hydrogen sulphid or arsenid, pyrogallic acid, etc.). It was formerly observed after *transfusion of the blood of another species.* It also occurs after *burns,* in *severe infectious diseases* (rare), and finally as an independent affection in the form of the so-called *periodic hemoglobinuria.* Hemoglobin can often be recognized in the

[1] *Skandin. Arch. f. Physiol.*, vol. vi., p. 332, 1895.
[2] *Sitzungsbericht der physik-med. Gesellsch. zu Würzburg*, 1902.

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urine by its characteristic color (see p. 453). In *hematuria* the blood-corpuscles always make the urine turbid, whereas in *hemoglobinuria* the urine may be perfectly clear. The urine may, however, be turbid in hemoglobinuria, because it may contain casts (see p. 571) and granular masses of hemoglobin (see p. 561), and because, too, hemoglobinuria is usually combined secondarily with nephritic processes (casts, renal cells, red and white corpuscles). Nevertheless, in such cases, if the urine is allowed to settle, the clear supernatant fluid will still exhibit a bloody color. Of course, we must remember that after a hematuria urine has stood for some time many of the blood-corpuscles will dissolve. Hence, we need as fresh a specimen as possible to distinguish between hematuria and hemoglobinuria.

The presence of red corpuscles can be demonstrated under the microscope. (Compare Organized Admixtures and Sediments of Urine.) Hemoglobin itself, whether dissolved or still contained in the red corpuscles, can be shown to be present in the urine by the following means:

Chemical Detection of Hemoglobin.—The different derivatives of hemoglobin which are found in the urine react alike to the chemical tests. (Compare Spectroscopic Test, p. 471).

Heat Test.—In performing the heat test for coagulable proteid (p. 460 *et seq.*), if hemoglobin is present a brownish coagulum will result. This test is not very delicate. In contradistinction to the proteid precipitate, the coagulum tends to float upon the surface of the fluid. Shaking with alcohol and sulphuric acid will bleach the color.

Heller's Blood-test.—To a test tube half-full of urine, 5 drops of potassium or sodium hydroxid are added and the mixture heated. If hemoglobin is present a brownish-red or blood-red flocky precipitate appears. It consists of the phosphates and carbonates of the earthy alkalies, which have carried down with them the hematin that has been formed from the hemoglobin in the reaction. In alkaline urines the above method often produces no precipitate, because the phosphates and carbonates have already completely separated out spontaneously. The necessary quantity of phosphates and carbonates may be supplied by adding to the specimen about the same volume of a normal urine. With this test the coloring matters which appear in the urine after the use of chrysarobin, senna, rheum, or rhamnus may react very much like hemoglobin, and so may lead to confusion. But in the latter case the red color which arises in the fluid portion of the specimen upon the addition of an alkali after cooling, and the decoloration upon the addition of acetic acid, are characteristic (see p. 503).

Teichmann's Hemin Test.—After filtration, the precipitate from the heat or Heller's test or, better still, the precipitate produced by a solution of tannic acid is washed and dried in the air. Any hemoglobin present will be contained in this dried precipitate, which we employ for Teichmann's delicate test. A small bit of the dry material is placed upon a glass slide with a particle of sodium chlorid, a drop of glacial acetic acid is added, a cover-slip is laid upon the mixture, and the whole is heated to the steaming-point for about one minute. A little acetic

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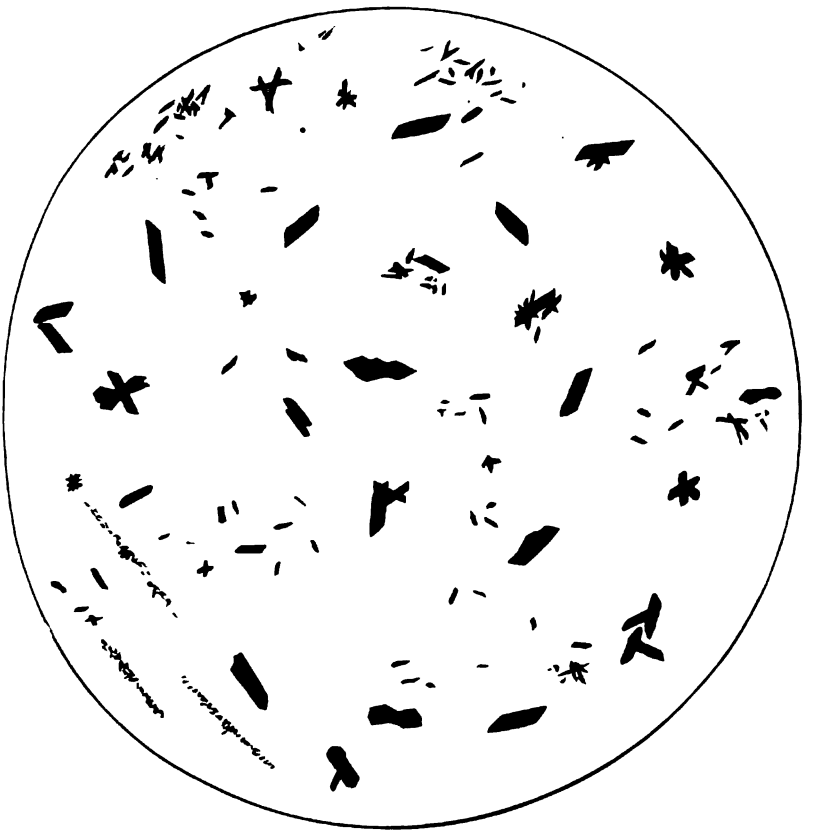
PLATE 5.



Teichmann's Hemin Crystals.

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acid is added from time to time to make up for evaporation. If the fluid turns brown, it is warmed gently and then allowed to evaporate. Teichmann's characteristic hemin crystals (Plate 5) are then evident on microscopic examination. Chemically, they consist of the hydrochlorid of hematin. The test frequently fails because excessive heat is employed and the mixture evaporated too quickly, so that, of course, the characteristic crystals are not formed so readily. Very beautiful crystals are sometimes obtained when the test is performed in the cold.

Schönbein-Almén's Turpentine-Guaiac Test.—This is one of the most delicate of the blood-tests. A layer consisting of a mixture of equal parts of tincture of guaiac[1] and oil of turpentine is carefully stratified upon the top of the urine. If hemoglobin is present a cloudy ring gradually forms at the junction of the two layers, little by little becoming an intense blue. This test will sometimes give a positive result when the spectroscopic test fails. The oil of turpentine employed must be ozonized—*i. e.*, must be old. It may be well to test the reagent first with a well-diluted blood-solution. Before performing the test we should see that the urine is acid (adding acetic acid to an alkaline urine, if necessary).

Pus in the urine may turn the reagent blue, but it is claimed that this blue color appears quite as well without the oil of turpentine (Tappeiner). The same modification which we mentioned upon p. 447 (shaking with ether) for examining the gastric juice may be employed with the urine to avoid the above confusion.

Spectroscopic Detection of Hemoglobin.—In employing the spectroscopic test, it does not make any difference whether the hemoglobin is still contained in the blood-corpuscles or whether it is in solution in the urine. In the urine hemoglobin usually occurs in three modifications—in the form of oxyhemoglobin, reduced hemoglobin, or methemoglobin. The spectra of these substances differ; they are represented in Fig. 167.

These various derivatives of hemoglobin may occur in the urine simultaneously and so produce a mixed spectrum. With a recent and profuse hemorrhage into the urinary passages we find chiefly oxyhemoglobin. With hemoglobinuria and with true nephritic hemorrhages, on the other hand, we find principally methemoglobin. The latter, however, may be altered to reduced hemoglobin, and eventually to oxyhemoglobin by the bacterial decomposition in the urine.

For clinical purposes the spectroscopic test can be performed by looking at a layer of urine 1–2 c.c. thick, by transmitted bright daylight, sunlight, or lamplight, through a small hand spectroscope (see Fig. 166). If the urine is very dark or cloudy, it must first be diluted with water.

DETECTION OF HEMATOPORPHYRIN.

Hematoporphyrin occurs in the urine principally after the protracted administration of sulphonal, trional, and tetronal, but in rare cases it occurs under pathologic conditions about which little is known.[2] Traces occur also in normal urine.

[1] Alcoholic solution of resina guaiac, 1 : 5.

[2] *Cf.* Schulte, Aus der Quinckeschen Klinik, *D. Arch. f. klin. Med.*, 1897, vol. lviii., parts 4 and 5.

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It is considered to be a red pigment derived from hematin and free from iron. Nencki and Sieber consider that it is isomeric with the biliary pigment bilirubin.

It is demonstrated by Salkowski[1] as follows: 30 to 50 c.c. of urine are completely precipitated by an alkaline barium chlorid solution (a mixture of equal parts of a cold saturated solution of barium hydrate and a 10 per cent. solution of barium chlorid). The precipitate is washed with water and then with absolute alcohol, and the hematoporphyrin extracted by treating it with alcohol acidulated with hydrochloric acid.

The extraction is best performed by repeatedly pouring a warm mixture of 10 c.c. of alcohol and 6 to 8 drops of hydrochloric acid upon the precipitate in the filter. The resulting red-violet gives the two bands of acid hematoporphyrin (compare Fig. 167, No. 6, p. 448). Supersaturation with ammonia turns the solution yellowish, and the four bands belonging to hematoporphyrin in alkaline solution appear in the spectroscope.

DETECTION OF BILIARY PIGMENTS.

The most important biliary pigments are bilirubin and biliverdin. The latter is derived from the former by oxidation in the spontaneous decomposition of bile by putrefaction. These two pigments (and more especially bilirubin) always appear in the urine whenever biliary pigment gets into the blood—*e. g.*, in jaundice (p. 42 *et seq.*).

Icteric urine can usually be recognized by its color, which varies from a dark yellowish red or brown to a greenish black. A yellowish foam and yellowish stains of the urine upon clothes are particularly characteristic. The sediment also is usually stained yellow.

The following methods are employed to demonstrate the presence of biliary pigments:

Gmelin's Test.—Filtered urine is allowed to slowly trickle down the side of a test tube and stratify itself upon a layer of nitric acid. If biliary pigment is present, color changes occur at the line of junction of the two fluids—from greenish blue through violet red to yellow. The individual layers of urine pass through this play of colors with varying rapidity according to their distance from the nitric acid, so that we usually see several of the colors superimposed. Sometimes the green ring is the only one we can see plainly, but this is usually enough. A red-violet[2] alone, however, may be produced by skatol or indol coloring-matters (p. 477). Gmelin's reaction depends upon the various steps in the oxidation of bilirubin; hence, the nitric acid should contain some nitrous acid. Crude yellow nitric acid fulfils this qualification, or the pure nitric acid may be employed if it is first heated with some organic substance—*e. g.*, some bits of wood. Both bilirubin and biliverdin react to Gmelin's test; only with biliverdin the reaction is abbreviated, as it were, because the biliverdin is already the partially oxidized green product, to which bilirubin must be changed as the first step in the process of oxidation.

Rosenbach's Modification of Gmelin's Test.—Icteric urine is filtered after having been acidulated with hydrochloric acid, and nitric acid is then dropped

[1] *Zeits. f. phys. Chemie*, vol. xv., 1891. *Cf.* also Hammarsten, *Skand. Arch. f. Physiol.*, 1891, vol. iii.

[2] Compare below with regard to the possibility of confusion with indican.

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upon the paper. The play of colors which surrounds the drops of nitric acid will demonstrate even very slight traces of bile pigment.[1]

If this modified test is doubtful, the bile pigment may be first extracted with chloroform. About 2 c.c. of the latter and 3 drops of hydrochloric acid[2] are added to a test tube almost filled with urine. The two liquids are then intimately mixed, but without violent shaking. The chloroform takes up the bile pigment and becomes yellow; but the solvent will not settle readily if the mixture is shaken vigorously. The chloroform is now separated from the supernatant urine and an equal volume of water and a few drops of sodium hydrate are added, and then mixed by repeatedly inverting the tube. The pigment will then be dissolved by the water, because the salt combination of the pigment is insoluble in chloroform. Gmelin's test can now be employed with this concentrated solution of bile pigment. It will also succeed, though not so well, if tried on the chloroform extract. The least trace of an aqueous solution of potassium iodid or a drop of diluted ferric chlorid solution will gradually turn the chloroform extract green. The bile pigment may also be demonstrated as microscopic crystals by evaporating the chloroform extract in a watch glass (compare Fig. 211, *d*).

Recently, Jolles has described a procedure which he claims increases the delicacy of Gmelin's reaction. It is as follows: Fifty cubic centimeters of urine are shaken in a separatory funnel with 5 c.c. each of a 10 per cent. barium chlorid solution and chloroform. Part of the bile pigment will be extracted by the chloroform and part precipitated by the barium chlorid. The precipitate and the chloroform are separated from the urine and the chloroform evaporated upon a water bath. A little yellow nitric acid is now added to the residue, and Gmelin's characteristic play of colors appears immediately.

In Gmelin's test, nitric acid may, of course, bring out an indican reaction (p. 477). The blue of the indigo mixed with the yellow of the urine pigment will produce a green tint, and so may simulate Gmelin's reaction. But the indigo ring always has a blackish shade and consists of a fine precipitate. In a doubtful case the question may be settled by first isolating the bile pigment by chloroform extraction (see above) and then performing Gmelin's test. This method is also necessary before we can be sure that both indican and bile pigment are present. (See also the following test of Salkowski.)

The presence of proteid in the urine, as a rule, does not interfere with Gmelin's test unless the amount of bile pigment is very small. In such an event it is better to first extract the bile pigment with chloroform. The test cannot be well performed with urine from which the proteid has been removed, because the precipitated albumin will carry a small amount of bile pigment with it.

Salkowski's Test.—The urine is rendered alkaline by the addition of a few drops of a sodium carbonate solution; a solution of calcium chlorid (1 : 10) is then added drop by drop until the solution over the precipitate shows after thorough mixing the normal color of the urine. The precipitate is then filtered, well washed, placed in a test tube with alcohol, and then dissolved by adding hydrochloric acid and shaking. If the clear solution contains bile pigment, boiling will turn it green; if not, the fluid will not change color. The green solution will change to blue, violet, and red. This test is often successful when Gmelin's test gives no reaction, and Salkowski recommends it particularly in those cases where the indican in the urine interferes with Gmelin's test.

Trousseau's Test.—A few drops of tincture of iodin are added to the urine. If bile pigment is present the specimen will turn a beautiful emerald green. If the tincture of iodin is diluted ten times with alcohol, and the dilution is then stratified upon the urine, a green ring will appear at the line of junction of the two fluids. This is more delicate than Gmelin's test.

[1] This test is to be recommended. Caution must be observed, however, that a positive test is not due to the filter paper itself, since impure paper may give a similar reaction.

[2] The object of acidity is to free the bile pigment (the latter reacts chemically as an acid) from the alkaline combination in which it occurs in the urine. In this way the extraction is rendered more complete, because free bile pigment is insoluble in water but readily soluble in chloroform, while the reverse is true of the salt of the bile pigment.

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Hammarsten's Test.[1]—A mixture of 19 parts of 25 per cent. HCl and 1 part of 25 per cent. HNO_3 is allowed to stand at room temperature for from several hours to a day until it has turned slightly yellowish. One part of this acid mixture is added to 5 parts of 95 per cent. alcohol. A few drops of urine are added to a few cubic centimeters of this acidulated alcohol. If the urine contains bile pigment, a characteristic green color appears almost immediately even at room temperature. The author considers the test sensitive, but not more delicate than Salkowski's.

Stockvis' Test for Cholecyanin (*bilicyanin*).—5 to 10 c.c. of a 20 per cent. solution of zinc acetate are added to 20 to 30 c.c. urine. Sodium carbonate solution is added to slightly diminish the marked acidity. The abundant precipitate will contain all the bile pigments; it is washed on a filter and then dissolved in a little ammonia. This transforms the bile pigment to cholecyanin. A neutral solution of the latter is blue green, and exhibits red fluorescence and a characteristic spectrum with three absorption bands. One of these bands is in red, sharply outlined and dark, between C and D, nearer to C; a second, less sharp, in yellow, covering D; and the third, a very faint one, in green, between D and E.

Haycraft claims that in urine which contains bile pigment powdered sulphur will immediately or almost immediately sink to the bottom. This peculiarity is even more characteristic of urine which contains biliary acids. The test is not very accurate, since after a certain time some of the powdered sulphur will sink in normal urine. It is probable that the temperature of the urine has something to do with the result of the experiment, but it has not been determined to what extent.

Microscopic Detection of Bile Pigments.—The urine must contain a sufficient amount of bile pigment. It is acidified with hydrochloric acid and allowed to stand for some time in the cold, after which bilirubin will be precipitated as bundles of microscopic needles colored intensely brown. (Fig. 211, *d*.) These little bunches of needles are often observed when icteric urine is evaporated to test for the presence of leucin or tyrosin (p. 498). The bilirubin needles are, however, much browner than tyrosin needles.

Method for Removing Bile Pigment from the Urine in Order to Perform Other Tests.—The color of bile pigment may be removed very easily either by extracting the acidified urine with chloroform or boiling the specimen with some animal charcoal. This is necessary before testing for salicyluric acid with ferric chlorid, because this reagent, like the tincture of iodin, is apt to produce a green discoloration in icteric urine, which hides the violet color of the iron salicylurate. The urine must not be boiled too long with the charcoal, as the latter is apt to combine with the salicyluric acid. It is always advisable to make a control test, in order to be sure that the process of decolorizing has not destroyed the substance we are trying to detect.

DETECTION OF BILE ACIDS.

The bile acids occur in the urine chiefly in cases of obstructive (retention) jaundice. These acids should be isolated before we attempt to test for them.

Hoppe-Seyler[2] gives the following directions: Lead acetate and a little ammonia are added to the urine. The resulting precipitate is washed with water, then boiled with alcohol, and filtered while hot. The lead salts of the bile acids dissolve in hot alcohol. A few drops of soda solution are added to this solution, which is then evaporated over a water bath till dry, and the residue boiled out with absolute alcohol, when the sodium salts of the bile acids dissolve. The filtered alcoholic extract is evaporated to a small volume, then precipitated,

[1] *Skand. Arch. f. Physiol.*, vol. ix., p 313; ref. in *Centralbl. f. Physiol.*, vol. xiii., p. 644.
[2] *Handbuch der Physiol. u. path.-chemischen Analyse*, 1893, p. 378.

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and allowed to stand with an excess of ether in a closed bottle. In this way the sodium salts of the bile acid scan often be obtained as crystals. But it is not necessary to wait for them, as the resinous precipitate may be dissolved immediately in a little water, and Pettenkofer's test applied as follows:

Pettenkofer's Test.—To the solution of the bile salts we add two-thirds of its volume of concentrated sulphuric acid very slowly, so that the mixture does not become heated above 60° C. Then we add 3 to 5 drops of a solution of 1 part of cane sugar in 4 to 5 parts of water, and shake, whereupon the liquid turns a beautiful violet color.

Strassburg[1] has at times succeeded in demonstrating the presence of bile acids in the urine directly by adding a small amount of cane sugar and then filtering. To the dried filtrate is added a drop of pure concentrated sulphuric acid. If bile acids are contained in the urine a beautiful violet spot appears within a quarter of a minute in the particular place at which the drop was added. This spot soon turns to a dark purplish red, which is particularly plain by transmitted light.

v. Udránsky considers a direct detection sometimes possible. He dilutes 1 drop of urine with 1 c.c. of water and 1 drop of furfurol water (obtained by shaking 1 drop of furfurol thoroughly in one-half test tube of water), and then adds 1 c.c. of concentrated sulphuric acid.

Haycraft claims that his method (see p. 474) for determining the presence of biliary acids is perfectly trustworthy. But, as a matter of fact, in positive cases the test does not discriminate between biliary acids and biliary coloring matter, even when the latter occurs in very slight traces. (See p. 474 in regard to the significance of biliary acids in icteric urine.)

DETECTION OF INDICAN AND INDIGO.

Indican, or indoxyl sulphuric acid, in the urine is the product of the putrefaction of proteids, and is a derivative of indoxyl, which in turn is an oxidation product of indol. This decomposition occurs in the intestine normally, but is more pronounced in digestive disturbances with diminished peristalsis (intestinal obstruction). Indican may also be found under some circumstances in various other parts of the body, from suppurative processes.

An increased amount of indican in the urine is of some diagnostic importance in helping to locate the seat of an intestinal obstruction. Experience has shown that an *obstruction in the small intestine* is quickly followed by a marked increase in the amount of indican in the urine, as contrasted with an *obstruction in the large bowel*, where there is very rarely any such increase, unless perhaps in the later stages. This is probably because the trypsin of the pancreatic juice favors decomposition and the formation of indican. Both factors aid each other in the decomposition of the proteids. In deep-seated obstruction in the large intestine the stagnation of the contents, which favors decomposition, occurs primarily where trypsin is no longer active. (The latter, as is well known, is destroyed or reabsorbed in the course of the intestine.) In obstruction in the small intestine the stagnation takes place where the trypsin favors the decomposition. A further explanation is

[1] *Arch. f. d. ges. Physiol.*, vol. iv., p. 461.

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that the greater portion of the proteids which furnish the indican have been already absorbed in the large intestine.

If the duct of the pancreas is occluded, the amount of indican in the urine will be diminished. However, as the indican in the urine is normally quite small in amount, or even absent altogether, a diminution can be clinically significant of an occlusion of the pancreatic duct only under conditions which would ordinarily favor the production of a large amount of indican—*e. g.*, jaundice and a meat diet.

In peritoneal affections, particularly in perityphlitis, any increase in the amount of indican suggests an increase of trouble; any diminution, an improvement in the condition.

We can frequently demonstrate indican in normal urine by one of the following methods. They depend upon the fact that oxidizing agents transform indican into indigo. The reaction must be very striking to enable any safe diagnostic conclusions (as to increased intestinal decomposition or any other process of decomposition in the interior of the body, or as to the location of an obstruction).

Jaffe's Indican Test.—One-quarter of a test tube of urine is mixed with an equal volume of concentrated hydrochloric acid, and a drop of a half-saturated solution of commercial chlorid of lime (chlorinated lime) is added. If no reaction takes place immediately, more of the chlorid of lime solution is added, drop by drop, without shaking. If the urine contains considerable quantities of indican, a blue-black ring of precipitated indigo will appear in the upper part of the tube, at the lower zone of action of the chlorid of lime solution. This ring will become more intensely colored upon standing. If a very large amount of indican is present, the entire fluid will turn black. We must be very careful not to add too much of the chlorid of lime solution, since in that case the *indigo* formed by the oxidation of the *indican* will become further oxidized to yellow *isatin*. Proteid, if present, should be first removed by boiling and filtration. The indigo formed in this way will dissolve in a few cubic centimeters of chloroform by gentle agitation, coloring the latter blue. If only a small amount of indican is present, there is considerable danger of adding too much chlorid of lime. Hence, the test has been modified at the Bern Clinic in the following way: First a few cubic centimeters of hydrochloric acid are poured into a test tube and a drop of the chlorid of lime solution is added. The mixture is well shaken, and then the urine is carefully stratified upon it, either by letting the urine flow down the side of the tube or, better still, by letting it fall upon the surface of the hydrochloric acid, drop by drop, through a filter. The indican reaction will then be shown very beautifully at the line of junction of the two fluids.

Obermayer's Test.—Obermayer[1] claims that he can avoid the danger of excessive oxidation, which is a defect in Jaffe's test, by the use of ferric chlorid as an oxidizing agent instead of chlorid of lime. He adds to the urine a small amount of a 20 per cent. solution of sugar of lead, in order to precipitate those substances which prevent the shaking out of the indigo by the chloroform. After-

[1] *Wien. klin. Woch.*, 1890, No. 9.

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removing the precipitate by filtration, he adds an equal volume of fuming concentrated hydrochloric acid which contains 4 gm. of ferric chlorid to the liter. The mixture is then shaken. The reaction will take place, he claims, within a few moments, and the indigo may then be extracted with chloroform. Obermayer believes that this process is adapted for an accurate colorimetric determination of the amount of indican present.

Amann's Test.—J. Amann[1] recommends sodium pyrosulphate ($Na_2S_2O_7$) as an oxydizing reagent. He claims that this reagent also will not cause any loss of indigo by excessive oxidation. The test is performed as follows: To 20 c.c. of urine are added a few drops of pure sulphuric acid, 5 c.c. of chlorform, and then 5 c.c. of a 10 per cent. solution of sodium pyrosulphate. They are mixed gently for several minutes (too much shaking may produce an emulsion). The chloroform is then allowed to settle and will be colored blue by the indigo. The method can be utilized for an exact colorimetric determination.

Other oxidizing reagents often produce a more or less distinct indican reaction—*e. g.*, nitric acid in the test for bile pigments. (See p. 473). In the employment of nitric acid for the detection of indican there is great danger of decolorizing the indigo. It will be recalled that the decolorization of indigo is one of the reactions of nitric acid, which is therefore not a reliable agent for the detection of indican.

As already mentioned, *indican* is sometimes oxidized spontaneously to indigo in the urine before or after being voided. Urine which contains indigo presents a black, green, or bluish tint. All that is necessary to prove that the color is due to indican is to shake the urine with chloroform and then allow the latter to settle, when it will be seen to have turned blue. The needle-shaped or thin plate-like crystals of indigo may sometimes be detected microscopically in the urine sediment, or even in the residue of the dried chloroform extract.

DETECTION OF MELANIN (PHYMATORRHUSIN) AND MELANOGEN.

A peculiar brownish-black coloring matter, the so-called melanin (phymatorrhusin), sometimes appears, partly as such, partly as a colorless chromogen, in the urine of patients suffering with melanotic neoplasms. Urine which contains melanin is blackish. Oxidizing agents, such as nitric acid or ferric chlorid, will turn it still darker when exposed to the air. Urine which contains colorless melanogen exhibits a dark color only after the melanogen has been changed by these agents into melanin. The addition of bromin water, and sulphuric acid or chlorin water also produces a dark color. An excess of bromin or chlorin water will cause a decolorization and throw down a dense dirty-yellow precipitate. Boiling with fuming nitric acid will, under certain circumstances, decolorize melanotic urine. Melanin may be confounded with indigo, and melanogen with indican (see p. 475 *et seq.*). The solubility of indigo in chloroform, and the resulting blue color, will serve to differentiate them.

ROSENBACH'S REACTION (Red Indol and Skatol Pigments).

Several years ago Rosenbach described the following reaction. Concentrated nitric acid is added drop by drop to a test tube of urine while it is being continually boiled. The urine gradually assumes a Burgundy red color, while the foam produced by shaking turns a bluish red. A reddish or brownish-red color without the foam becoming violet red is not distinctive, as it may be due to the urobilin in the urine. Contin-

[1] *Revue. méd. de la Suisse Rom.*, 1897, No. 6, p. 449.

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ued addition of nitric acid will turn the red color rather rapidly to a yellowish red or yellow with a yellow foam. The addition of a solution of soda or ammonia drop by drop will cause a bluish-red precipitate, which dissolves in an excess with the formation of a reddish-brown color. In these cases the urine is sometimes already of a reddish shade, or sometimes a slight reddish tinge occurs upon the addition of nitric acid while the specimen is still cold. The reaction seems to depend principally—although perhaps not exclusively—upon the formation of indigo-red (indirubin, indigo-purpurin), an oxidation product of indoxyl or indican (see p. 475). It may, too, perhaps depend upon the formation of skatol pigments by means of the oxidizing influence of nitric acid. Indigo is often produced in this way—hence the violet shades of the reaction. The test has a diagnostic importance similar to that of the indican reaction.

UROROSEIN (Urrhodin).

A red color is not infrequently noticed in the urine, appearing soon after the addition of mineral acids, especially hydrochloric acid. It occurs both in health and in many conditions of disease. Nencki and Sieber recognized it as due to a pigment which they called *urorosein*, the chromogen of which is contained in the urine. It is probably identical with the pigment described by Heller as *urrhodin*, evidently differs from indigo-red, and may possibly be a derivative of skatol. In contrast to Rosenbach's reaction (indigo-red, p. 477), the red color disappears upon the addition of alkaline carbonates to the urine. This pigment, unlike indigo-red, is insoluble in chloroform and ether.

UROERYTHRIN.

This pigment occurs in normal urine, particularly after the ingestion of large quantities of food and alcohol, and after profuse perspiration; it is also found in pathologic urines in digestive disturbances, in gout, and in febrile conditions. This is the pigment which is responsible for the beautiful rose tint in many of the uratic sediments. It was formerly regarded as identical with urorosein, but is now known to be a different substance. Urines containing large quantities of this pigment are of a decided orange color. When concentrated sulphuric acid is added to the pigment, a carmin-red color is produced; alkalies (not ammonia) change the color of the pigment first to a purplish blue and then to a green. These reactions obtain only with pure solutions of the pigment, however, and such solutions are difficult to obtain.

DETECTION AND OCCURRENCE OF UROBILIN.

Urobilin is in all probability a derivative of bilirubin. Modern investigations have, however, cast doubt upon the old assumption that it was identical with hydrobilirubin. Urobilin occurs in small amounts even in normal urine, but, according to Saillet, not until the urobilinogen has been acted upon by light. The normal yellow color of the urine is not due to urobilin, but to urochrome.

Pathologically, urobilin is excreted in increased amounts in some forms of icterus (urobilin icterus, see p. 44), and in all diseases associated with destruction of red blood-corpuscles, such as fever, scurvy, internal hemorrhage, and pernicious anemia.

The demonstration of such an increase is sometimes of diagnostic importance in proving the occurrence of internal hemorrhage—*e. g.*, cerebral hemorrhage, hemorrhagic infarction, retro-uterine hematocele,

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and hemorrhage in ectopic gestation. The blood-pigment of the transuded blood is first transformed into bilirubin or hematoidin, and is then excreted as urobilin. But if fever complicates any of the above conditions, an increase in the excretion of urobilin will be of no diagnostic importance, for fever alone is a frequent source of urobilinuria. Unfortunately, the amount of urobilin in the urine is of slight value in the diagnosis of brain hemorrhage, however desirable it would be to possess a reliable criterion to differentiate cerebral softening from hemorrhage. On the one hand, most cerebral hemorrhages are much too slight to produce any marked increase in the amount of urobilin in the urine. Large cerebral hemorrhages, on the other hand, usually result fatally before the necessary changes of the blood-pigment have taken place. Even in favorable cases an excess of urobilin does not appear in the urine for some time after the hemorrhage, when the differential diagnosis between hemorrhage and softening is of no particular therapeutic value. Further, fever so frequently occurs in the acute focal lesions of the brain as to decidedly influence the amount of urobilin in the urine.

Urine which contains a very large amount of urobilin is often very dark. This is not invariably the case, because urobilin does not possess very intense coloring properties. Usually, other pigments which may be increased in quantity along with it are principally responsible for the dark color. In fact, a very dark urine may contain very little, and a light urine a large amount of, urobilin.

The most accurate method for the demonstration of the presence of urobilin is by means of the spectroscope, after first acidifying the urine with a little hydrochloric acid to make the spectrum more distinct. Acid urobilin solutions when very concentrated or in thick layers absorb the entire blue end of the spectrum as far as the middle of green. On the other hand, a thin layer or a less concentrated solution shows an absorption band between green and blue (Fig. 167). In contrast to urobilin, the biliary pigments proper absorb the spectrum diffusely. A simple chemical method for demonstrating the presence of urobilin is as follows: The urine is rendered strongly alkaline with ammonia, filtered, and a few drops of a 10 per cent. alcoholic or aqueous solution of zinc chlorid are added. A beautiful green fluorescence occurs if urobilin is present.

To demonstrate chemically a very small quantity of urobilin in the urine, it must be first extracted by gently shaking a specimen of urine acidulated with a few drops of hydrochloric acid with one-third its volume of amyl alcohol. When the alcohol has extracted the urobilin, it will be colored brown. If the layers are not distinct and the amyl alcohol portion remains cloudy, like an emulsion, the separation and the clarifying may be aided by the addition of a few drops of ethyl alcohol. Several drops of an alcoholic ammonium chlorid solution (spiritus Dzondii) and some 1 per cent. alcoholic solution of zinc chlorid are then added to the amyl alcohol layer, and the resulting fluorescence shows the presence of urobilin.

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Whether the urobilin as characterized by the above-mentioned tests is a specific substance, or whether there exist several urobilins, has been recently much discussed.[1] A final verdict is not yet possible. In any case a differentiation (suggested by Jolles) of physiologic and pathologic urobilins has not been sufficiently worked out to be of any clinical value.

QUALITATIVE DEMONSTRATION OF GRAPE SUGAR (GLUCOSE; DEXTROSE).

Preparation of the Urine for the Qualitative Test for Sugar.

If the urine contains proteid, this must be removed before proceeding with the tests for sugar (see p. 463). If it contains hydrogen sulphid (hydrothionuria, see p. 455), this compound may be removed by shaking the urine with white lead, and filtering.

Almost all of the qualitative tests for sugar are most successful when the acid urine is precipitated with lead acetate. About 5 c.c. of a solution of lead acetate are added to 50 c.c. of urine; the mixture is filtered, and the excess of lead removed from the filtrate by the addition of several cubic centimeters of a saturated solution of sodium phosphate, this solution being added until precipitation no longer occurs. The decolorized filtrate is then employed for the tests, and is better adapted for this purpose than the original urine, since various substances which might have interfered with the reactions have been removed. The original urine must be distinctly acid, and also remain acid after the addition of the sodium phosphate, since sugar is precipitated from an alkaline urine by the addition of lead. The acid reaction should be finally insured by the addition of several drops of acetic acid. All concentrated urines should be diluted with two or three volumes of water, in order to minimize the effect of certain substances which interfere with the reactions.

An appropriate preparation of the urine for the qualitative sugar tests is also the treatment with mercuric nitrate, as recommended upon p. 516 for the polarimetric examination.

Very small amounts of dextrose probably occur in every normal urine. We have no simple reaction to prove this absolutely, but we do know that normal urine reduces less after than before fermentation by yeast. Pathologic amounts of sugar can, on the contrary, be demonstrated by very simple methods. We must, in the first place, discriminate between a permanent or persistent dextrose excretion (*diabetes mellitus*) and one of the transient type (*glycosuria*). No sharply defined distinction can as yet be drawn between these two. *Glycosuria* accompanies various disorders, particularly cerebral and digestive affections, certain forms of poisoning (morphin, carbon monoxid, chloral hydrate, oil of turpentine (in the two latter cases the presence of glycuronic acid is responsible for the condition), corrosive sublimate, amyl nitrate, nitrobenzol, curare, phloridzin, etc.), and prolonged hunger. It sometimes happens that otherwise perfectly healthy people temporarily excrete dextrose in the urine after too abundant ingestion of sugar or of other carbohydrates (*alimentary* or *physiologic glycosuria*).

[1] *Cf.* Jolles, *Centralbl. f. inn. Med.*, 1895, and *Pflüger's Arch.*, vol. lxi., pp. 623–637; and Archibald S. Garrod and F. Gowland Hopkins, *Jour. of Physiol.*, vol. xx., pp. 112–114.

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It is, however, quite essential to distinguish cases of *alimentary glycosuria* due merely to excessive ingestion of sugar from those attributable to undue consumption of starches. The latter presumably indicate some grave disorder, because the tolerance for starches is much greater than for sugar, probably for the reason that the absorption of starch takes place much more slowly. Hence, when abundant starch ingestion gives rise to sugar excretion, we may best consider the case as related to diabetes mellitus, and reserve the term "*physiologic* or *alimentary glycosuria*" for cases where the starches can be freely ingested without exciting sugar excretion, but where excessive ingestion of sugar occasions a glycosuria. As a matter of fact, excessive ingestion of sugar may overstep the tolerance of perfectly healthy individuals.

Urine containing much dextrose is peculiar in presenting a light or pale color combined with a high specific gravity. In *true diabetes* the amount of urine is usually, although not always, increased. Hence a pale urine, excreted in excess of the normal daily quantity, and with a specific gravity of 1030 or more, allows of the most decided suspicion of diabetes mellitus. Urine containing dextrose ferments spontaneously after standing for some time. This characteristic alcoholic fermentation may be recognized either from the development of carbonic acid bubbles or from the microscopic demonstration of yeast in the sediment. Therefore sugar tests must always be performed with a fresh specimen.

To detect a small quantity of dextrose in a doubtful case we naturally select for examination a specimen voided during the day and a short time after eating, because the quantity of sugar is apt to be then at a maximum. If the meal was rich in starch or sugar we are still more liable to obtain a positive result. Of course, we must remember that apparently perfectly healthy people may excrete sugar after too abundant consumption of these two foods. If an excessive ingestion of starch alone produces glycosuria in such people they may be considered as straddling the border line between health and diabetes mellitus.

If it be desired to preserve the urine for some time before making the qualitative test for sugar, it has been recommended to add 1 per cent. of cupric sulphate, in order to prevent fermentation. Trommer's test may then be employed.

Dextrose may be detected in the urine by the following chemical tests:

Moore-Heller's Test.

A few cubic centimeters of potassium or sodium hydrate are added to about three times this volume of urine in a test tube and then boiled. If considerable sugar is present the mixture will turn a dark brown as a result of the oxidation of the dextrose. The chemical reaction upon which this test is based has not yet been definitely ascertained. The reaction is absolutely characteristic of sugar only when the color becomes dark brown or, at least, yellowish brown, or with a diluted urine an intense clear yellow. The test is rather delicate, a characteristic reaction being obtained even with a considerably diluted diabetic urine. Unless the result is absolutely certain, it is a good plan to compare the reaction with that obtained from a normal urine, and in doubtful cases even with that from a diluted urine. If the mixture of

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diabetic urine and potassium hydrate is allowed to cool, and is then cautiously acidulated with sulphuric acid, an odor of burnt sugar should be developed.

Reduction Tests.

Trommer's, or the Copper Test.—One-third the volume of potassium or sodium hydrate is added to the urine in a test tube, and then drop by drop a solution of cupric sulphate (1 : 10), the mixture being shaken constantly until a slight excess of the precipitated cupric hydroxid remains undissolved. If sugar is present we can add a good deal more of the cupric sulphate solution without producing a precipitate, and the mixture will turn a beautiful blue color. This depends upon the fact that in the presence of dextrose the formation of an easily soluble double combination takes place, which holds much more cupric hydroxid in solution. The blue color does not, however, positively prove the presence of sugar, because it also occurs if the urine contains glycerin or tartrates, or if ammoniacal decomposition has set in. Urine which contains proteid also holds some cupric hydroxid in solution, but here a violet color is produced. If we heat this dark-blue solution just to boiling in the presence of sugar, cuprous hydroxid ($Cu_2(OH)_2$) and cuprous oxid (Cu_2O) separate in greenish-yellow clouds, which gradually turn brick red and finally diffuse throughout the fluid. This is due to the fact that dextrose has the power in alkaline solution to reduce cupric hydroxid to cuprous hydroxid and cuprous oxid. These separate out as a yellow to brick-red precipitate[1] and the liquid becomes decolorized. If a large amount of sugar is present, metallic copper (Cu) may even separate out upon the side of the test tube in the form of a brownish-red coating.

A *positive* and *typical* copper reaction certainly permits no doubt in regard to the presence of sugar in the urine, for no other substances occur in the urine which can furnish in every detail a corresponding reaction. At most, in some individual instances, it is only necessary to distinguish whether it is dextrose or some other reducing carbohydrate that is present, and, from the much greater frequency of dextrose, it is generally safe to attribute a positive copper test to it. In regard to the differentiation between the individual carbohydrates and the recognition of the copper-reducing combined glycuronates which appear more or less abundantly after the ingestion of certain drugs (camphor, morphin, phenol, derivatives such as paracresol, salicyl, salol, thallin, chrysophanic acid, saccharin, santonin) and sometimes, under both physiologic and pathologic conditions, abundant skatol and indol formation, compare p. 477 *et seq.*

If the copper reduction is less typical, a reasonable doubt may arise as to whether the urine contains dextrose or not, because normal urine, in virtue of the presence of uric acid, kreatin, kreatinin, and combined glycuronic acid, does reduce copper to a certain extent. In order to

[1] Cuprous hydroxid is yellow; the oxid is brick red. The more alkaline the solution, the more pronounced the brick-red color, the oxid taking the place of the yellow cuprous hydroxid.

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differentiate a reaction due to dextrose from a similar one occasioned by one of the above constituents of the urine, the following distinction should be noted: If the reduction is due to dextrose, the blue solution must not only be decidedly decolorized, but there must also be formed a distinct granular precipitate of cuprous oxid and cuprous hydroxid. On the contrary, the reduction by normal urine is never associated with the immediate formation of a yellow or brick-red precipitate, but there is produced merely a dirty-yellowish fluid, because kreatinin, ammonium salts, and other substances keep in solution the small amount of cuprous oxid which is formed. It does often happen, however, that after allowing the test to stand for some time in the cold, normal urine will bring down a yellowish-red precipitate. Such a late separation is not suggestive of dextrose. The above distinction in regard to the rapidity of reduction is in reality a quantitative test. Urine containing dextrose keeps a much larger amount of cuprous hydroxid in solution, but is also able to still further reduce this soluble excess to the red suboxid, which cannot remain in solution. It is evident that the above is the real difference between urine containing sugar and one free from it, since, if the urine contains less than 0.2 per cent. of sugar, the reduction occurs just as in normal urine without precipitation, for the moderate amount of cuprous oxid will remain in solution. Heating urine which contains such slight amounts of sugar simply turns it yellow, but without any turbidity. Yet even in these cases we must acknowledge (and this is of some diagnostic importance) that the yellow color is very much more intense, clearer, and in a measure more translucent than with normal urine, very likely because the fluid contains a considerable amount of suboxid, despite the fact that it remains clear. In such instances where Trommer's test produces a clear-yellow color, a granular precipitate of the red suboxid usually appears a short time after cooling. By paying attention to these facts, an expert can frequently make use of such atypical reactions in recognizing a doubtful case of diabetes mellitus which has been influenced by the diet. Moreover, the reducing action of the normal urinary constituents may be prevented if we perform Trommer's test at a temperature of 60° to 70° C. This is a more certain method in doubtful cases, because at this temperature the normal constituents do not reduce to any noticeable degree. This temperature is easily obtained by first boiling the urine, then adding one-third its volume of cold potassium hydrate, and then the cupric sulphate solution.

To make Trommer's test as delicate as possible, the first essential is to have as much cupric oxid as possible in solution, so that the abundantly reduced cuprous oxid may be precipitated. This end is accomplished, as already mentioned, by continuing to add cupric sulphate until a small amount remains undissolved. On the other hand, too much should not be added, because an excess of unreduced cupric hydroxid, when heated with potassium hydroxid, will become blackened from the formation of cupric oxid, and so very likely hide the reaction.

The delicacy of the test may sometimes be further increased by diluting the urine containing sugar two to five times. This dilution will diminish the power of the urine to dissolve the suboxid and will cause a granular precipitate to form

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where the undiluted urine gave no reaction. If the urine is free from dextrose, this dilution will not produce any change, because the slight trace of cuprous oxid which is formed will remain in solution in spite of the dilution.

Another method of improving the delicacy of Trommer's test is to shake the urine first with finely powdered animal charcoal, and finally filter. The animal charcoal evidently retains certain substances which dissolve the suboxid and prevents its precipitation.

Seegen's modification of Trommer's test can be recommended for the detection of very small quantities of sugar. It depends upon the fact that animal charcoal will absorb dextrose while in solution, and that the latter can be washed out of the charcoal again. Seegen makes a thin paste with purified animal charcoal and the urine to be examined. A few minutes later this paste is poured upon a filter. After the urine has filtered through he washes the charcoal remaining upon the filter paper with as much water as he employed urine, and then repeats the process. The filtrate of each washing is kept separate, and Trommer's test is performed with each of these filtrates. The substances which cause the solution of the cuprous oxid will be retained in the carbon much longer than will the dextrose. Frequently the test succeeds best with the second or third wash water, as the first may contain too many of the substances which dissolve the suboxid. This method is very much more sensitive for urine which contains only a slight amount of sugar, than Trommer's test upon the urine itself. Seegen claims that a positive reaction is absolute proof of the presence of sugar, because the second or third wash water from a normal urine will no longer reduce.

The copper test may also be performed with the so-called *Fehling's solution*, such as is primarily used for quantitative determinations (see p. 507). This should be freshly prepared each time before use; it consists of equal parts of solutions I. and II. The test should be performed as follows: About 5 c.c. of urine are first boiled in a test tube, and allowed to cool for about twenty seconds; then about 1 c.c. of Fehling's solution is added. If sugar is present the reaction appears immediately. The reason we allow the specimen to cool off to about 60° or 70° C. is because at that temperature normal urine will no longer reduce. If an insufficient amount of copper is added, the cuprous hydroxid which is formed will not become precipitated, but will remain dissolved in a yellow solution. In such an event the test must be repeated with an increased quantity of Fehling's solution. Fehling's solution possesses no particular advantage for the qualitative test; on the contrary, the selection of exactly the amount of copper solution to procure a maximum precipitation of cuprous oxid is much more difficult with this than it is with Trommer's test, where the least excess of copper is easily recognized by the appearance of undissolved cupric hydroxid.

Proteid in the urine does not interfere with the reduction of the copper, but it does hinder the precipitation of the red suboxid. For this reason the proteid should be removed from the urine before performing the test (see p. 643). It is equally important to perform all copper tests with fresh urine, because the ammonium carbonate and the free ammonia formed in alkaline fermentation will also prevent the precipitation of red cuprous oxid.

Provided they are properly performed and the above precautions are observed, the copper tests are still the most practical and safest methods for the qualitative detection of sugar. Only a large amount of the other carbohydrates or of the salts of glycuronic acid can lead to confusion. (See p. 490 *et seq.* in regard to the appearance of the latter, and its differentiation.) It should be remarked that after successful treatment of diabetes has caused the dextrose to disappear from the urine, we often find large quantities of combined glycuronates present.

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The latter, however, can be easily distinguished from dextrose by the very tardy appearance of the copper reduction. In doubtful cases the tests yet to be described may serve as controls, particularly Almén-Nylander's, the fermentation test, and Rubner's test.

(*b*) **Almén-Nylander's Test (Modified Böttger's Test).**—The original Böttger test depended upon the reduction of bismuth subnitrate, $NO_3Bi(OH_2)$, by the dextrose in alkaline solution. But the test was not reliable until modified by Almén-Nylander. The reagent employed is prepared as follows: Four gram of Rochelle salt (potassium sodium tartrate, tartarus natronatus) are dissolved in 100 c.c. of 10 per cent. sodium hydroxid (specific gravity at 19° C. = 1.115) with gentle heat, and then as much bismuth subnitrate is added as will dissolve (about 2 gm.). After cooling we filter off with glass-wool any undissolved bismuth subnitrate which remains. If kept in a dark bottle, the reagent can be preserved for years. To the urine is added about one-tenth its volume of the reagent, and the mixture boiled for a few minutes. In order to prevent the urine from boiling over and burning the fingers, a small coil of platinum wire may be dropped into the fluid, or the test tube may be held to one side of the flame, so that violent ebullition shall not occur. If dextrose is present the fluid darkens and a black precipitate of suboxid of bismuth separates. If the solution turns dark only upon cooling, the test is not positive. A white precipitate of phosphate will be all that is formed upon boiling with urine free from sugar. If a very small trace of sugar is present, the precipitate of the phosphates may be seen to be grayish after it has settled. Nylander's test is delicate enough to indicate ¼ per cent. of sugar.

Before attempting Nylander's test we must first remove any proteid from the urine, because an albuminous urine will produce a precipitate of sulphid of bismuth. With small quantities of proteid the latter precipitate may be easily differentiated from the oxid of bismuth by its reddish-brown color. But if the proteid contents of the urine are large, a brownish-black precipitate will appear, which can very readily be confounded with the dextrose reaction. If the urine is ammoniacal, the appearance of the reaction may be interfered with.

With the above-mentioned reservations, Nylander's is one of the most reliable of the sugar tests. It is always negative with normal urine, and so is particularly well adapted to decide a case where Trommer's test is doubtful. Considerable quantities of combined glycuronic acids or pentose will also plainly reduce Nylander's solution, and in doubtful cases these two substances must be excluded (according to p. 492) before we can assume that the urine contains sugar. The reduction of Nylander's reagent, which occurs after the ingestion of senna, of rhubarb, of eucalyptol, of kairin, of quinin, and of oil of turpentine, probably depends upon the presence of combined glycuronates.

Manifold Significance of the Reduction Tests.

The following table arranged by G. Bruckner[1] indicates the number of substances occurring in the urine which may effect reduction in the tests of Nylander and Trommer, and the necessity of employing other methods of examination before concluding that such a reduction is positive evidence of the presence of dextrose:

I. Normal urinary constituents which, according to their presence in larger or smaller amounts, may produce a varying degree of reduction or change of color of the earthy phosphates (in Nylander's test).

[1] *Aerztliche Rundschau*, 1899, Nos. 42-44.

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Trommer's Test.	*Almén-Nylander's Test.*
Traces of carbohydrates as dextrose, isomaltose, pentoses, animal gum, glycuronic acid compounds.	Uro-erythrin.
Pyrocatechin.	Urinary pigments (urobilin in particular) when present in greatly increased amounts cause a brownish discoloration of the phosphatic deposit.
Bile pigment.	Indican.
Urinary pigment.	
Uric acid.	
Indican.	
Kreatinin.	
Urobilin.	
Urobilinogen.	

Concentrated urines are particularly apt to effect reduction, and the same is true of urines containing a moderate or large quantity of formed elements (leukocytes, erythrocytes, epithelial cells).

II. Products of abnormal metabolism which effect reduction:

Trommer's Test.	*Almén-Nylander's Test.*
Homogentisic acid.	Hexoses (dextrose, levulose, isomaltose, lactose [in puerperal women]), pentoses, glycogen, increased quantities of glycuronic acid compounds.
Uroleucinic acid.	Blood-pigment.
	Increased quantities of hematoporphyrin.
	Uroleucinic acid (weak).
	Homogentisic acid (weak).

III. Reducing substances added to the urine as preservatives, which consequently make the reduction tests for sugar impossible:

Trommer's Test.	*Almén-Nylander's Test.*
Formaldehyd.	
Chloroform.	

IV. Drugs or the derivatives obtained from them as the result of metabolic change:

Trommer's Test.	*Almén-Nylander's Test.*
Acetphenetidin.	Antipyrin.
Antifebrin.	Arbutin.
Arbutin.	Benzoic acid.
Benzoic acid.	Benzosol.
Benzosol.	Quinin (large doses).
Copaiba balsam.	Chloral.
Chloral.	Eucalyptol.
Glycuronic acid compounds of drugs (see p. 492).	Glycuronic acid compounds of drugs (see p. 492).
Morphin.	Indican.
Phenacetin.	Kairin.
Saccharin.	Rheum (also frangula and cascara sagrada).
Salicylic acid.	Salol.
Salol.	Senna.
Sulfonal.	Sulfonal.
Turpentine.	Turpentine.
Thallin.	Trional.
Urethan.	

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Phenylhydrazin Test (Fischer-v. Jaksch).

Two drops of a concentrated solution of lead acetate are added to about 10 c.c. of urine, and the precipitate filtered off; the filtrate is acidified with a drop of acetic acid; phenylhydrazin hydrochlorid the size of a pea, and a piece of sodium acetate the size of a bean, are added. The mixture is heated for about a half-hour over a water bath and then allowed to cool.

If dextrose is present, a yellow precipitate separates out upon cooling. This consists of characteristic microscopic aggregates of needle-like crystals of phenylglucosazone (Fig. 169). If the precipitate is not crystalline or of a yellow color, or if the crystals are not of the form represented in the accompanying plate, we must not consider that the test is positive. The test is very delicate and even sufficient to indicate the presence of 0.01 per cent. of sugar in the urine. As a matter of fact, it is almost too delicate for clinical purposes, as under certain conditions the normal amount of sugar in a urine may give a positive reaction. Besides, there is a source of error in the test, since certain other substances that are normally found in urine may produce a similar reaction. By the determination of the melting-point of the osazone which is formed, a definite identification may be obtained, but this is too difficult for ordinary clinical methods. This is also the best method of distinguishing the osazones of other carbohydrates which, though rarely, may be found in this test from the osazones of dextrose. Before performing the phenylhydrazin test, it is advisable to first remove the proteid from the urine.

FIG. 169.—Crystals of phenylglucosazone (phenylhydrazin test for grape sugar) (after v. Jaksch).

The phenylhydrazin test has been simplified by *Cipollina*[1] under Salkowski's direction. Five drops of pure phenylhydrazin (the base), ½ c.c. of glacial acetic acid, and 4 c.c. of urine are mixed together in a test tube. The mixture is heated over a small flame for about a minute, being carefully and constantly shaken so as to prevent bumping. Four or 5 drops of sodium hydroxid of a specific gravity of about 1160 are next added, but the fluid must still remain acid; this mixture is heated a moment more and then cooled. With this method of procedure, especially in the case of a urine of low specific gravity, the characteristic roset crystals appear immediately or, at least, within twenty minutes. In doubtful cases it is necessary to allow the test that amount of time. Thorn-apple crystals are not at all characteristic, but yellow balls which are gradually transformed into sheaths of needles.

[1] *Deutsch. med. Woch.*, 1901, No. 21, p. 334. The other modifications of the test (Neumann, Kowarski) are also mentioned and criticized here.

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Rubner's Test.[1]

Ten cubic centimeters of a concentrated solution of neutral lead acetate (1 part of lead acetate to 10 parts of distilled water) are added to 10 c.c. of urine. The mixture is filtered, and ammonia carefully added to the filtrate drop by drop until a cheesy precipitate remains. The mixture is then heated over a water bath to 80° C. During the heating, if dextrose is present the precipitate will become a beautiful rose or salmon red. The chemistry of this reaction is not positively known. The test is reliable, very delicate, and therefore particularly appropriate when Trommer's test seems doubtful. If the precipitate is heated too much the color becomes brown, like café au lait, and is no longer characteristic. Milk sugar gives a yellow-red to brown color. The writer does not know how the pentoses and glycuronates react to this test.

Fermentation Test.

Several of the above tests for dextrose are sometimes uncertain unless the reaction is very characteristic, and often even when the result is positive.

Trommer's and Nylander's tests merely indicate the presence of a reducing substance, and do not prove that this substance is really dextrose. If the reduction tests result positively again and again with the same patient, and if, furthermore, the patient presents clinical appearances of diabetes mellitus (specific gravity, quantity of urine, and general appearance), the probability of dextrose being present in the urine becomes for all practical purposes a certainty. But where the reducing substance appears temporarily, and when there are no further data for diagnosing diabetes mellitus, we need a more definite proof that the reducing substance in question is some carbohydrate. Either Rubner's test, the phenylhydrazin test, or the fermentation test will answer this purpose. The last mentioned is one of the surest and most convincing of all sugar tests, and should be resorted to in all doubtful cases. It depends upon the fact that the addition of yeast to any urine which contains fermentable sugar produces a decomposition or fermentation—*i. e.*, the carbohydrates are transformed into alcohol and carbon dioxid gas. Proof of the fermentation is obtained by demonstrating the presence of the carbon dioxid. (Concerning the conclusions as to the type of carbohydrate, see p. 490 *et seq.*)

The fermentation test is performed as follows: A test tube is filled to the brim with the urine, a piece of ordinary compressed yeast the size of a pea is dropped in, and the tube is then gently shaken until the yeast is finally divided, without allowing any air to enter. The tube is closed with a perforated cork which carries a V-shaped piece of glass tubing (Fig. 170, *b*). The test tube is then inverted, placed in a beaker, and left in some moderately warm place (best 25° to 30°C.). If the urine contains dextrose, carbonic dioxid will be formed from it by fermentation inside of a few hours. The gas will accumulate at the upper end of the tube by expelling the urine through the V-shaped tube. Schrötter's gas tubes or fermentation tubes[2] are still more convenient (Fig. 170, *a*).

[1] *Zeits. f. Biol.*, vol. xx., p. 397.

[2] Einhorn's tubes are very serviceable; in this connection see p. 515.

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It is a good plan to prepare two other similar tubes, the one filled with normal urine and yeast, and the other with normal urine, yeast, and a knife-point of saccharose or dextrose. These will serve to control any error which might arise, and to prove that the yeast is active. The fermentation test is performed with all the necessary precaution, provided it is delicate enough to show $\frac{1}{10}$ to $\frac{1}{20}$ of 1 per cent. of sugar in the urine.

The first of the two control tests is absolutely necessary. The author has repeatedly observed that compressed yeast often develops gas in perfectly normal urine. This is due partly to the so-called "self-fermentation" of the yeast, which bears some relation to the amount of contained glycogen, and partly to a presumed bacterial contamination, which very quickly produces an ammoniacal fermentation of the urine and a consequent development of carbonic acid (from the ammonium carbonate derived from the urea). In this fermentation, however, the gas is not formed so quickly as in the fermentation of sugar, and it

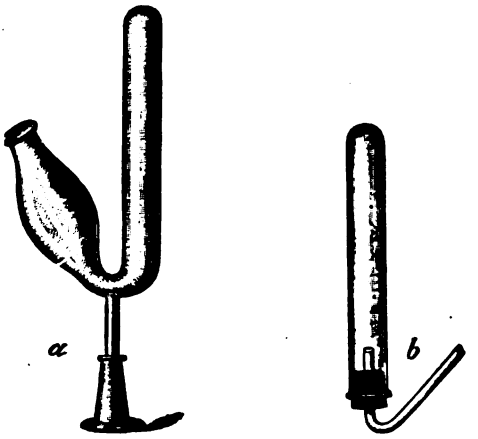

FIG. 170.—Apparatus for the quantitative test for sugar: *a*, Schrötter's fermentation tube; *b*, improvised apparatus.

should consequently be required not only that the formation of gas be considerable, but that it occur within a few hours and be unaccompanied by the signs of ammoniacal fermentation. This ammoniacal fermentation may always be suppressed by adding enough tartaric acid to make the urine decidedly acid in reaction.

Another precaution is to avoid shaking the urine too much when mixing it with the yeast, for in that case the liquid may absorb a considerable quantity of air, which afterward, upon standing, accumulates over the urine and may lead to error.

Test by Evaporation and Carbonization.

The author believes that a very simple and rather sensitive test for sugar is to evaporate a drop of urine in a porcelain dish and then heat it a little more over a gentle flame. If any considerable quantity of sugar is present the residue will be colored a pure yellow brown, even when the urine is much diluted; an odor of caramel can be appreciated before it is reduced entirely to ash, and if touched with a moistened

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finger the residue will feel excessively sticky. Urine free from sugar will exhibit only a dirty-grayish-brown residue.

OCCURRENCE AND DETECTION OF OTHER FORMS OF CARBOHYDRATES AND DIFFERENTIATION OF DEXTROSE FROM THEM. (Levulose; Maltose; Isomaltose; Lactose; Pentoses).

Strictly speaking, the fermentation test only proves the presence of a fermentable sugar in the urine; or, in other words, it merely demonstrates the presence of some carbohydrate. Now, besides dextrose, other types of fermentable sugars do occur in the urine; for instance, *levulose* and *maltose*. Thus far these have nearly always been observed in urine which also contains dextrose, and they therefore do not materially influence the value of the fermentation test in the recognition of diabetes mellitus and of glycosuria. In addition to maltose, isomaltose (which is incapable of fermentation) has also been found in urine containing dextrose (see below). The lactose which is sometimes found in the urine of nursing women and of individuals fed upon large and exclusive milk diet, and after the ingestion of large quantities of lactose (at least 100 gm.), does not ferment with yeast, although it reacts to Trommer's test. Other important points of differentiation between the various carbohydrates which occur in the urine are the varying polarimetric properties of their solutions, especially as compared with their reducing power, and, further, the more or less characteristic osazones which are formed in the phenylhydrazin test (p. 487 *et seq.*), and more especially the melting-point of the osazones. More accurate particulars upon this point will be found in text-books of chemistry. *Maltose* and *isomaltose* can be surely recognized only by the demonstration of their osazones. Maltose and isomaltose are dextrorotatory and reduce an alkaline solution of copper. Maltose is fermented by yeast; isomaltose is not. If the polariscopic determination indicates a smaller amount of sugar than that shown by the titration method, we are justified in assuming the simultaneous presence of levulose and dextrose. The levorotatory power of levulose is destroyed by yeast fermentation, while the similar rotation of oxybutyric acid and combined glycuronic acid remains unchanged (see p. 493). For the demonstration of levulose, Seliwanow's reaction may also be employed. To apply this test, the suspected urine should be mixed with an equal volume of fuming hydrochloric acid and a small quantity of a solution of resorcin (0.5 resorcin, 30 water, 30 strong hydrochloric acid). The mixture is then cautiously heated, and if an eosin or Bordeaux color appears the presence of levulose may be assumed.

If the urine contain dextrose, levulose, and oxybutyric acid (see p. 497), the recognition of each becomes still more difficult. The polarimetric and titration methods do not give the same results; the solution remains levorotatory after fermentation, but the degree of this left-handed rotation is not sufficiently large to explain the discrepancy in the results obtained by the polarimetric and titration methods of examination of the original urine.

Still another complication may be presented by the not infrequent occurrence of combined glycuronic acid in diabetic urine (see p. 492), since this substance is also levorotatory.

Detection of the Pentoses.—Pentoses are carbohydrates, each molecule of which contains 5 atoms of carbon, or some multiple of five. They have been found repeatedly in diabetic urine, but frequently also in urine without dextrose.[1]

Urine which contains pentoses possesses the power of reducing copper,[2] but such urine is either almost or quite inactive optically. They do not ferment with yeast.

[1] Salkowski and Jastrowitz, *Centralbl. f. d. med. Wissensch.*, 1892, vol. xix.; Salkowski, *ibid.*, vol. xxxii., or *Berlin. klin. Woch.*, 1895, No. 17; compare the collected account of Pentosuria by G. Bendix, Stuttgart, 1903.

[2] The reduction ordinarily occurs only after prolonged heating, and then quite suddenly throughout the entire solution.

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Tollen's reaction is commonly employed for the detection of pentoses. Salkowski describes it as follows: A little phloroglucin is heated in 5 to 6 c.c. of fuming hydrochloric acid, care being taken to leave a slight excess undissolved; the solution is divided into approximately equal parts. To one portion ½ c.c. of the urine to be examined is added; to the other portion ½ c.c. of normal urine. Both specimens are then warmed over a water bath. If the urine contains pentose an intense red zone will appear at the top in a few moments and gradually spread downward through the fluid. The control urine will not show any marked change of color. The tubes should be removed from the water bath as soon as the color has developed distinctly, because if they are heated too long the clearness of the reaction is interfered with. The red coloring matter which appears in the reaction can be extracted with amyl alcohol, and will then show spectroscopically an absorption band between the Frauenhofer lines D and E—*i. e.*, between yellow and green.

Even normal urine will sometimes furnish a doubtful coloration in this phloroglucin reaction. In such an event Salkowski recommends the *orcin test* employed by Reichel.[1] The urine is heated for twenty to thirty seconds with an equal volume of concentrated hydrochloric acid and a few particles of orcin. If the urine contains pentoses a dark-green color appears; this is due to the formation of furfurol as the result of the boiling of urine containing pentose with HCl. The only other substance which gives the same reaction is glycuronic acid. This occurs, however, in the urine only in combinations which cannot be split up with such a brief boiling with HCl. Practically, therefore, we need not consider glycuronic acid when performing this test.

A dark-green precipitate gradually forms in the dark-green solution. This precipitate may be dissolved in amyl alcohol, producing a beautiful dark bluish-green color. The spectroscopic examination of this solution shows a characteristic absorption band between C and D—*i. e.*, between red and yellow.

The urinary pentoses may be further identified by the preparation of their osazones, which is accomplished by *Salkowski* as follows: In a beaker of about 400 c.c. capacity are placed 5 gm. of phenylhydrazin, the same quantity of 50 per cent. acetic acid, and 200 c.c. of urine. The mixture is well shaken, gently heated upon a piece of wire gauze, and then in a boiling water bath. The fluid is then filtered while hot, and cooled by placing the vessel in cold water; the crystals of pentosazone are then collected upon a filter, and purified by repeated recrystallizations with hot water. The final pure product is a golden-yellow powder, and, if pentoses were present in the urine, should have a melting-point of 156° to 160° C. (166°–168° C., according to Späth).

Bial[2] has recently recommended the following procedure for the practical demonstration of pentoses in the physician's office. He believes it to be sufficiently accurate to differentiate confusing cases of pentosuria from those of glycosuria. His reagent consists of 500 c.c. of 30 per cent. hydrochloric acid, 1 gm. of orcin, and 25 drops of the official liquor ferri sesquichlorati (German Pharmacopeia). Four to 5 c.c. of this reagent are boiled in a test tube, and, after removal from the flame, several drops or, at most, 1 c.c. of urine are added. A green color should appear at once or almost immediately. If the test is carried out in this manner, Bial states that the small amount of heat employed is not sufficient to cause any reaction between the reagent and the most unstable glycuronates. Moreover, Bial recommends the procedure, not for the differentiation of pentoses and combined glycuronates, but for the differentiation of pentosuria from glycosuria. It should, however, be remembered that both pentose and dextrose may occur together in diabetic urine.

To demonstrate pentose in diabetic urine, the dextrose must be first removed by fermentation with yeast.

Pentosuria is observed temporarily, as so-called alimentary pentosuria, after the ingestion of large quantities of vegetables which are rich in pentoses, such as

[1] *Cf.* Blumenthal, *Zeits. f. klin. Med.*, vol. xxxvii.; Bial, *ibid.*, vol. xxxix.
[2] *Deutsch. med. Woch.*, 1903, No 27.

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cherries, plums, huckleberries, and other fruits. In other cases, on the contrary, the elimination of pentose is chronic and independent of the quantity and nature of the pentoses in the ingested food. In these cases there is a specific pentose which is recognized by Neuberg as optically inactive arabinose. The amount of pentose in the urine may vary between 0.1 and 1 per cent., so that the daily quantity excreted can never exceed 20 gm. A theory for this actual pentosuria is still quite impossible. But pentosuria certainly bears no relation to diabetes mellitus. Bial[1] has also described pentosuria as a common anomaly. Pentosuria does not usually give rise to characteristic clinical phenomena; and the demonstration of pentose in the urine is clinically important only because the patient is thus guarded from an erroneous diagnosis of diabetes mellitus. This is all the more important since it has repeatedly been observed that patients with pentosuria are injured by the diabetic diet containing no carbohydrates, and that such a diet does not influence the amount of excreted pentose in the slightest degree.

DETECTION OF GLYCURONIC ACID.

Glycuronic acid is formed in the body, probably even under physiologic conditions, by the oxidation of dextrose, from which it differs but little in its elementary composition. It appears in the urine when an opportunity is furnished for it to become combined in the organism with other bodies, and so to escape complete oxidation. There are a great many substances which may combine with glycuronic acid. Among them are chloral hydrate, morphin, camphor, oil of turpentine, salicylic acid, saccharin, santonin, thallin, chrysophanic acid, menthol, most of the phenols and phenol derivatives—*i. e.*, indol, skatol, naphthol, etc. Flückiger[2] has proved that, aside from the uric acid and kreatinin contained, the reducing power of normal urine depends upon the presence of the paired combinations of glycuronic acid, especially the phenol, parakresol, indol, and skatol glycuronates. P. Mayer[3] has more recently confirmed these observations, and in addition has shown that the excretion of the combined glycuronates has a bearing upon diabetic mellitus and upon alimentary glycosuria. He claims that after excessive ingestion of carbohydrates, and especially of dextrose, large quantities of the glycuronic acid salts are sometimes excreted in human urine before sugar appears. In these cases it seems as if the organism were able to oxidize the ingested sugar up to the stage of glycuronic acid, but no further. This hypothesis of the origin of glycuronic acid will explain the frequently coincident occurrence of combined glycuronates and dextrose in the urine which has been observed both in alimentary glycosuria and in diabetes mellitus. Here a part of the dextrose which is not utilized has been oxidized only to glycuronic acid. Glycuronic acid is capable of reducing but not of fermenting. This explains the peculiarity which has been observed in the urine of a diabetic patient upon a successful diet—*i. e.*, it still reduces but does not ferment. Even the reduction is peculiar in that, unlike the dextrose reduction, it occurs very slowly (see below). Many discrepancies between the quantitative estimations of the amount of sugar by the polarization, by the fermentation, and by the copper tests can thus be explained by the simultaneous presence of dextrose and some combined glycuronates. The latter, unlike the sugar, rotate to the left, but like it, reduce. Glycuronic acid has never been found in the urine except in combination. P. Mayer claims that these combinations can be split up by boiling for one to five minutes with 1 per cent. H_2SO_4, and that the peculiarities of the combined and of the artificially separated glycuronic acid can be examined in the urine. The duration of the boiling must be determined in the case of each combined glycuronate by demonstrating that the urine has changed its character in the maximum manner expected. No definite time can be given for all cases.

The following are the reactions usually employed to detect the combined glycuronates in the urine:

[1] *Berlin. klin. Woch.*, 1904, No. 21. [2] *Zeits. f. Physiol. Chemie.*, No. 9, 1885.
[3] *Berlin. klin. Woch.*, 1899, p. 617 *et seq.*, and *Deutsch. med. Woch.*, 1901, Nos. 16 and 17.

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1. The urine reduces an alkaline solution of copper. Since the combined glycuronates reduce but slowly, this reduction occurs only after prolonged heating. It will, on the contrary, take place immediately if we boil the urine beforehand, by the method mentioned above, with 1 per cent. H_2SO_4, in order to cause the clearage of the glycuronate combinations.

2. The free acid but not the combined glycuronates will enter into combination with phenylhydrazin. Hence the phenylhydrazin test, employed for demonstrating the presence of sugar (p. 487 *et seq.*), will give a negative result if we employ the ordinary urine containing the combined glycuronates; but, on the contrary, it will furnish a positive result if we precede the test by first boiling the urine with 1 per cent. H_2SO_4.

3. Glycuronic acid does not ferment, but it does reduce alkaline copper solutions; therefore a urine containing glycuronates, in spite of its reducing power, does not ferment. If sugar is also present, fermentation will produce an incomplete loss of the power of reduction.

4. The free glycuronic acid is dextrorotatory, while the combined glycuronates rotate to the left. With urine containing salts of glycuronic this levogyrate power will be changed to dextrorotation after boiling with H_2SO_4 (see above). Again, if there is also dextrose in the urine, the dextrorotatory power will be increased after boiling.

5. Tollen's reaction (see p. 491 *et seq.*) is positive with urine containing glycuronic acid as with urine containing pentoses, but not if dextrose is present alone. See Pentoses.)

6. Urine containing glycuronates reacts to the orcin test only after clearage of the combination has occurred by boiling with 1 per cent. H_2SO_4. This is in contrast to urine containing pentoses (p. 490).

DETECTION OF ACETONE (CH_3—CO—CH_3).

Traces of acetone occur in every normal urine. The amount will be increased in fever, with starvation, with a purely meat diet, in diabetes mellitus, in certain forms of digestive disturbances, and in some cases of carcinoma. According to Hirschfeld, a considerable amount of acetone may be found in non-diabetic urine if proteid substances are decomposed in the body without the simultaneous oxidation of carbohydrates. In diabetes, however, other causes are also brought to bear. Urine which contains a good deal of acetone always has a peculiar fruity odor, an odor which may be detected even in the breath of such a patient. No especial symptomatology can be determined for decided acetonuria or acetonemia. Decided acetonuria in diabetes, despite a mixed diet, suggests a gravé prognosis, although there are numerous exceptions to this rule. In other conditions the presence of acetone in the urine is as yet of no diagnostic value. In diabetes, acetonuria will be favored by a purely meat diet, and can frequently be diminished or suppressed by administering carbohydrates.

The test for acetone can first be tried with the urine itself. If the result is negative, the urine should be distilled. Acetone is very volatile, so that the distillate will be much richer in acetone than the urine, and smaller quantities can thus be detected.

Distillation can be performed without any complicated apparatus in the following way: About 50 c.c. of urine acidified with a little phosphoric acid (sufficient for a marked Congo reaction, to prevent foaming) are poured into a fractionation flask (Fig. 171) and heated to gentle

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boiling, preferably over a water bath or over a wire gauze. A test tube is then slipped over the projecting arm for a receiver and fastened with a piece of string or wire. The upper, open, end of the flask is closed with a cork. The distillate will now collect in the test tube without any special cooling apparatus. Within a few minutes several cubic centimeters will have distilled over, and the acetone test can then be performed upon the distillate.

Hoppe-Seyler claims that if diacetic acid is also present the distillation will produce acetone artificially, so that in such an event another method must be selected. Instead of being distilled, the urine is rendered slightly alkaline and then extracted with pure ether, the ether shaken with water, and the test performed upon the aqueous solution.

FIG. 171.—Fractionation flask.

To demonstrate the presence of acetone, one of the following tests is usually employed:

Gunning's Iodoform Test.—A little tincture of iodin or Lugol's solution [1] is added to the distillate, and then enough ammonia to produce an intense black precipitate of nitrogen iodid. This precipitate will gradually disappear upon standing, and when acetone is present a yellowish sediment consisting of iodoform will take its place. It can be recognized by its odor, or microscopically, as it consists of hexagonal plates or small stars (compare Fig. 172); occasionally the precipitate is amorphous. We must be careful not to confound with the iodoform crystals the stellate crystalline aggregations of triple phosphate which are often produced along with them, especially where the urine itself, instead of the distillate, is employed. (This is also possible with Lieben's test, see below.) (See Fig. 190.) If the urine contains but a small amount of acetone, it may be necessary to wait twenty-four hours before the iodoform crystals can be demonstrated. If the crystals are not distinctly formed they may be recrystallized for more certain recognition, first dissolving them in ether and then allowing this to evaporate slowly. Gunning's test has a great advantage over Lieben's (see below), because with it no other known substance, and especially neither alcohol nor aldehyd, will produce iodoform. On the other hand, it is somewhat less delicate than Lieben's. But even so, it is extremely delicate, and often readily permits the detection of acetone in diabetic

[1] Composition of Lugol's solution: 1.2 gm. iodin, 1.8 gm. potassium iodid, 30 parts of water.

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urine without distillation. The author recommends Gunning's test first of all for clinical use, and particularly when it is desirable to demonstrate acetone in the urine directly without distillation.

Lieben's Iodoform Test.—Some potassium hydrate and Lugol's solution are added to the urine, or better to its distillate. If acetone is present a yellowish precipitate will be formed, and will be recognized, as in Gunning's test (see above), by its odor and by the shape of the crystals. If only a very small amount of acetone is present, the iodoform precipitation may be delayed some hours, even up to twenty-four. This test is extremely delicate, but it is less distinctive than Gunning's, because with it other substances besides acetone may give rise to iodoform (alcohol, for example); Gunning's test is therefore preferable.

Legal's Test.—Three drops of a freshly prepared concentrated solution of sodium nitroprussid (1 : 10) are added to the urine itself, or better to its distillate, and the mixture made strongly alkaline with a few drops of sodium or potassium hydroxid. The red color which

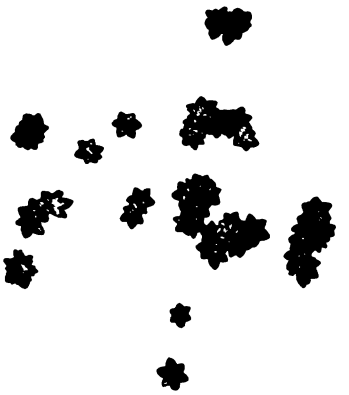

FIG. 172.—Iodoform crystals from the precipitate in Gunning's test (after a microphotograph).

arises gradually changes to yellow. This color reaction depends upon the presence of kreatinin, and will be seen in every urine. But if any considerable amount of acetone is contained in the urine the addition of acetic acid will discharge the yellow color, and the solution will become purple red or violet. This test may lead to the erroneous supposition of the presence of acetone if the urine contains parakresol (v. Jaksch). It is the one which is usually recommended for the direct examination of urine without distillation; but, according to the author's experience, it is not in that case very delicate, and he does not recommend it. If the distillate is used, however, the test is a useful one, and has recently been recommended by Studer.

DETECTION OF DIACETIC ACID ($CH_3.COCH_2.COOH$).

Diacetic acid does not occur in the urine of healthy individuals upon an ordinary diet except, perhaps, in very small quantities. It has been observed pathologically, usually in combination with ammonia, in severe

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forms of diabetes, in fever, in dyspeptic conditions and their concomitant auto-intoxications, in gastric carcinoma, and in alcoholism. Since acetone is derived from diacetic acid, the conditions requisite for diaceturia are identical with those for acetonuria, and, as a matter of fact, the two substances are almost always associated in the urine. If little diacetic acid is formed, it is all transformed into acetone; if much is formed, then both substances will be found in the urine. It frequently happens that the mother substance of diacetic acid, β-oxybutyric acid, is also present. Diaceturia, like acetonuria, also bears some relation to the metabolism of the carbohydrates, since in the diaceturia of non-diabetic individuals the proteids are decomposed without the simultaneous oxidation of the carbohydrates, and it may frequently be suppressed by the administration of carbohydrates. Diacetic acid is excreted in the urine of individuals upon a pure meat diet and in cases of starvation. The excretion of a large amount of diacetic acid by a diabetic upon a mixed diet, like the excretion of acetone under the same circumstances, is an indication of a severe form of the disease. Such diaceturia is favored by a rigid meat diet, and may frequently be diminished by the ingestion of carbohydrates. It would be just as erroneous to assume that diaceturia has a definite symptomatology as it would be to state that acetonuria is a distinct clinical entity. It should also be noted that the appearance of diacetic acid in the urine is usually accompanied by an increased elimination of ammonia, as is also the excretion of β-oxybutyric acid.

The presence of diacetic acid can be demonstrated as follows:

Gerhardt's Diabetic Ferric Chlorid Reaction.—One or two drops of a ferric chlorid solution are added to the urine. If diacetic acid is present a Bordeaux-red color appears, which is most distinct after filtering off the precipitate of ferric phosphate. If an insufficient quantity of the ferric chlorid solution has been added, an extra amount must be employed after the phosphate precipitate has been removed by filtration. Sulphocyanids, sodium acetate, salicylic acid, antipyrin, thallin, kairin, and other aromatic substances may produce a similar red color. For this reason the presence of diacetic acid should be assumed only after positive results have been obtained by the two following control tests:

1. If boiled urine is employed for the test, the red color should be very much fainter, because boiling gradually transforms diacetic acid into acetone. 2. The urine is acidified with sulphuric acid and then shaken with ether. Diacetic acid, if present, will be taken up by the ether, and, if the latter is shaken with a diluted solution of ferric chlorid, the aqueous layer will turn red. The coloring will disappear spontaneously in twenty-four to forty-eight hours. The sulphuric acid merely serves to free the diacetic acid from its salts, so that it may be taken up by the ether. Sulphocyanid salts react similarly in this control test, because sulphocyanic acid also dissolves in the ether. In that case, however, the red color does not disappear spontaneously. The color produced by sodium acetate, salicylic acid, antipyrin, thallin, and kairin also remains permanent for days.

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DETECTION OF β-OXYBUTYRIC ACID.

Thus far β-oxybutyric acid has been found in diabetes mellitus, scarlet fever, measles, scurvy, and in the insane who refuse food and drink. It is probably a preliminary stage in the formation of diacetic acid.

In diabetes mellitus a considerable amount of β-oxybutyric acid in the urine suggests the onset of acid intoxication or "acidosis" (coma diabeticum).

β-oxybutyric acid appears to be present in the urine only when diacetic acid is there also (see above).

Unfortunately, the process for demonstrating β-oxybutyric acid is very complicated. The acid must first be isolated, and then a well-characterized salt of oxybutyric acid is formed.[1] This acid is levogyrate, while dextrose is dextrogyrate. In some cases of diabetes mellitus we may therefore suspect the presence of β-oxybutyric acid if the amount of dextrose, as determined by titration, is distinctly larger than that obtained by the polariscope. Proteid must, of course, be absent, as that, as well as β-oxybutyric acid, is levogyrate. Levulose may lead to confusion.

E. Külz first removes the proteid from the diabetic urine, removes the sugar by fermentation (which will also dispose of the levulose), then decolorizes by precipitating with lead acetate and ammonia, and finally examines with the polariscope. If after this process the urine still exhibits a tendency to left-handed rotation, β-oxybutyric acid is in all probability present. To be certain we must exclude the presence of combined glycuronates. The latter condition must also be fulfilled, if we are to assume the presence of β-oxybutyric acid from the left-handed rotation of the ethereal extract obtained from the fermented urine which has been strongly acidulated with phosphoric acid.

An almost positive proof for the presence of β-oxybutyric acid in cases of diabetes mellitus is the detection of acidosis, or of a considerable increase in the quantity of ammonia excreted in the urine (see p. 538).

DETECTION OF ALKAPTON (HYDROCHINON ACETATE OR HOMOGENTISIC ACID).

The substance which has been named *alkapton* lends to the urine the peculiar property of becoming extremely dark after standing for some time. The urine turns a dark to brownish-black color from oxidation, especially if it is alkaline in reaction. Urine stains upon the clothes are dark-red to brownish. Urine containing alkapton reacts positively to Trommer's test, but it can be distinguished from urine containing sugar by its negative reaction to Nylander's test and by its inability to ferment, and by the greenish tinge produced by the addition of a dilute solution of ferric chlorid. This color will disappear promptly, but can be brought out again by the fresh addition of the reagent.

The chemical nature of alkapton was for a long time unknown. Baumann and Wolkow's[2] investigations have, however, definitely placed the substance as a derivative of hydroquinon, and regard it as the acetate of hydrochinon or homogentisic acid. Only in one case were they able to demonstrate with it a related acid—lactate of hydrochinon or uroleucinic acid. Alkaptonuria is for the most part unassociated with any discomfort, and has generally been demonstrated as a rare circumstance occurring in an apparently healthy individual. In Stange's case it was associated with dysuria. Its etiology is still quite unknown. The amount of homogentisic acid excreted seems to depend upon the amount of proteid ingested. E. Meyer, (vol. lxx., *Deutsch. Arch. f. klin. Med.*) mentions the latest work upon the subject of this puzzling anomaly, and quotes the older literature as well.

DETECTION OF LEUCIN AND TYROSIN.

Any appreciable quantity of leucin and tyrosin in the urine is always pathologic. They are nearly always in solution. Their presence is

[1] For a detailed description see Minkowski, *Arch. f. exp. Path.*, vols. xviii. and xxi., 1884, and Külz, *ibid.*, or Külz, *Zeits. f. Biol.*, vols. xx. and xxiii.

[2] *Zeits. f. physiol. Chemie*, vol. xv.

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very characteristic of acute yellow atrophy of the liver and phosphorus-poisoning, and is found more rarely in typhus, variola, pernicious anemia, and leukemia.

In order to detect them, the urine is first evaporated to about one-tenth its volume and a little alcohol then added. The characteristic crystals of the two substances can then be recognized microscopically. Tyrosin crystallizes in the shape of needles; leucin, in nodules (Fig. 173). The latter can be differentiated from the crystals of ammonium urate (see Fig. 189) by their much sharper line of contour, their less refractive power, and their absence of prickles. Leucin and tyrosin are precipitated, as a rule, in icteric urine (acute yellow atrophy of the liver and phos-

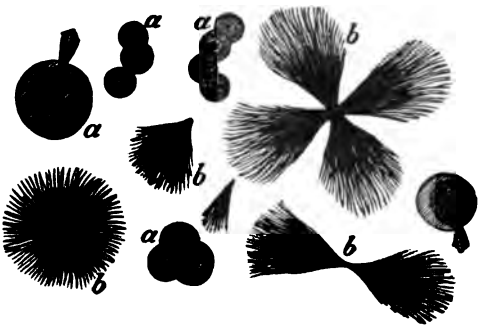

FIG. 173.—Crystals of leucin (*a*) and tyrosin (*b*) (after Ultzmann and Hofmann).

phorus-poisoning). Hence we must not confound the tyrosin needles with bilirubin needles, which are sometimes precipitated (Fig. 216) by the concentration of the urine. The latter can usually be recognized by their intense brownish-red color.

Unless the crystals of leucin and tyrosin are very characteristic, it is always much safer to perform the following test (taken in part verbally from Huppert): The urine is precipitated with basic lead acetate (liq. plumbi subacetatis). The filtrate is freed from the excess of lead with hydrogen sulphid, then evaporated as much as possible, the residue extracted with a small amount of absolute alcohol (to remove the urea), then the undissolved residue extracted with slightly ammoniacal alcohol. The extract is evaporated to a small volume and left to itself until leucin and tyrosin crystallize out. Leucin and tyrosin may now be separated by crystallization from water. That substance crystallizes first which saturates the solution first according to the amount and degree of solubility. By recrystallizing from the ammoniacal alcohol, both substances may be obtained fairly pure. The two substances may be separated by recrystallization in a little alcohol, in which leucin is more soluble than tyrosin.

The pure tyrosin which is thus formed must react to *Piria's test.* This is performed by boiling the dry tyrosin in a dry test tube with a few drops of sulphuric acid in a water bath for one-half hour. The resulting reddish solution of tyrosin sulphuric acid is allowed to cool, and then poured into several times as much water, the test tube rinsed with water, and the solution neutralized with barium carbonate. The mixture is then filtered and boiled down to a few cubic centimeters; when cool, some dilute neutral ferric chlorid is carefully added. A beautiful violet color results, quite like that of iron salicylate (p. 502). Besides this (according to Hoffmann) a hot aqueous solution of tyrosin, to which has been

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added potassium nitrate and mercuric nitrate, should produce a beautiful dark color and an abundant red precipitate so long as it is kept hot.

Leucin, on the other hand, when gently heated, is characterized by subliming without melting, and at the same time emitting an odor of amylamin. It can also be identified by *Sherer's test.* This is performed by evaporating leucin with nitric acid on a piece of platinum foil; sodium hydrate is added to the cold residue and then warmed. When the resulting solution is concentrated, it contracts to an oily drop, which rolls around and does not adhere to the platinum. Salkowski also tries the following reaction: A sample of the substance, not too small, is dissolved in water, then decolorized, if necessary, with bone charcoal; the filtrate is made alkaline with NaOH, and 1 to 2 drops of copper sulphate solution (1 : 10) added. The cupric hydroxid, which is first precipitated, dissolves, and a blue solution results from the formation of leucin-copper, which is not reduced on heating. Leucin crystals dissolve in solutions of caustic potash and are insoluble in ether. This serves to differentiate them from fat, with which they might be confused.

DIAZO-REACTION.

Ehrlich[1] tried to discover unknown constituents of the urine by employing the diazo-compounds, which produce colored combinations with a large number of aromatic substances. The nature of the body which is found in certain pathologic urines, and which gives the so-called diazo-reaction, is still unknown. Späth claims that the reaction should be ascribed to oxyproteic (uroproteic) acid, a recently discovered[2] normal urinary constituent, in case the urine contain an increased quantity of this substance. The author cannot confirm this statement.

The test is performed as follows: Two stock solutions are employed. (1) A 0.5 per cent. solution of sodium nitrite. (2) A solution of 5 gm. of sulphanilic acid in 50 c.c. of hydrochloric acid and 1000 c.c. of distilled water. Fifty cubic centimeters of the latter, freshly mixed for each test with 1 c.c. of the former solution, make up the reagent. It may be prepared approximately in a test tube, in the absence of a measuring glass, by adding to about 3 c.c. of solution No. 2 a drop of the nitrite solution (No. 1). Equal parts of urine and of the reagent are mixed and then quickly supersaturated with ammonia. The characteristic reaction now consists in the mixture turning more or less intensely red, the foam which forms upon shaking being tinged from a rose to deep red; the reaction is then positive. The brownish-yellow color which appears when the test is performed with any normal urine must not be mistaken for the characteristic rose-red coloration. When the test is positive and the mixture is allowed to stand, after some time a precipitate forms, whose upper margin exhibits a dark zone of a green or greenish-black or even violet shade. Any deviation from the above procedure will produce other results, and should therefore be avoided—*e. g.*, the supersaturation with ammonia must be done at once, and not drop by drop.

Although it may be incorrect to assume that the diazo-reaction is dependent upon an increased excretion of uroproteic acid, the reaction may nevertheless be regarded as an indication of a pathologic decomposition of the proteids.

[1] *Zeits. f. klin. Med.*, vol. v., p. 285, 1882.
[2] Almost simultaneously by Bondzynski and Gottlieb and by M. Cloetta.

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So far as the clinical results are concerned, Ehrlich claims that the reaction is never found in healthy individuals, and in non-febrile diseases only exceptionally, and in the following instances: 1, In advanced cardiac disease; 2, in chronic hepatitis; 3, in carcinoma, especially of the pylorus; 4, in leukemia; 5, in marasmus senilis; 6, in the cachexia of malaria; 7, in cold abscesses. So far as febrile affections are concerned, they may be divided into three subdivisions: 1, Those in which the reaction is almost always absent—*e. g.*, joint rheumatism, meningitis; 2, those in which it may occur sometimes more often, sometimes less often, according to the nature of the attack—*e. g.*, pneumonia, scarlet fever, diphtheria, erysipelas, phthisis; 3, those in which it is of nearly constant occurrence—*e. g.*, typhoid, typhus, measles.

The occurrence of the reaction in either of the first two categories of febrile disorders appears to render the prognosis more grave.

In typhoid the reaction may be of diagnostic value in one of two ways: First, the persistent absence of this reaction in a disease which simulates a severe typhoid speaks considerably, although not absolutely, against such diagnosis. Secondly, typhoid recurrences or relapses may be distinguished by this reaction from intercurrent lung affections and other febrile complications. With a recurrence or a relapse the reaction will return or be increased; otherwise, this is not the case.

The reaction is found quite frequently in phthisis, especially in severe and progressive cases. Here it suggests a bad prognosis, and is, as it were, an index to the general serious condition. Unfortunately, the reaction does not seem to be of any value in differentiating acute miliary tuberculosis from typhoid, as it is often present and very intense in the former.

In puerperal infection the appearance of the reaction is coincident with the fever.

Rüttimeyer has also observed the reaction in pulmonary actinomycosis.

The diazo-reaction must be considered as a metabolic symptom of certain diseases, which (like splenic swelling and fever) is not of diagnostic value in itself, but only when considered in connection with the other symptoms. It should also be noted that a similar reaction may be obtained after the administration of opium, morphin, chrysarobin, naphthalin, heroin, dionin, tannic acid, alcohol in large quantities, phenol, cresol, creosote, and guaiacol. This fact must consequently be borne in mind before drawing conclusions as to the diagnostic value of the diazo-reaction.

The so-called *egg-yellow reaction*, as described by Ehrlich, is said to be somewhat characteristic of pneumonia just before and during the crisis. It is a preliminary reaction—*i. e.*, a diazo-reaction which occurs before the addition of ammonia. The urine becomes deep yellow after the addition of the reagent, the color showing most distinctly in the foam. It does not turn red when ammonia is added, but becomes lighter yellow. Oppenheim found it constantly in 28 of his cases at the crisis. In some conditions we might predict the crisis from this reaction.

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Ehrlich claims that the chromogen of urobilinogen is an alteration product of the bilirubin arising from the hemoglobin in the exudate.

EXAMINATION OF THE URINE FOR SUBSTANCES INTRODUCED INTO THE BODY FROM WITHOUT (DRUGS AND POISONS).

DETECTION OF LEAD.

A shining strip of magnesium free from lead is placed in the urine and left there for some time; the deposit is dissolved in nitric acid and then tested according to the rules of inorganic chemistry.[1] If the urine contains but a small amount of lead, this method will not give the desired result; a larger quantity of the urine must be used, the organic substances destroyed with hydrochloric acid and potassium chlorate, and the lead sought for in the evaporated residue.[2]

DETECTION OF MERCURY.

To 500 to 1000 c.c. of urine are added 2 to 4 c.c. of hydrochloric acid (Fürbringer). It is then digested at 60° to 80° C. for five to ten minutes in a flask with ¼ to ½ gm. of brass-wool, and frequently skaken. The metal is washed first with water, then with alcohol, and finally with ether, and dropped into a glass tube of high melting-point, which has been prolonged into a capillary tube at the upper end. It is there brought to a red heat, the capillary end being kept uppermost. The mercury, which has become amalgamated with the brass-wool, becomes volatile and will be deposited as a mirror at the capillary end of the tube. If a particle of iodin is evaporated in the tube, the deposit will be colored red by the formation of iodin of mercury. (In reference to other methods, see Späth, p. 399 *et seq.*)

DETECTION OF IODIN.

Iodin occurs in the urine as potassium iodid after the internal and external administration of iodin or one of its preparations. It may be easily demonstrated as follows: A few cubic centimeters of urine are boiled with a piece of starch about the size of a pea until the latter is dissolved. After cooling the fluid is carefully stratified over concentrated nitric acid. If iodin is present, a blue-violet ring that gradually disappears is formed at the line of junction of the two fluids. In this test iodin is liberated from the potassium iodid by the nitric acid, and unites with the starch to form blue iodid of starch.

Another way is to add to the urine 5 to 10 drops of crude nitric acid and ½ c.c. of chloroform, and then shake gently. If iodin is present the chloroform which settles to the bottom will be colored a rose red to violet, due to the solution in the chloroform of the free iodin.

To prove that a patient has really taken a drug prescribed, introduce a grain or two of potassium iodid into the prescription. Iodid should then be demonstrated both in the urine and in the saliva.

Both the above tests are very delicate. But if the urine contains but a very slight trace of iodin, the chloroform test may not be very specific, since the nitric acid may set free indol and skatol pigments as well as urorosein (see p. 478), and so tinge the chloroform reddish. But in this case the urine itself usually appears more deeply colored than the chloroform. The starch reaction may not be of much help in deciding in such a case, because the blue ring of the iodid of starch may be simulated by an indican reaction, for in a urine so rich in such chromogen not only calcium chlorid, but sometimes even nitric acid (see p. 477), will produce the change. Here, however, it is easy enough to show that the nitric acid alone is capable of producing this blue ring without the addition of any starch.

Palladium chlorid furnishes still another iodin reaction. The urine suspected

[1] Salkowski, *Practicum der physiol. u. path. Chemie*, 2d ed., 1900.

[2] For the accurate procedure, see Lehmann, *Zeits. f. physiol. Chemie*, vol. vi., p. 4, 1882.

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to contain iodin is strongly acidulated with hydrochloric acid, and then a few drops of a moderately saturated solution of palladium chlorid in hydrochloric acid[1] are added.

If iodin is present a brown discoloration occurs, and gradually a black precipitate. This reaction has but one meaning, but it is much less delicate than the starch or the chloroform reaction.

DETECTION OF BROMIN.

The test for bromin is performed in exactly the same way as the iodin test with chloroform, except that a few drops of a calcium chlorid solution and hydrochloric acid are used to liberate the bromin. If bromin is present the chloroform will be colored yellowish brown by the free bromin. This test, although much less delicate than the iodin test, is sufficiently accurate to recognize the therapeutic ingestion of large doses of bromin salts. To add to the uncertainty of this test, we must remember that the chloroform can also be tinged yellow by some of the urinary coloring matters. To prevent this latter source of error, 10 c.c. of the urine, to which have been added 2 c.c. of caustic potash and 2 c.c. of potassium nitrate, are incinerated, the ash dissolved in water, and the resulting solution tested for bromin, as above.

A. Jolles[2] has described an admirable test which is quite simple. It depends upon the fact that bromin can be freed from the urine by potassium permanganate in acid solution, and that such free bromin gives a red color with *p*-dimethylphenylendiamin. Filter paper is moistened with an aqueous solution of the hydrochlorid of *p*-dimethylphenylendiamin, dried, and cut into strips. Ten cubic centimeters of urine are acidulated in a narrow-necked flask with H_2SO_4, saturated with potassium permanganate. The color is now a permanent red. A moist strip of the above test paper is suspended in the neck of the flask, and the latter is warmed over a water bath. If bromin is present the paper will be colored from violet, through blue and green, to a brown. Iodin and chlorin present other brownish shades different enough not to be confusing.

The demonstration of bromin in the urine is of no special interest except, of course, in verifying a diagnosis of suspected bromism.

DETECTION OF SALICYLIC ACID.

Ferric chlorid is added to the urine drop by drop. If the latter becomes a more or less intense deep violet, the reaction is positive. It is very delicate, and can be used in the same way as the iodin reaction to control the ingestion of drugs. Salicylic acid and its salts, in which latter form the administered salicylic acid partly appears in the urine, both give this reaction. (Compare p. 496 with reference to the differentiation of the salicylic acid reaction from the similar reaction of diacetic acid.)

DETECTION OF PHENOL.

Phenol appears in the urine largely as phenol sulphate. Ferric chlorid will produce a violet-blue color in the distillate from a phenol urine to which 5 per cent. sulphuric acid has been added. Phenol urine turns dark to black upon exposure to the air. This is due to the fact that it contains hydroquinone and pyrocatechin, which form dark-colored derivatives upon oxidation.

DETECTION OF ANTIPYRIN.

The urine appears dark and is dichroic—in reflected light greenish, in transmitted light reddish. A brownish-red color gradually appears upon the addition of ferric chlorid solution.

[1] This solution is prepared by adding 1 gm. of palladium chlorid to a few drops of hydrochloric acid, allowing it to stand for a day, and then diluting the resulting solution with enough hydrochloric acid to make a faintly transparent brown solution.

[2] *Wien. klin. Rundschau*, 1898, No. 12.

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DETECTION OF THALLIN.

The urine is yellowish green to dark brown, and turns brownish red upon the addition of ferric chlorid solution. If the urine is shaken with ether, the latter will be tinged green [1] upon the addition of ferric chlorid.

DETECTION OF PHENACETIN.

The urine is dark yellow, and turns reddish brown upon the addition of ferric chlorid solution. The color gradually becomes black after prolonged standing.

DETECTION OF ANTIFEBRIN.

The urine is extracted with chloroform, and to the extract mercuric nitrate is added. The mixture is then heated, and if a green color is produced [2] antifebrin is present.

DETECTION OF PYRAMIDON.

The urine is frequently clear, purplish red in color, and deposits a sediment consisting of small red needles. If the urine be mixed with an equal volume of a 2 per cent. solution of ferric chlorid, a dark-brownish amethyst color is produced. According to Jolles, a violet ring, gradually becoming red, is obtained when the urine is carefully covered by a layer of dilute alcoholic solution of iodin (a 10 per cent. tincture of iodin diluted with 10 parts of alcohol).

DETECTION OF TANNIN.

Tannin is eliminated in the urine partly as gallic acid. Urine which contains tannic and gallic acids will turn blue black upon the addition of ferric chlorid solution (ink reaction).

DETECTION OF BALSAM OF COPAIBA AND SANDALWOOD OIL.

After the administration of balsam of copaiba the urine will reduce cupric oxid (Trommer's test) but not bismuth (Nylander's test). If hydrochloric acid is added to the urine drop by drop, a precipitate of resinous acids appears with a reddish to violet coloration. Also after the use of sandalwood oil the urine possesses reducing properties, and will exhibit a precipitate of resinous acids when hydrochloric is added, but with a reddish-brown color. Karo claims that this color is not characteristic. Alexander [3] emphasizes the fact that the intensity of the resinous acid precipitate is not proportioned to the color reaction produced, and that, with regard to the amount of the resinous acid excreted, individuals differ greatly when the same dose of copaiba balsam or sandalwood oil has been administered.

DETECTION OF SANTONIN.

Santonin urine is of a saffron-yellow to greenish color. The addition of sodium hydrate will turn it a rose-red shade. If this rose pigment is shaken with amyl alcohol, it will be immediately dissolved by the latter, giving it a beautiful and intense coloring, while the urine will become decolorized.

DETECTION OF EMODIN, CHRYSOPHANIC ACID, AND SUBSTANCES RELATED THROUGH THE OXYMETHYLANTHRAQUINONE GROUP, RHUBARB, SENNA, RHAMNUS (Cascara Sagrada), ALOES.

After the ingestion of rhubarb, of the different kinds of rhamnus (R. frangula and R. purshiana or cascara sagrada), of senna, and of large doses of aloes, the urine will be tinged a brownish yellow, and upon the addition of an alkali (sodium, potassium, or ammonium hydroxid) will become more or less distinctly red. This peculiarity depends upon the presence of emodin (trioxymethylanthra-

[1] v. Jaksch, *Zeits. f. klin. Med.*, vol. viii., p, 551.
[2] Yvon, *Jour. de Pharm. et de Chemie*, 1887, No. 1.
[3] *Deutsch. Med. Woch.*, 1893, No. 14, p. 324.

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quinone) and of chrysophanic acid (dioxymethylanthraquinone). These substances are excreted in the urine partly as such and partly in combination. Even after the external use of chrysarobin the urine sometimes exhibits the same peculiarities, probably because chrysophanic acid is formed in the body from chrysarobin. Urine containing one of these substances differs from santonin urine in this respect: the red coloring matter produced by the addition of alkalies will not dissolve in the amyl alcohol. The reaction common to chrysophanic acid and emodin, and recently named the *oxymethylanthraquinone reaction*, has been modified and made more delicate, according to Tschirch, by boiling the urine in a test tube with 1 or 2 drops of caustic potash, in order to split up the combined bodies, then acidulating with HCl, extracting with ether, and shaking the ether with ammonia. After a short delay the ether separates from the ammonia, and the latter is tinged a beautiful cherry red. Chrysophanic acid passes from the ether into the ammonia with difficulty, so that, if the ether remains yellow despite the red coloration of the ammonia, we are justified in deciding upon the presence of chrysophanic acid instead of emodin.

QUANTITATIVE URINARY ANALYSIS.

Preliminary Note.—Since in the quantitative analysis of any urine the amount of the particular constituent excreted in twenty-four hours is required, the specimen examined must be a portion of a well-mixed twenty-four-hour urine.

QUANTITATIVE ESTIMATION OF UROCHROME.

Klemperer[1] has recently made a noteworthy attempt to attach a diagnostic value to the amount of normal urinary pigment contained in the urine. The normal urinary pigment, and frequently the only one, is urochrome; it holds a close chemical relation to urobilin, into which it may be converted by cautious oxidation. According to Klemperer, the normal color of the urine corresponds approximately to 0.15 per cent. urochrome solution. He determines the amount of contained urochrome colorimetrically by comparison with a solution of "pure yellow" ("Echtgelb" of Leitz). Klemperer prepares his solution for comparison by dissolving 0.1 gm. of dry "pure yellow" in a liter of water, and diluting 5 c.c. of this solution to 90 c.c. According to Klemperer, the shade of this solution of "pure yellow" (1 : 180,000) corresponds to a 0.1 solution of urochrome, and by diluting the urine or the test solution, the quantity of contained urochrome may easily be determined by the colorimetric method. The urine and the test solution must, of course, be contained in vessels of the same shape when the comparison is made. Stronger solutions of "pure yellow" cannot be employed, since their color does not agree with that of urine. Should the urine contain considerable quantities of other pigments, such as urobilin or hematoporphyrin, an exact colorimetric estimation of the urochrome is, of course, impossible. Klemperer nevertheless believes that if the presence of these other pigments is not disclosed by absorption bands in the spectroscopic examination (urochrome absorbs light diffusely), the method given above will be sufficiently accurate. He also believes that the estimation of the daily excretion of urochrome furnishes an indication of the functional activity of the kidneys, since he assumes that urochrome is formed in the kidneys. It is nevertheless clear that if the latter assumption be incorrect, and it has not yet been proved, the amount of excreted urochrome is not of such simple significance, since it would not depend upon the kidneys alone, but also upon the unknown function upon which the formation of urochrome in the body is dependent. The fact that pale urine is frequently excreted in uremia could also be due to the fact that, in addition to the impairment of the excretory function of the kidneys, other important functions holding some relation to the formation of urochrome are also implicated.

[1] *Berlin. klin. Woch.*, 1903, No. 14, p. 314.

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QUANTITATIVE ESTIMATION OF PROTEIDS.

ESTIMATION BY OBTAINING AND WEIGHING PURE (COAGULABLE) PROTEID, OR KJELDAHL'S METHOD.

An exact quantitative estimation of coagulable proteid must depend upon a complete precipitation of the proteid by boiling after the addition of dilute acetic acid (2 per cent.). (See Removal of Proteid from Urine, p. 463.) The precipitate must then be washed, and dried upon a dry-weighed filter at 110° to 120° C. to a constant weight, the dried residue weighed, and the weight of the dry filter subtracted. To save time in drying the precipitate, the amount of urine should be small, so that the weight of the dry proteid will not exceed 0.2 to 0.3 gm. (preliminary estimation). (See below, Estimation of Quantity of Proteid according to Esbach.)

Proteid is exceedingly hydroscopic; hence, the weighing should be very carefully carried out. The material should be placed between watch glasses, with the ground edges carefully applied to each other. For very great accuracy the albuminous precipitate must be first washed with alcohol and ether, in order to remove the fat before drying, and finally the content of ash determined and subtracted. For most clinical purposes this method, which is the only one absolutely accurate, is too complicated. Approximate estimations usually answer the purpose. There are quite a number of such methods, of which the following are the most practicable:

FIG. 174.—Esbach's albuminimeter.

ESBACH'S ESTIMATION OF PROTEID.

This method consists in determining the *volume* of proteid which is precipitated from a definite amount of urine by a certain solution. A graduated test tube of thick glass, called an albuminimeter (Fig. 174), is filled to the mark U with the acidified urine to be examined, and then a solution (10 gm. of picric acid and 20 gm. of citric acid in 1 liter of distilled water) for precipitating the proteid is added up to the mark R. The tube is closed with a rubber cork, and the liquids mixed by repeated inversion without shaking. The glass is now set upright in a test-tube rack. The precipitated proteid gradually settles to the bottom, and after twenty-four hours the height of the layer can be quickly seen. The graduations indicate the grams of proteid in the liter. With some instruments the graduation reads as high as 1.2 per cent. If the amount of proteid is so large (0.5 per cent.) that the precipitate settles unevenly, it is safer to dilute the urine once or twice, or even four times, before performing the test. In Esbach's method the limit of accuracy for a moderate amount of proteid is perhaps 0.1 per cent. If the amount of proteid is large, or if it is less than 0.05 per cent., the chances for error become considerable. A very small quantity will not sediment, so that it cannot

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be estimated by this method. The empirical graduation of Esbach's albuminimeter presupposes, of course, the average room temperature. The result will be decidedly different if the surrounding temperature varies much from the normal. If the urine contains a considerable quantity of resinous acids—*e. g.*, after ingestion of the balsams or of santalwood oil—this method cannot be employed, because these acids will be precipitated by the picric acid along with the proteid (p. 463). Weighing will then have to be resorted to, and any resinous acid which may happen to be precipitated will be removed from the proteid precipitate by the washing with alcohol and ether.

ROBERTS-STOLNIKOW'S (BRANDBERG'S) METHOD FOR THE ESTIMATION OF PROTEID.

A method described by Roberts and Stolnikow simultaneously, and then tested more accurately by Brandberg, can be recommended if we do not happen to have an albuminimeter. This test depends upon the fact that the turbidity of proteid in the nitric acid test appears more or less quickly according to the amount of proteid contained in the urine. If the urine contains 0.0033 per cent. of proteid, the cloudiness first appears within two and one-half to three minutes. By diluting the urine sufficiently to cause the turbidity to appear within that interval, the amount of proteid actually contained in the specimen can be readily determined. Naturally, great care must be exercised in performing the reaction. If a cloudiness occurs inside of three minutes, we employ a dilution of 1 : 10 (A). If this dilution is still not sufficient, mixture B may be prepared by diluting 1 part of A with 2 parts of water. If even this mixture (B) is too concentrated, we make up a third dilution (C), consisting of 1 part of B with 4 parts of water; and again, if necessary, a fourth dilution (D), consisting of 1 part of C with 1 part of water.

A	corresponds to	0.033	per cent.	of proteid.
B	"	0.10	"	"
C	"	0.50	"	"
D	"	1.00	"	"

If the urine contains either nucleo-albumin or resinous acids, this method is not available, because nitric acid precipitates both of these bodies along with the proteid (p. 462).

QUANTITATIVE ESTIMATION OF DEXTROSE.

In diabetes mellitus the amount of sugar in the urine generally ranges from 4 to 5 per cent., but it may be as high as 10 per cent., so that in the course of twenty-four hours 1 kgm. or more may be excreted.

ESTIMATION OF THE AMOUNT OF SUGAR FROM THE SPECIFIC GRAVITY AND THE QUANTITY OF IRON.

Naunyn constructed an approximately accurate table for estimating the amount of sugar in diabetic urine, as follows:

With a daily quantity of:

2 liters	and with a	specific	gravity	between	1028–1030,	about	2–3 per cent.
3	"	"	"		1028–1032	"	3–5 "
5	"	"	"		1030–1035	"	5–7 "
6 to 10	"	"	"		1030–1042	"	6–10 "

The percentage of sugar in the urine can be approximately determined from the quantity and specific gravity if we deduct as much from the specific gravity found as would represent the specific gravity of a normal

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non-sugar urine diluted up to the same quantity by the excessive ingestion of water. For instance, if 2 liters represent the amount of the twenty-four-hour excretion, the specific gravity would ordinarily be about 1015. Then if the volume is still further increased by abundant water-drinking, a urine whose twenty-four-hour quantity amounts to 3 liters will have approximately a specific gravity of

$$\frac{2 \times 1015 + 1000}{3} = 1010,$$

and one of 6 liters, a specific gravity of

$$\frac{2 \times 1015 + 4000}{6} = 1005.$$

Now, suppose a diabetic urine (*a*), whose twenty-four hour amount equals 3 liters, and another (*b*), whose twenty-four-hour amount equals 6 liters, have the same specific gravity, 1030. The sugar contained, then, in urine *a* is sufficient alone to produce a specific gravity of 1030 — 1010 = 1020, and in urine *b* to 1030 — 1005 = 1025. From this the amount of sugar present can be approximately calculated by multiplying the last two figures of the specific gravity by 0.23. (See p. 513, *f.*, Quantitative Areometric Fermentation Test.) This gives for urine *a* 4.6 per cent., and for urine *b* 5.7 per cent. Such an estimate is, of course, inaccurate, if for no other reason than that diabetes mellitus also alters the amount of urea and salts eliminated, which in turn also influences the specific gravity of the urine.

ESTIMATION OF THE QUANTITY OF DEXTROSE BY TITRATION.

Fehling-Soxhlet's Method.

Dextrose will reduce cupric oxid in an alkaline solution to cuprous oxid. This property is the one most frequently employed in titrating for sugar; because when certain conditions are maintained the reduction of the alkaline cupric oxid solution by the sugar takes place quantitatively. *Fehling's solution* is the one generally employed. It is prepared as follows:

34.64 gm. purest crystalline copper sulphate, 173 gm. Rochelle salt (sodium potassium tartrate), 100 c.c. sodium hydrate solution of specific gravity 1.34, distilled water up to 1000 c.c.

Fehling's solution cannot be preserved in its original form. It is therefore advisable to keep two separate solutions, which should not be mixed until just before using. Solution 1 contains 34.64 gm. of copper sulphate dissolved in 500 c.c. of distilled water acidulated with a drop of concentrated sulphuric acid. Solution 2 contains 173 gm. of Rochelle salt with 100 c.c. of NaOH solution (specific gravity 1.34), and the volume made up to 500 c.c. with water. By mixing equal volumes of these solutions, a fresh Fehling's solution can be obtained for every test. This mixture contains sufficient cupric oxid, so that 10 c.c. of the solution when diluted five times with water will be reduced to the red cuprous oxid when boiled with 0.05 gm. of grape sugar (or, according to Soxhlet, 0.0473 gm). If the urine contains more than 1 per cent. of dextrose it should be diluted with water, before titration, until it does not contain more than that amount. The above-mentioned rules for approximately estimating the amount of sugar from the specific gravity can be used as the basis for this procedure. As a rule, more than 5 c.c. of urine are required to reduce the 10 c.c. of Fehling's solution. Albuminous urine must be freed from proteid by boiling the acidified urine, and filtering before titration (see p. 463).

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Method: 5 c.c. of solution 1 and 5 c.c. of solution 2 are mixed in a small flask, diluted with water up to 50 c.c., heated to boiling, and the urine, appropriately diluted, added a few drops at a time from the buret. The mixture is kept boiling gently until it is approximately decolorized and an abundant precipitate of the red cuprous oxid has appeared. We cannot, of course, expect to obtain accurate results with this method (Fehling's original method), because the end-reaction cannot be recognized accurately, and because a portion of the suboxid always dissolves in the ammonia liberated from the urine and becomes reoxidized. This test really gives only approximate values.

For accuracy Soxhlet's modification should be resorted to. He pours the approximate amount of urine into the boiling Fehling's solution at once (he also uses 10 c.c. of Fehling's solution diluted to 50 c.c. with water), then allows the solution to boil for two minutes, and then takes the flame away. The shining meniscus at the upper margin of the fluid is his index of the end-reaction. If this persists in being still slightly blue he repeats the test with a fresh and slightly larger volume of urine and fresh Fehling's solution until he determines the exact amount that is required to decolorize the fluid, as shown by the meniscus.

The calculation of the result is very simple. If 9 c.c of urine diluted 10 times are required to reduce 10 c.c. of Fehling's solution, then 0.9 c.c. of urine contains 0.05 gm. of dextrose.

$$0.9 : 0.05 = 100 : X$$

$$X = \frac{5}{0.9} = 5.5 \text{ gm. of grape sugar}$$

—*i. e.*, the urine contains 5.5 per cent. of dextrose.

Or if we employ the exact figures of Soxhlet:

$$0.9 : 0.0473 = 100 : X$$

$$X = \frac{4.73}{0.9} = 5.26$$

Various other modifications of Fehling's test have been suggested to overcome the difficulty of sharply determining the change of color of the solution—*i. e.*, the end-reaction. Pavy's[1] method, one of the best known, adds a certain amount of ammonia to the Fehling's solution to produce a colorless cuprous oxid combination. The end-reaction will then consist in a complete decoloration of the blue copper solution. Pavy keeps the fluid boiling, and adds the urine from a buret, drop by drop. The difficulties of this procedure are, however, scarcely less than those of Fehling's titration. First of all, air must be excluded during the titration, so that the buret has to be fastened air-tight into the flask, otherwise the colorless solution always oxidizes. Secondly, the ammonia fumes which arise are most annoying. Thirdly, as a consequence of the gradual addition of the urine, cuprous oxid will be precipitated by the removal of ammonia occasioned by the too-prolonged boiling of the solution. Pavy's method, therefore, necessitates, if anything, more practice than Fehling's. It is more useful when Soxhlet's modification is adopted (addition of all the urine at one time).

Practically speaking, Soxhlet's method is the only safe one for the practitioner; the others require especial practice and skill, otherwise they are too uncertain. It is well to note here that many if not most of the old results from Fehling's method were probably inaccurate; hence, we should be on our guard against accepting them unreservedly.

Dextrose Titration According to Drechsel-Klimmer.

Klimmer[2] has published Drechsel's method for sugar titration since the latter's death. It seems to offer a sharp end-reaction. It is based upon the fact that in

[1] "Physiologie der Kohlehydrate," German by K. Grube, Wien, Deuticke, 1895.

[2] "Ist Zucker ein normaler Bestandteil des Harnes unserer Haussäugetiere?" and "Zwei neue klinische Methoden der quantitativen Zuckerbestimmung im Harne," I. A. D. Bern, 1898.

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the presence of guanin the red suboxid which is formed in Trommer's test will unite with guanin to form a less readily oxidizable combination of a white color, so that in titration with Fehling's solution to which a certain amount of guanin has been added the solution may be filtered off from the precipitate and tested for copper. For all clinical purposes the decoloration of the filtered fluid is a sufficiently sharp end-reaction. But when great accuracy is essential the filtrate must be examined for copper by acidifying and then adding potassium ferrocyanid (brown precipitate of copper ferrocyanid). The filtration must be performed through a double filter. Albuminous urine must be freed from proteid. The technic is as follows: A 1 : 20 normal guanin solution is prepared by dissolving 9.375 gm. of guanin hydrochlorid in 1000 c.c. of 1 per cent. solution of NaOH, so that 1 c.c. of the 1 : 20 normal guanin solution contains 0.00755 gm. of pure guanin. Fifteen cubic centimeters of this guanin solution are added to 10 c.c. of Fehling's solution, and the mixture then diluted with 25 c.c. of distilled water. Before titration the urine should be diluted so that it does not contain more that 0.5 to 1 per cent. of sugar. The total amount of urine employed to produce the end reaction will contain 0.05 gm. of dextrose. If the reduction produces a red color, the titration must be repeated with the addition of more guanin to the Fehling's solution. For a very exact determination, the titration should be performed before and after fermentation, in order to eliminate any error caused by the other reducing substances of the urine, and then the difference in the quantity of urine used in both cases made the bases for further calculation.

This method is accurate, but only when very pure guanin is used, and that is rather expensive.

LEHMANN'S IODOMETRIC TITRATION METHOD FOR DEXTROSE.[1]

In this method a definitely measured amount of urine is boiled with Fehling's solution [just as in Soxhlet-Allihn's (p. 510)], and the copper which remains in solution in the filtrate is titrated. This is accomplished by adding a measured amount of potassium iodid solution of a definite specific gravity to the filtrate acidified with sulphuric acid. Iodin, which is set free according to the equation $2CuSO_4 + 4KI = 2K_2SO_4 + Cu_2I_2 + I_2$, is then titrated with sodium thiosulphate, using starch paste as an indicator. Every atom of free iodin corresponds to one atom of copper in the solution.

In addition to what is essential for Soxhlet's estimation, this method requires a decinormal sodium thiosulphate solution, prepared as follows: 24.8 grams of sodium thiosulphate and 2 gm. of ammonium carbonate are each dissolved in water, and the solution diluted up to one liter. One cubic centimeter of this solution corresponds to 0.00635 gm. of copper.

A good asbestos filter is also necessary. The one described upon p. 510 may be employed. However, we do not really need a suction filter; and, as a matter of fact, a sufficiently accurate asbestos filter can be readily constructed by shoving a small plug of glass-wool into the opening at the neck of an ordinary glass funnel, in order to support the asbestos, and then pouring upon this packing a finely divided emulsion of asbestos in distilled water until the layer of filtering asbestos is sufficiently thick. It is a good plan, after the water has drained away, to compress the asbestos a little with the finger.

We now mix in a small flask 10 c.c. of the copper sulphate solution used for the Soxhlet-Allihn's solution with 10 c.c. of the corresponding alkaline tartrate solution, and add 30 c.c. of water, heat to boiling, and then pour in 10 c.c. of the urine to be examined. This urine[2] must be free from proteid, and, if necessary, must be diluted sufficiently not to contain more than 1 per cent. of dextrose (see p. 507). After the addition of the urine the mixture is allowed to

[1] *Arch. f. Hygiene*, vol. xxx., and *Zeits. f. anal. Chemie*, 1898, part 4, and *Pharmac. Post*, 1898, No. 30. *Cf.* also E. Riegler, *Zeits. f. anal. Chemie*, 1898, part 1, and Benjamin, *Deutsch. med. Woch.*, 1898, No. 35, p. 552.

[2] The urine must first be freed from proteid, because otherwise the reduced copper oxid would not filter off.

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boil for two minutes more; then the cupric oxid which has been formed is filtered off by the asbestos filter and finally washed with water.

The filtrate is iodometrically examined. For this purpose 2 c.c. of concentrated sulphuric acid are added to it, and, after cooling, 1 gm. of potassium iodid dissolved in about 10 c.c. of water. Iodin immediately separates out with a change of color. Now we deliver from a buret enough decinormal sodium thiosulphate solution to make the fluid begin to clear up, then (or if preferred even before beginning to add the thiosulphate) add a few cubic centimeters of a thin starch paste as an indicator, and titrate again until the blue coloration disappears. According to the author's experience, the thiosulphate solution should not be added too quickly, because the decoloration does not take place immediately. Shortly after the blue color has disappeared (it very frequently soon reappears) to estimate the end-reaction we must be very careful to see that the decoloration persists for at least five minutes.

In making the calculation it must be remembered that the 10 c.c. of the copper solution which we have employed corresponds to 27.8 c.c. of a decinormal thiosulphate solution. If v equals the number of cubic centimeters of thiosulphate solution employed, then $27.8 - v$ equals the number of cubic centimeters of thiosulphate solution which correspond to the amount of reduced copper; consequently, $27.8 - v \times 0.00635$ is the amount of reduced copper. The amount of sugar corresponding to this reduction will be found in Allihn's table, p. 512.

According to the tests which the author has made with this method he believes that it can be recommended as one of the most trustworthy and convenient of the clinical methods for the determination of sugar. The only real difficulty is the preparation of the asbestos filter; but after a little practice this can be done easily enough if the directions given above are followed.

SOXHLET-ALLIHN'S METHOD OF DEXTROSE ESTIMATION.[1]

This is a thoroughly accurate method, perhaps the most reliable of all, and certainly to be recommended for scientific investigation. It is rather too complicated for the practising physician, but in a clinical laboratory, after the necessary apparatus is once put together, it may be performed in a reasonably short time. In this method a definite excess of Fehling's solution is partly reduced by a measured volume of urine; after filtering off the solution the red cuprous oxid previously formed is collected upon an asbestos filter, reduced in a current of hydrogen, and the resulting metallic copper weighed, from which the amount of sugar contained in the urine can be calculated. Albuminous urine must first be freed from proteid (compare p. 463 *et seq.*). The following two solutions are required:

Solution 1: 173 gm. of Rochelle salts, 125 gm. of potassium hydroxid dissolved in water, and the volume made up to 500 c.c.

Solution 2: 34.6 gm. of crystallized copper sulphate dissolved in water and the volume made up to 500 c.c.

Both solutions are preserved separately, and are mixed in equal volumes for use. The technic of the determination is as follows: 60 c.c. of alkaline copper solution (30 c.c. Rochelle salt solution, 30 c.c. copper sulphate solution) are placed in a beaker holding about 300 c.c., diluted with 60 c.c. of water, and then heated to boiling over the free flame or on a sand bath. Twenty-five cubic centimeters of urine are then added to the actively boiling fluid. The urine must not, of course, contain more than 1 per cent. of sugar; otherwise it must be diluted according to the rules given on p. 507 *et seq.* The boiling is allowed to continue for two minutes. The precipitated red oxid is then immediately filtered off. The asbestos suction filter recommended by Soxhlet is the best for this purpose. It consists of a small tube of hard glass, 15 cm. long, one half of which has a lumen of about 2 cm. diameter, which suddenly diminishes to a lumen of about ½ cm. in the other half. The wide half is filled, perhaps, 2 cm. deep with long-fiber asbestos. A little glass-wool is first placed over the constricted portion in order to furnish the asbestos a better hold. Upon this glass-wool enough long-

[1] *Jour. f. praktische Chemie*, Neue Folge, vol. xxii., 1880, p. 52.

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fiber asbestos is poured in to make the filter sufficiently thick, then a thoroughly shaken emulsion of short-fiber asbestos is added. If the precipitate escapes through the filter, the layer of asbestos can be made sufficiently compact by pressing it down with a glass rod. We must first wash the asbestos with pure dilute hydrochloric acid, and then wash it free from the chlorin. To pour the fluid in more conveniently, a small funnel can be inserted into the wide end of the filter tube with the aid of a perforated rubber stopper. The constricted portion is placed vertically in the perforated cork of a suction bottle, and the latter connected with a suction pump. Before using the asbestos filter, it must always be dried at 120° C., cooled in the exsiccator, and then weighed. The fluid can then be rapidly sucked through. After the cuprous oxid has been removed to the asbestos filter, alcohol is allowed to flow through, and, then the filter is dried in an air bath at 120° C. for about one-half hour. By means of its perforated rubber stopper and a small glass tube, the filter is next connected with the rubber tube of a Kipp hydrogen apparatus, and hydrogen is passed through. The hydrogen must first have been thoroughly purified by passing it through concentrated sulphuric acid and then through a potassium permanganate solution. While the hydrogen is being passed through, the filter tube should be so placed that the narrow half is a little lower than the wide one, so that the heavier air can be more readily and completely displaced by the hydrogen. After a few minutes a test can be made to see whether the hydrogen comes through the filter free from air. For this purpose a narrow test tube is held vertically over the opening for a few moments, its mouth carefully closed with the thumb, and the contained hydrogen then ignited by holding the mouth of the tube near a flame. If all the air has been displaced from the apparatus, the hydrogen will explode with but a slight noise, and afterward a small blue flame, hardly visible by daylight, will travel slowly and gently to the bottom of the tube. But, on the other hand, if the hydrogen which escapes from the filter still contains air, the flame will immediately shoot into the tube with a loud whistling noise, and then instantly die out. In such an event hydrogen must be passed through until the last vestige of air has been removed from the tube, so that all danger of explosion from the subsequent heating of the asbestos may be entirely avoided. As soon as we are sure of this freedom from air, the asbestos end of the tube is fastened over a gas flame, and kept at a red heat in the current of glowing hydrogen until all of the *brick-red* cuprous oxid has become reduced—*i. e.*, until the precipitate upon the filter has changed to a characteristic *brown red*—the color of metallic copper. The completion of the reduction may also be recognized by the fact that no more tiny drops of water form at the cold end of the tube, and that when the hydrogen is ignited at the small end of the tube it no longer burns with an increasing flame. The tube is now allowed to cool in the current of hydrogen, and is then set in the exsiccator until weighed. The weight of the reduced copper can then be readily determined. The empirical table (next page) compiled by Allihn shows at a glance the amount of sugar corresponding to a given weight of the copper.

If Allihn's directions are adhered to exactly, and particularly if the filtration is performed immediately after *two minutes'* boiling of the fluid, these figures are accurate.

S. Pflüger has carefully criticized Soxhlet-Allihn's method. His article (weighing the cuprous oxid found) also includes his improvement[1] upon the cuprous oxid method, Prager's method[2] (transforming the cuprous oxid to cupric oxid and then weighing), and Volhard's estimation of cuprous oxid as cyanid. Pflüger recommends an appropriate modification of the asbestos filter. (See p. 439 of the first-mentioned article.)

Ambühl[3] has more recently demonstrated that a more practical method is to omit the reduction of the cuprous oxid to copper, which Allihn recommends, and to wash the cuprous oxid immediately with hot water, alcohol, and ether, and then to weigh it after having dried it for an hour at 98° C. to a constant weight. From

[1] *Arch. f. d. gesammte Physiol.*, vol. lxix., 1898. [2] *Cf. Ibid.*, vol. lxvi., 1897.
[3] *Chemiker-Zeitung*, vol. xxi., I. Sem, p. 137.

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this the corresponding amount of sugar can be calculated. According to Ambühl's table, the difference in the amount of sugar found by his method and that of Allihn's ordinarily amounts to less than 0.1 per cent. Multiplying the amount of cuprous oxid (Ambühl's method) by 0.888 will give the corresponding weight of copper, and then Allihn's table can be employed to estimate the corresponding amount of sugar.

Copper (milligrams).	Dextrose (milligrams).	Copper (milligrams).	Dextrose (milligrams).
10	6.1	250	129.2
20	11.0	260	134.6
30	16.0	270	140.0
40	20.9	280	144.5
50	25.9	290	151.0
60	30.8	300	156.5
70	35.8	310	162.0
80	40.8	320	167.5
90	45.9	330	173.1
100	50.9	340	178.7
110	50.0	350	184.3
120	61.1	360	190.0
130	66.2	370	195.7
140	71.3	380	201.4
150	76.5	390	207.1
160	81.7	400	212.9
170	86.9	410	218.7
180	92.1	420	224.5
190	97.3	430	230.4
200	102.6	440	236.3
210	107.7	450	242.2
220	113.2	460	248.1
230	118.5	463	249.9
240	123.9		

COLORIMETRIC ESTIMATION OF DEXTROSE.

Because of the blue color peculiar to combinations of dextrose in alkaline solution with copper, and the blue color of copper combinations in general, the writer has made an effort to utilize this color in one way or another for the purpose of estimating colorimetrically the quantity of dextrose in the urine. He has conducted numerous experiments, and will briefly report the results.

A conceivable method is to add to a constant amount of sodium hydroxid a constant amount of copper sulphate solution, which is so chosen that the copper is always in excess—*i. e.*, when, subsequently, sugar containing urine is added, the copper cannot be held entirely in solution, but is sufficient, in spite of the sugar, to cause a precipitate of cupric hydroxid. Urine, suitably diluted, if necessary, is added in measured quantities, and the precipitate filtered from the deep-blue solution by means of an asbestos filter. The intensity of the color is determined colorimetrically and the quantity of the sugar estimated in this way. The solution may be diluted and compared with a test solution of copper kept in a hermetically sealed tube. It has, however, been proved that this method is unreliable. The intensity of the color proves to be quite independent of the amount of sugar added, giving the impression that only small quantities of sugar enter into the blue combination. For instance, we found that it was quite immaterial whether a 0.5 or a 1 per cent. solution of sugar was added to the constant quantity of sodium hydrate and copper sulphate.

Another method of colorimetric estimation proved equally unreliable. Copper sulphate solution is added drop by drop from a buret to a solution containing equal parts of sodium hydroxid and of the urine to be examined until the excess of copper commenced to separate as cupric hydroxid. The shade of blue is estimated colorimetrically, but, just as before, the intensity of the color appears to be quite independent of the amount of sugar. In these experiments a 0.5 and 1 per cent. sugar solutions hold in solution about an equal amount of copper oxid. For

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this reason one may not draw conclusions as to the quantity of sugar contained in the urine from the volume of copper sulphate solution necessary to produce cloudiness.

One might endeavor to utilize the reduction test colorimetrically. After performing the reduction (Soxhlet-Allihn p. 510) and filtering off the suboxid the quantity of non-reduced copper in the filtrate might be estimated colorimetrically instead of iodometrically. Even this method is not practical, for we found that the color-intensity of Fehling's solution with constant quantities of alkali and Rochelle salt does not run parallel with the amount of copper contained.

However, the following method of utilizing the reduction test colorimetrically gives approximate results. The test is the same as Soxhlet-Allihn's, up to the filtering off of the reduced suboxid, for iodometric estimation. The cuprous oxid or the reduced copper is not determined by weighing, but nitric acid is poured on the asbestos filter until the red oxid is dissolved. Water is next poured over the filter, and the quantity of copper contained in the solution estimated colorimetrically by comparing with a copper solution of known strength. We began by using a solution of copper nitrate, so as to have exactly comparable conditions. We found, however, that its color was exactly that of a copper sulphate solution containing an equal amount of copper, and so have since used the copper sulphate solution utilized in preparing Fehling's solution.[1] In two graduated cylinders of equal diameter are placed respectively some copper sulphate solution and an equal volume of the nitric acid solution of cuprous oxid. Water is then added to the darker of the two solutions until the shade of color of the two is the same. The amount of copper contained in the two solutions is then proportional to their volumes, so that it becomes possible to estimate not only the amount of copper reduced, but also the amount of sugar in the urine, inasmuch as we know just how much dextrose corresponds to each cubic centimeter of the copper sulphate solution. This method of employing graduated cylinders only, and without accurate colorimetric apparatus, gives only approximate values, which are far less accurate than the results obtained by weighing the copper or cuprous oxid or by iodometric titration. The practising physician, however, who is not familar with the technic of the more accurate methods, and who desires a method more rapid than the fermentation test, may obtain with this method fairly satisfactory approximate results. By using accurate colorimetric apparatus, the correctness of the values obtained is much greater.

QUANTITATIVE FERMENTATION TESTS FOR DEXTROSE.

Upon the addition of yeast, dextrose possesses the property of fermentation—*i. e.*, of splitting into alcohol and carbon dioxid. Three tests based upon three different results of this property have been devised to determine the quantity of sugar in urine.

Robert's Quantitative Areometric or Densimetric Test.—This is based upon the fact that fermentation considerably diminishes the high specific gravity of sugar containing urine. From this difference in specific gravity before and after fermentation the percentage of sugar may be determined.

The technic of the test is as follows: The specific gravity of the diabetic urine is estimated in the ordinary way. A piece of compressed yeast (not containing sugar, or, if so, washed out with water) the size of a hazelnut is then added to a definite amount of urine (about 100 c.c. are sufficient for testing the specific gravity readily). The mixture

[1] This is explained by the assumption that the color is dependent only on the copper ion which is present in equal quantities, or, at least, in equal dissociation in both solutions.

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is shaken gently, loosely covered with a piece of paper or an inverted beaker, and allowed to remain at the room temperature for twenty-four to thirty-six hours. When fermentation is complete, the yeast will settle to the bottom, the foam will no longer form and the fluid will become clear above. To be perfectly sure that the fermentation is completed, Trommer's test can be employed. The specific gravity of the completely fermented urine is then determined. If the yeast which has settled to the bottom is stirred up, the urine must first be filtered. If the difference between the two specific-gravity estimations (before and after fermentation) is multiplied by the empirical number 0.230, the product equals the percentage of the sugar contained in the urine. Comparative estimation with other methods has shown that a difference in density of B corresponds to a sugar content of 0.230 per cent.; consequently a difference in density of D will correspond to a sugar-content of D $\times$ 0.230 per cent. If a very accurate estimation is necessary, we must determine the temperature of the fluid before and after fermentation, and if they are not equal must make a correction. If the temperature is higher after fermentation than before, one-third of a degree of the urinometer scale must be added to the specific gravity determined after fermentation for each degree of temperature. If the temperature after is lower than before fermentation a similar deduction must be made. The urinometer should be very accurate, with subdivisions from 1000 to 1050, and sufficiently wide apart for careful reading; the urinometer glass of good size, so that the amount of urine is considerable. An excellent plan is to employ one urinometer for densities between 1000 to 1025, and a second for those between 1025 to 1050. Many authors have proved that this method is sufficiently accurate for clinical purposes (up to 0.1 per cent.), although it has been often questioned,[1] and certainly it is so simple as to be very practical for the ordinary physician.

The diminution in specific gravity can be determined directly without the use of an urinometer by weighing a measured quantity of urine upon an ordinary apothecary's hand-scale.

Quantitative Gas-volumetric Fermentation Test.—This method is based upon the fact that the amount of sugar in urine can be computed by measuring the volume of the carbon dioxid which is evolved by fermentation. To obtain useful results with this method, the volume of the carbon dioxid produced in the eudiometer tube must be read off with all the precautions necessary in gas-volumetric analyses—*i. e.* with consideration of the barometric pressure, the temperature, the tension of the water vapor, the weight of the fluid shutting off the gas in the eudiometer tube, etc. The gas, too, must be measured enclosed in mercury, on account of the solubility of carbonic acid in water. The so-called idiopathic fermentation of the yeast renders the accuracy very questionable, and, in any event, the process is too complicated for the ordinary practitioner. Einhorn's attempt to simplify the method for the practising physician the author regards as a failure.

[We must disagree with the author in this statement. The results obtained in this country with Einhorn's saccharometer have been found

[1] *Pflüger's Archiv*, vol. xxxiii., 1884, p. 211; vol. xxxvii., 1885, p. 479. Lohnstein, *Berlin. klin. Woch.*, 1896, No. 6, p. 120.

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to be sufficiently accurate to warrant the widespread and general use of the apparatus. Of course, when the fermentation method is employed with any modification, only relative, and not absolute, amounts can be expected. The introduction of physical details into the procedure is an approach at accuracy not warranted, on account of the basic inaccuracy of the general method.

Einhorn's saccharometer is shown in Fig. 175. A determination with this instrument is performed as follows: One gram of yeast ($\frac{1}{16}$ of the ordinary cake of compressed yeast is about equal to a gram) is carefully shaken with 10 c.c. of the urine to be examined, and the mixture is poured into the bulb and upright tube, completely filling the latter. No bubbles of air must be allowed to remain at the top of this tube, nor should any collect there within the first few minutes after the filling of the apparatus. It is always advisable to make a control test with normal urine or with water. The apparatus is then allowed to stand at 30° C. for twelve hours, after which a reading on the graduation must be made. The scale is calibrated directly into percentages of sugar (dextrose) in the urine. It must be emphasized that the total volume of the twenty-four-hour excretion of urine must be taken into consideration in order to calculate the *quantity* of sugar eliminated *in the twenty-four hours*. Percentage results are of no value as aids to diagnosis.—H. C. J.]

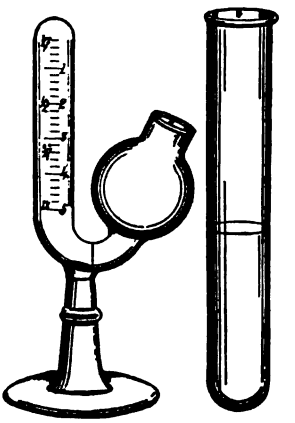

FIG. 175.—Einhorn's saccharometer.

Lohnstein's Accurate Fermentation Saccharometer.[1]—Lohnstein's Accurate Fermentation Saccharometer is constructed upon manometric principles, measuring the pressure produced by the development of the carbonic acid. Its only disadvantage is that one of its parts cannot be regulated by the experimenter, who is consequently dependent upon the maker of the instrument. The writer will omit a description of the saccharometer. It is easily used, and is praised by many authors as an accurate instrument.

The presence of proteid in the urine does not in any way disturb the technic or accuracy of the fermentation tests. This is a great advantage, because with most other methods of determining the amount of sugar the urine must be first freed of proteid.

Compressed yeast is employed so universally now throughout America that it seems hardly necessary to suggest any substitute.

POLARIMETRIC ESTIMATION OF DEXTROSE.

The polarimetric estimation of dextrose is probably the most convenient of all. It does, however, require a rather expensive apparatus, and it fails to estimate very small quantities. Almost any of the polariscopes are good enough, but the author especially recommends Wild's polaristrobometer. The principle, which

[1] *Allg. med. Centralzeit.*, 1899, No. 101, 1900, No. 30, *ff.*

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is the same with all polariscopes, depends on the fact that a solution of dextrose has the power to turn the plane of polarized light to the right. The angle of deflection is proportionate to the amount of sugar in the solution. It is known that when a ray of polarized light is taken up by an analyzing Nicol prism, the portion of light which passes through the analyzing Nicol varies decidedly according to the position of the Nicol relative to the plane of vibration of the polarized ray of light. If the plane of vibration of the analyzing Nicol is parallel to that of the polarized ray of light, the maximum amount of light will be transmitted; if at right angles to it, the minimum of light. If the position of the analyzing Nicol is now determined for this maximum or minimum, then the interposition of an optically active substance—*e. g.*, a solution of dextrose—will rotate the plane of the polarization of the ray of light with relation to the analyzing Nicol. Then to determine again a maximum or minimum of light intensity, the Nicol itself must be rotated through a certain angle. The size of this angle will be proportional to the amount of the interposed substance—*e. g.*, sugar; this amount can therefore be calculated from the angle through which it is necessary to rotate the Nicol to produce the maximum or minimum of light. All polariscopes constructed for quantitative analysis depend upon this principle. Their only differences depend upon the employment of various optical contrivances in order to facilitate the determination of the position of the planes of vibrations to each other—*e. g.*, the interposition of doubly refracting bodies, peculiarly ground plates of quartz, etc. They produce peculiar optical appearances, according to the position of the Nicol, colors, stripes, etc.

Wild's instrument uses, as an indicator of the position of the planes of vibration, the system of parallel dark bands which, in a homogeneous polarized light (sodium flame), are made by two crossed quartz plates cut at an angle of 45° to the axis. The advantage of homogeneous light and parallel bands without color, which appear simply dark upon a light background, is that the test depends upon the examiner's sensibility to light alone, and not to color. The test with other instruments—*e. g.*, Soleil-Ventzke's—depends upon the sensibility of the eyes to color. Fig. 176 shows Wild's instrument. The analyzing Nicol prism and the quartz plates are set in the tube *a c*, and the polarizing Nicol prism in the tube *d e*. In looking through the instrument, in a dark room, from *a* to the sodium flame at *b*, with the Nicols parallel and crossed, the parallel bands will appear upon the bright background of the field of vision (Fig. 176, IV.). If one of the Nicols is rotated 45° by means of the screw *f*, the bands will disappear; further rotation will bring them back.

It should be noted that Pfister and Streit, of Bern, are now supplying Wild's instrument with a number of marked improvements which increase its accuracy, and make it possible by employing the so-called half-shadow principle to use a non-homogeneous strong light, and under some circumstances to omit entirely decolorizing the urine.

To estimate the amount of dextrose by polarization, 50 c.c. of urine are decolorized by shaking with about $\frac{1}{6}$ of this volume of the best animal charcoal and then filtering. If the urine is dark-colored it must sometimes be allowed to stand for several hours with the animal charcoal, during which time it is frequently shaken; or it may be necessary to boil the urine containing the charcoal for a brief period. The decolorization should be complete. As Späth fears that some of the dextrose may be absorbed by the charcoal, he recommends the following method of Patéin and Dufau for decolorization of the urine: A solution of mercuric nitrate is prepared by dissolving 200 gm. of acid nitrate of mercury in 500 to 600 gm. of water, and sodium hydroxid is added until a slight precipitate results. Sufficient water is added to bring the quantity up to 1 liter, and the mixture is filtered. Small quantities of this solution are added to 50 c.c. of urine until no further precipitation occurs; a dilute solution of sodium hydroxid is added drop by drop until the reaction of the mixture is neutral or, at most, feebly alkaline; the quantity is brought up to 100 c.c. by adding sufficient water; and the mixture is filtered. No precipitate should be produced by the addition of sodium hydroxid to the filtrate.

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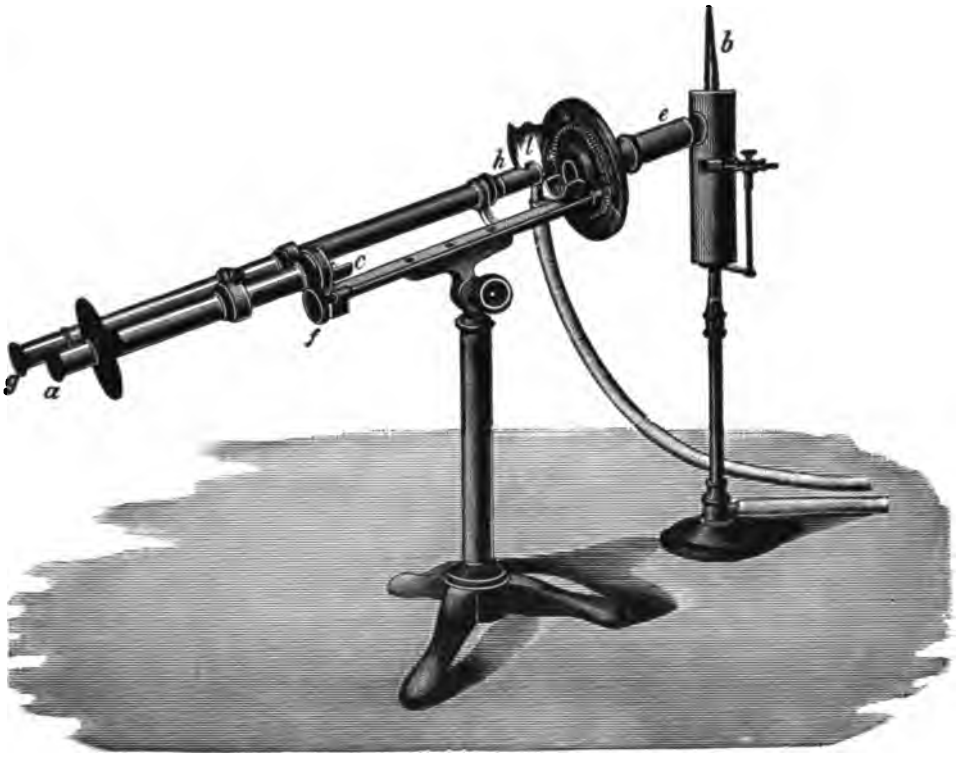
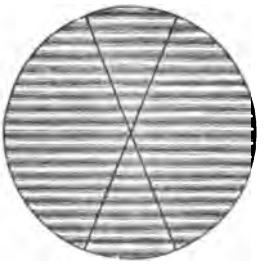


I.

II.



IV.

III.

Fig. 176.—Wild's polaristrobometer.

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A metal tube (200 mm. long) (Fig. 176, II.), which can be closed at both ends with parallel plain glass disks and metal caps, is filled with the decolorized urine. We must be careful not to include any air bubbles, and not to press the glass plates by screwing the metal caps too hard, because under pressure the glass becomes doubly refractive, and the dark bands will not disappear with any position of the Nicol. When so arranged the tube will contain a layer of urine exactly 200 mm. thick. The instrument is adjusted with a sodium flame in a dark room so that the parallel bands absolutely disappear, and the tube which contains the urine is then set upon the rest *c*, *d*. If the urine contains sugar the bands will immediately reappear. They are then made to disappear by rotating the polarization plane with the polarizing Nicol by means of the screw *f*. At the moment when they disappear, the size of the angle through which the Nicol prism has been rotated is read off through the telescope *g h*, upon the scale *i*, by means of the vernier *k*. The gas flame at *l* serves to illuminate the scale. When the amount of sugar contained is very great, the tube III. is substituted for tube II. In the latter, one of the parallel glass disks is placed at *M*, so that the thickness of the urine is *M m*—*i. e.*, 100 mm. The part *M o* is simply an empty continuation of the tube necessary for its adjustment. The angle of rotation is read off and the amount of sugar in tenths of 1 per cent. calculated directly from the following table:

Angle of rotation.	Thickness of urine layer, 100 mm. (length of tube).	Thickness of urine layer, 200 mm. (length of tube).
1 degrees	1.984 per cent. dextrose	.992 per cent. dextrose.
2 "	3.968 " "	1.982 " "
3 "	5.952 " "	2.976 " "
4 "	7.936 " "	3.968 " "
5 "	9.920 " "	4.960 " "
6 "	11.904 " "	5.952 " "
7 "	13.888 " "	6.944 " "
8 "	15.872 " "	7.936 " "
9 "	17.856 " "	8.928 " "
10 "	19.840 " "	9.920 " "

The scale of the instrument is not graduated in minutes and seconds, but in tenths of degrees. Hence, the figures in the above table indicating the amounts of sugar corresponding to each degree can be used directly to express the amounts of sugar corresponding to tenths of degrees by simply moving the decimal point one place to the left. If, for instance, 3° rotation with a layer 100 mm. thick corresponds to 5.952 per cent. of dextrose, then 0.3° will correspond to .5952 per cent. The calculation then becomes extremely easy—*e. g.*, if the rotation equals 3.2°,

$$\begin{aligned} 3.0° &= 5.952 \text{ per cent.} \\ 0.2° &= .3968 \text{ "} \\ \hline 3.2° &= 6.3488 \text{ per cent.} \end{aligned}$$

The polarimetric estimation of the percentage of sugar can be, therefore, very speedily performed.

Proteids, contrary to dextrose, are levorotatory; hence, to obtain correct results with albuminous diabetic urine (see p. 463 *et seq.*), the proteid must first be removed. Polarimetric estimation of sugar is accurate to about 0.5 per cent., which suffices for all practical purposes, although results by means of the areometric fermentation test have a still narrower limit of error. With the polarimetric method, mistakes are apt to occur, due to the occurrence in the urine of levogyrate substances (levulose, combined glycuronates, and β-oxybutyric acid).

QUANTITATIVE DETERMINATION OF UREA.

Normal human urine contains up to 4 per cent. of urea. The average amount of urea excreted in twenty-four hours[1] for a man on a mixed diet is about 33 gm., varying between 24 and 40, with starva-

[1] Vierordt, *Daten und Tabellen*, 1888.

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tion and non-nitrogenous diet, 15 to 20 gm. Women excrete from 20 to 32 gm. Upon a very rich proteid diet even as much as 100 gm. has been excreted. Quantitative estimations of urea are most essential in all metabolic investigations.

The urea estimation may also be used to test the capability of complete digestion—*e. g.*, a very rich proteid diet is ingested for one day by an individual in a condition of approximate nitrogenous equilibrium. We then estimate the total amount of urea for that day and the day following, and determine whether it is excreted in an amount proportionate to the ingested food. By far the greater part of the nitrogen introduced in the form of proteid is eliminated as urea, and at most only a small amount of proteid will be stored up in one day; hence, nearly the whole of the ingested nitrogen must appear as urea in the urine of the particular day, and perhaps of the following, provided proteid food has been well utilized.

Following the suggestion of F. Hirschfeld, a patient ingests during one day a test diet of 500 gm. of meat (106.25 gm. of proteid), 8 eggs (48 gm. of proteid), and 200 gm. of bread (18 gm. of proteid), or a total of 172.25 gm. of proteid. If the proteid is well utilized the patient will excrete at least 59 gm. of urea (27.5 gm. of nitrogen). If he does not excrete as much as that the utilization is incomplete. Hirschfeld considers such a condition of insufficient utilization characteristic of certain cases of diabetes mellitus, and is inclined to attribute it to some functional disturbance of the pancreas. As a consequence of the slight elimination of urea, such cases are usually supposed not to present any increase in the quantity of urine.

ESTIMATION OF THE AMOUNT OF UREA BY MEANS OF THE SPECIFIC GRAVITY OF THE URINE.

Urea affects the specific gravity of urine more than any other constituent; hence, the specific gravity of the urine will furnish an approximate measure of the amount of urea, provided, of course, that sugar is absent. Experience has shown that a specific gravity of 1014 corresponds to about 1 per cent. of urea; of 1014 to 1020, to about 1.5 per cent.; of 1020 to 1024 to about 2.25 per cent.; of 1028, to about 3 per cent. This estimate must be modified in fever and in cachexia, in both of which the chlorid excretion is diminished.

If sugar is present, it must first be removed by fermentation before we can judge of the amount of urea from the specific gravity. In such an event it is better at the same time to employ the areometric method for the estimation of sugar (p. 513 *et seq.*), and then to recalculate the specific gravity found after fermentation, since the alcohol lowers this to some extent. The degree in which the specific gravity is thus lowered can be best determined by calculating the amount of alcohol from the result of the areometric estimation of sugar. Remembering that approximately equal amounts by weight of alcohol and carbon dioxid are produced in the fermentation of dextrose, and having already determined the percentage of dextrose, we can easily get the amount of alcohol in the fermented urine, for it will equal about half the amount of sugar found—*e. g.*, after fermentation, approximately a 1.5 per cent. solution of alcohol will result from a diabetic urine containing 3 per cent. of dextrose. According to Hirschfeld[1] the following specific gravities apply to watery solutions of alcohol at 15° C. (water = 1000): 1 per cent., 998.5; 2 per cent., 997; 3 per cent., 995.6; 4 per cent., 994.2. The specific gravity of the urine is thus diminished about 1.5 for every per cent. of alcohol in solution. Therefore the specific gravity of the fermented urine must be increased by 1.5 for every per cent. of alcohol contained before we can estimate the urea by the above method.

[1] *Zeits. f. klin. Med.*, 1891, vol. xix., p. 338.

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LIEBIG'S METHOD FOR UREA BY TITRATION.

Liebig precipitates the urea, in the form of urea mercuric nitrate, by means of a solution of mercuric nitrate. In its original form the method is very simple and convenient, but unless performed very carefully many errors may arise, and even with the greatest care a number of corrections should be made. On account of these difficulties several modifications have been suggested, all of which furnish better results, but which are more complicated and require more technical skill than the original process. As a matter of fact, moreover, Liebig's method has been abandoned because it does not estimate the amount of urea in the urine, but rather the approximate amount of total nitrogen (see p. 526).

FIG. 177.—Hüfner's apparatus for estimation of urea.

KNOP-HÜFNER'S METHOD OF ESTIMATING THE UREA.

This is not only the simplest, but also the most accurate, of all methods. It is to be recommended for clinical purposes. It depends upon the decomposition of urea into carbon dioxid, water, and nitrogen by means of a solution of sodium hypobromite in an excess of sodium hydroxid. The nitrogen liberated from a definite amount of urine is measured volumetrically, and from this the corresponding amount of urea is calculated. The carbon dioxid is absorbed by the excess of sodium hydroxid. For very accurate analysis the barometer and temperature must be considered. Other nitrogenous constituents of the urine (uric acid, kreatin, etc.) are partially broken up at the same time, liberating nitrogen; hence, the method is not absolutely accurate. Nevertheless, if the proteid is first removed, these other constituents will not add much to the figures for urea.

Hüfner's apparatus, represented in Fig. 177, has been modified again and again to more or less advantage. It consists of three separate parts:

1. The dilated glass receptacle, *C*, connected by means of a ground-glass stopper with a smaller one, *D*, which holds from 6 to 7 c.c.

2. The bell-shaped glass vessel, *B*, open at both ends, and connected with the upper end of *C* by means of a perforated hard-rubber stopper.

3. The eudiometer tube, *A*, divided into centimeters and fractions, to be placed when in use within vessel *B* over the upper end of *C*.

Preparation of the sodium hypobromite solution: 70 c.c. of a 30 per cent. sodium hydroxid solution are mixed with 180 c.c. of water and 5 c.c. of bromin. The latter dissolves with the formation of sodium bromid and sodium hypobromite.

$$2\,NaOH + Br_2 = NaBrO + H_2O + NaBr.$$

As soon as the bromin has dissolved, the reagent is ready for use. It should

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be freshly prepared every day or two, because the hypobromite gradually becomes decomposed. It should also be kept in a cool, dark place—best of all in an ice-chest—and never employed when warm from mixing.[1]

Technic of the Method.—The amount of urea in the urine to be examined is first approximately estimated by the specific gravity (p. 519 *et seq.*), and then, if necessary, the urine is diluted so that the amount of urea present is not above 1 per cent. Five cubic centimeters of the urine, or of a diluted portion (when necessary), are removed in a long, thin pipet and allowed to run into the vessel *D*, carefully avoiding wetting the sides of *C*. In the same pipet is then sucked up 1 to 2 c.c. of water, in order to remove any urine adhering to its sides. The receptacle *D* is then filled with the contents of the pipet exactly to the upper margin of the bore in the glass stopcock. This is now shut off. Then the vessel *C* is filled to overflowing with the hypobromite solution, and next the vessel *B*, as well as the eudiometer tube, is filled with concentrated sodium chlorid. The tube is inverted over the opening of the vessel *C* under the level of the liquid in *B*, its opening having been first closed with the thumb, and the inclusion of air avoided. *A* is now held in position by the retort stand, which also supports the other parts. Now, as soon as the stopcock is opened, the heavy hypobromite solution in *C* becomes mixed with the lighter urine in *D*, and nitrogen develops actively. This gas accumulates in the eudiometer tube, gradually displacing the salt solution. The decomposition is complete in from twenty minutes to one-half hour. The bubbles adhering to the sides of the vessel are then gently shaken into the eudiometer tube, which is closed under the level of the salt solution with the thumb. It is then removed, and submerged as completely as possible, opened downward, in a large cylinder filled with water at the room temperature. After about fifteen minutes the eudiometer tube and its contents will be at the same temperature as the surrounding water. The tube is then lifted far enough out of the water to equalize the levels of the fluid inside and outside, and at this moment the volume of the enclosed nitrogen is read off. The temperature of the water and the barometric pressure should also be recorded. The volume of gas estimated must now, for the purpose of calculation, be reduced to 0° C., 760 mm. barometric pressure, and absolute dryness, according to the formula:

$$v' = \frac{760\,(1 + 0.00366\,t)}{v\,(b - w)}.$$

v' = Reduced volume desired.
v = Volume read off.
b = Barometric pressure at the time of reading, in mm. Hg.
w = Tension of water vapor at a temperature t, in mm. Hg.
t = Temperature of water at time of reading.
0.00366 = Coefficient of expansion of gases for 1° C.

The values for w corresponding to the temperatures usually met with are as follows:

10° C.	9.126	18° C.	15.351
11° C.	9.751	19° C.	16.345
12° C.	10.421	20° C.	17.396
13° C.	11.130	21° C.	18.505
14° C.	11.882	22° C.	19.675
15° C.	12.677	23° C.	20.909
16° C.	13.519	24° C.	22.211
17° C.	14.009	25° C.	23.582

[1] [The method of preparation of the hypobromite solution as outlined by Rice can be highly recommended. Two solutions are necessary, and may be retained for any ordinary length of time.

1. A solution of 125 gm. of bromin and 125 gm. of sodium bromid in sufficient water to make the volume up to 1000 c.c.

2. A solution of sodium hydroxid of a specific gravity of 1250 (22.5 per cent.). To prepare the hypobromite for use, mix equal volumes of the two solutions, and dilute with 1½ volumes of water.—ED.]

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One gram of urea furnishes 354.3 c.c. of nitrogen (a little less than the theoretic quantity); hence, the following proportion will determine the amount of urea (x) in the 5 c.c. of urine employed.

$$354.3 : 1 = v' : x$$
$$x = \frac{v'}{354.3} \text{ grams.}$$

The percentage of urea contained in the urine can be very easily determined by multiplying this number by 20. Of course, if the urine was diluted before the estimation, this dilution must also be considered.

The author considers that Gerrard's[1] improvement upon Hüfner's apparatus has been shown to be the most practical for the practitioner (Fig. 178).

A graduated glass tube, *a*, fixed in a wooden stand, is connected at the lower

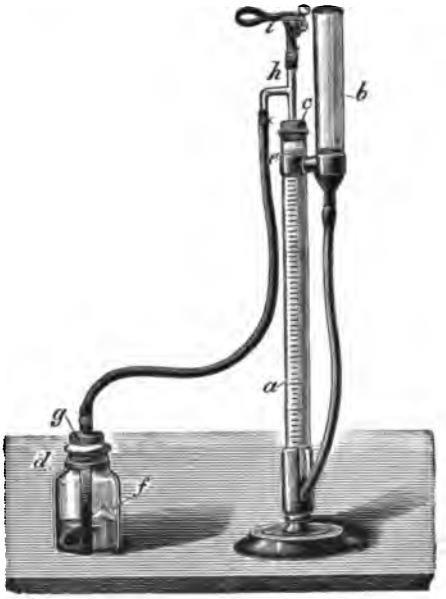

FIG. 178.—Gerrard's apparatus for estimation of urea.

FIG. 179.—Dupré's apparatus for estimating urea.

end by means of a side projection and a rubber tube with a vessel, *b*, which is open at the top, and at its upper end with a wide-necked bottle, *d*, by means of a perforated rubber stopper, *c*, and a rubber tube. Vessel *b* may be raised or lowered upon the graduated tube by the metal spring clamp *e*. The wide-necked bottle, *d*, is filled with 100 c.c. of sodium hypobromite solution; 5 c.c. of urine are poured into the small test tube, *f*, which is carefully placed in bottle *d*, so as to be, for the time being, protected from the hypobromite solution. The bottle is then con-

[1] *Lancet*, 1884, ii. p. 952.

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nected with the rest of the apparatus by means of the perforated rubber stopper *g* and tube *gh*. Next, the stopper *c* is removed and vessel *a* is filled with water up to the zero mark of the scale. Of course, the communicating vessel *b*, which has been pushed up toward the top, also fills to the same level. The stopper *c* is now replaced. While this is being done the spring clamp *i*, which closes the rubber tube attached to the glass tube *h*, is opened carefully so that at the moment of replacing *c* there will be no increase of pressure of the air upon the level of the water. The fluid therefore remains at the same level in the communicating vessels *a* and *b*. The spring clamp *i* is now very carefully closed again. After the apparatus has been connected in this fashion, the urine in the little glass *f* is easily poured out into the bromin solution by repeatedly tipping the vessel *d*. The development of gas follows immediately. Bottle *d* is placed in a large vessel of water at the room temperature (15° C.) while the development of gas continues. To make it sink, the bottle is furnished with a lead framework, as shown in the picture. The object of having the gas develop under water, at the temperature of the room, is to distribute the heat which arises as the result of the chemical reaction of the hypobromite solution. This heat will not only influence the decomposition, but will also increase the volume of the liberated nitrogen gas. We need not correct the gas volume, since both the gas and the apparatus may be considered to be at the room temperature of 15° C. The nitrogen set free accumulates in the upper part of the graduated tube *a*, gradually elevating the level of the water in *b*. The evolution of the gas may be materially hastened by gently shaking the bottle *d*. When the decomposition is complete, the gas can be put under ordinary atmospheric pressure by lowering vessel *b* sufficiently to equalize the level of the fluid in *a* and *b* again. Then the volume of the nitrogen is read off.

The scale upon the English instrument indicates empirically the amount of urea at room temperature and normal barometric pressure (760 mm.). Mariott's law can then be made use of to correct the reading according to the barometric pressure—viz.:

$$\frac{v}{v'} = \frac{b'}{b}$$

V and *b* and *v'* and *b'* represent the corresponding gas volumes and barometric pressures. *V* and *v'* may be directly replaced by the amount of urea proportionate to them, so that the formula then becomes, when *h* and *b* represent the amounts of urea corresponding to the barometric pressures,

$$\frac{h}{h'} = \frac{b}{b'}$$

It is advisable to control the empirical scale by adding a scale in cubic centimeters on tube *a*. Then the calculation for any temperature or barometric pressure can be made in the same manner as with Hüfner's apparatus (see p. 521). The same rules for dilution, of course, apply for this apparatus as for Hüfner's.

Not only is Gerrard's instrument more compact than Hüfner's, but the gas can be entirely evolved within a few moments by shaking bottle *d*.

The weak point of the apparatus is the closure of the upper end by a stopcock, which may easily allow of the escape of gas, and consequently give rise to an erroneous result. Dupré's modification of Hüfner's apparatus (Fig. 179) can be thoroughly recommended, and is the one which is employed in the author's clinic. The illustration should be sufficient without further description. It differs from Gerrard's apparatus in that the reduction of the gas to atmospheric pressure is accomplished by moving the buret in which the gas collects, and which is suspended in a cylinder filled with water. In this instrument the stopcock at the upper end of the buret should be carefully tested, in order to avoid losing any of the gas.

[Two modifications of the method of Knop, which depends upon the decomposition of the urea by sodium hypobromite or hypochlorite, have received especial

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attention in this country, and appear to be sufficiently accurate for relative values, so that they have been almost universally employed in this determination. The simplest procedure is that which makes use of the apparatus of Doremus (Fig. 180), or the modification of this by Hinds (Fig. 181).

The *Doremus ureometer*, as seen in the figure, consists of a bulb with an upright graduated tube, which can be obtained so as to rest upon a stand. The graduation reads in grams of urea per cubic centimeters of urine. When a determination is to be made, the upright tube and about one-half to two-thirds of the bulb are filled with the hypobromite solution (for preparation see above, p. 521). Care must be observed that no air remains at the top of the upright tube. The 1 c.c. pipet, which is provided with a small nipple, is filled with urine to the mark (1 c.c.), and the tip carefully introduced into the bulb and as far into the bend as it will go. The urine in the pipet is then completely delivered, but caution must be observed not to expel any air with it. The pipet is then withdrawn, and after the evolution of the gas has stopped (fifteen to twenty minutes) the meniscus of the solution is read off upon the scale. This value multiplied by the

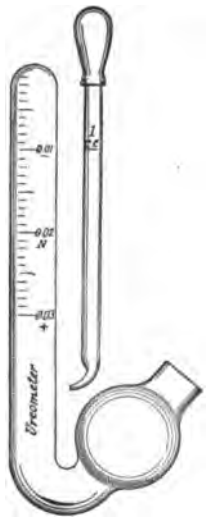

FIG. 180.—Doremus ureometer.

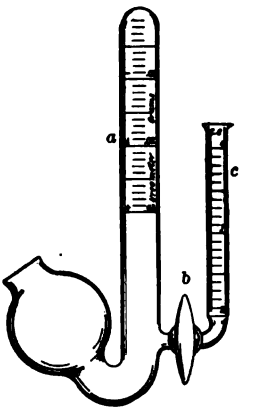

FIG. 181.—Hinds' modification of the Doremus ureometer.

number of cubic centimeters in the twenty-four hours will give the daily excretion of urea in grams.

The directions for using the *Hind's modification* are as follows: The tube *c* and the lumen of the stopcock *b* are filled with urine to be examined. Tube *a* is then washed out thoroughly with water and filled completely with the hypobromite solution. The bulb attached to *a* should contain enough of the solution to prohibit the passage of any air back into tube *a*. A reading is then made on tube *c*, and 1 c.c. of the urine is allowed to pass into *a* by means of the stopcock *b* (if the urine is concentrated, dilute with equal volume of water). After all the bubbles of gas have collected at the top of tube *a* a reading is taken. The graduation corresponds to grams in 1 c.c. of urine.

Another apparatus is that of *Squibb*. The chemical basis of this method is the same as that of the preceding, but the manipulation is somewhat more complicated, even though accuracy is not increased (see Fig. 182). There are required two 2-ounce bottles (*a* and *b*), each of which is supplied with a well-fitting double-bored rubber stopper. One opening of the stopper for *a* is left open for the subsequent introduction of a 2 c.c. pipet, closed at one end with a nipple;

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into the other opening is put an L-shaped glass tube, to which is attached a piece of rubber tubing *c*. One opening of the stopper for *b* is supplied with a straight piece of glass tubing, and the other with an L-shaped piece, which is attached to a rubber tube. This latter tube must be so placed that the free end just clears the the bottom of the graduate cylinder *g*. This free end is also provided with a glass plug. The operation is as follows :

The bottle *b* is filled with water at room temperature, and after the rubber stopper with glass tubes has been put in place sufficient water is allow to escape from the bottle into the rubber tube, to fill the latter completely. The glass plug, *e*, is then put into position, and the bottle placed upon its side as in the figure. Into bottle *a* are put 10 to 15 c.c. of the solution of chlorinated soda or lime (for preparation see below) (the hypobromite is also employed in this apparatus, but not frequently), and the rubber stopper for *b* is screwed tightly into position after the pipet, filled with urine up the 2 c.c. mark, has been pressed into the vacant opening. The rubber tubing *c* is then connected with the straight glass tube emerging from the stopper of *b*, and the glass plug removed from the end of tube *d*. A few drops of water will flow out of the free end of the tubing,

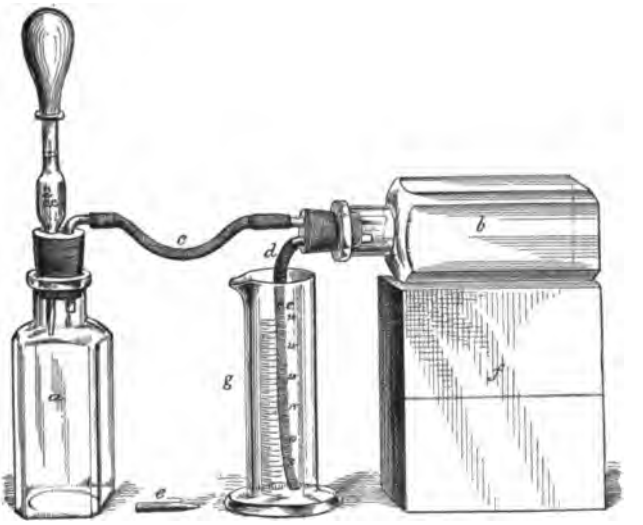

FIG. 182.—Squibb's urea apparatus for the approximate estimation of urea in urine.

but the flow must completely cease in a few seconds. This indicates that the apparatus is tight. The empty graduated cylinder must be placed under the open end of tube *d*. Finally the urine in the pipet is completely but carefully delivered. During the operation the contents of bottle *a* should be shaken from side to side in such manner that no fluid can get into the rubber tube *c*. Agitation of the contents of *a* must be continued as long as bubbles of gas come over into bottle *b*. This usually requires twenty to thirty minutes. If, however, bottle *a* is placed in a water bath at about 35 to 40° C., the reaction will be more rapid and complete in from six to eight minutes. In the latter event the bottle *a* must be cooled down to 18° C. for about five minutes.

The nitrogen gas which has been evolved during the reaction has replaced its own volume of water in the bottle *b*. This volume has escaped in the graduated cylinder *g*, and the value can be read off on the graduation and referred to the urea table (p. 521); from this, and from the volume of the twenty-four-hour sample, can be calculated the number of grams excreted in the twenty-four hours.

The solution of chlorinated soda can be prepared as follows : Shake 20 gm. of chlorinated lime (chlorid of lime, bleaching powders) in a bottle with 45 c.c. of

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water until the powder is thoroughly disintegrated. Let this settle a few minutes, and then filter off the supernatant fluid into a flask having a capacity of about 100 c.c. Wash the residue still in the bottle with 30 c.c. of water, and filter this off through the same paper into the same flask. Then dissolve 40 gm. of sodium carbonate in 30 c.c. of hot water, and add this to the solution already in the flask. Filter this mixture into a 100 c.c. graduated flask, and, after the solution has drained through, wash the precipitate with enough water to make the volume in the flask up to exactly 100 c.c. Ten to 15 c.c. of this solution are sufficient for each determination.

The solution of chlorinated lime is prepared as follows: Shake 40 gm. of chlorid of lime with 120 c.c. of water. After allowing this to settle, filter the supernatant fluid off into a 200 c.c. graduated flask. Wash the residue, shake with 80 c.c. of water, and again filter into the flask through the same filter paper. After the second portion has well drained, pour water on to the residue upon the paper until the volume of the solution in the flask is just equal to 200 c.c. Ten cubic centimeters of this solution are required for each determination. It remains active for a month.—ED.]

ESTIMATION OF UREA BY SCHÖNDORFF'S METHOD.

The principle of this method is as follows: The urine is precipitated by phosphotungstic acid containing hydrochloric acid, during which the urea remains in solution. The filtrate is then heated with phosphoric acid; ammonium phosphate is formed, and the ammonia is freed from this compound by the addition of caustic potash. The ammonia is then removed by distillation and determined by titration, as in Kjeldahl's method (see below).

As the opinions in reference to the value of this method are still very contradictory,[1] it will suffice to refer the reader to the original work of Schöndorff (*Pflüger's Archiv.*, vol. lxii); to *Jaksch Klin. Diagnostik innerer Krankheiten*, 5th ed.; to Pfaundler, *Zeits. f. physiol. Chemie*, 1900, vol. xxx., and to Folin, *ibid.*, 1901, vol. xxxii.

ESTIMATION OF THE AMOUNT OF TOTAL NITROGEN IN THE URINE BY KJELDAHL'S METHOD.

Approximate values for the amount of total nitrogen in the urine may be obtained from the estimation of the amount of urea (Knop-Hüfner's method).[2] In the calculation we must assume that 15 parts of urea contain 7 parts of nitrogen, and in this calculate the amount of nitrogen belonging to the urea present. This amount must then be multiplied empirically for normal urine by 1.136, and for fever urine by 1.18. This will approximately furnish the amount of total nitrogen excreted.

Liebig's method, the so-called urea titration, also approximately estimates the amount of total nitrogen. The figures found for the quantity of urea by this method are too high, so that, in fact, the nitrogen obtained from the calculation of the urea supposed to be present really corresponds approximately to the total nitrogen contained in the urine.

For a perfectly exact estimation of the amount of total nitrogen in the urine, Kjeldahl's method is to-day almost exclusively employed. The principle of the method is that the organic constituents of the urine are destroyed by oxidation when the urine is heated with concentrated sulphuric acid, and that the nitrogen of those substances which do not contain it combined with oxygen appears as ammonium sulphate. The urea is changed directly into carbon dioxid and ammonia. The nitrogen is estimated by liberating the ammonia from the acid solution with potassium or sodium hydroxid, distilling it off, and passing it into a measured amount of acid. The acid remaining is then titrated with an alkali.

[1] See Späth, *Die chemische u. mikroskopische Untersuchung des Harnes*, 2d ed., 1903.

[2] *Cf. Anleitung zur qualitativen und quantitativen Harnanalyse von Neubauer und Vogel*, Neubearbeitet von H. Huppert, 1890, p. 531.

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The details of the method, including certain modifications recommended by Wilfahrt and by Salkowski, are as follows: The acid used for oxidizing purposes is a mixture of 500 c. c. of concentrated sulphuric acid and 100 gm. of phosphoric anhydrid. Pure sulphuric acid may be employed, but oxidization takes longer. It is also necessary to have a $\frac{N}{2}$ oxalic acid solution prepared by diluting the normal acid with an equal amount of water. A $\frac{N}{2}$ solution of sodium hydroxid is also essential. This is prepared, according to Salkowski, in the following manner:[1] Enough water is added to 80 c. c. of sodium hydroxid solution, free from CO_2 and at 1340 specific gravity, to make the volume 1100 c. c. This is well stirred and a portion placed in a buret. 10 c. c. of the $\frac{N}{2}$ oxalic acid are placed in a beaker, and a few drops of rosolic acid or phenolphthalein solution added. The sodium hydroxid solution is added until the end reaction is reached—*i. e.*, until the mixture becomes a permanent red. The sodium hydroxid must be diluted until 10 c.c. correspond to exactly 10 c.c. of the $\frac{N}{2}$ oxalic acid solution. The volume of water (x) to be added is estimated according to the formula: $x = \frac{v(10-a)}{a}$ in which $v =$ the volume of the sodium hydroxid solution diluted (1100 c.c.), and $a =$ the number of c.c. used to bring about the end reaction.

10 c. c. of urine (or 5 c.c. of concentrated) are put into a Kjeldahl flask with round bottom and long neck, to which are added 10 c.c. of the sulphuric acid mixture, after about 0.4 gm. of finely divided mercuric oxid have just been placed in the flask. This mercury acts catalytically in aiding oxidation. The flask is next loosely closed with a glass ball, and heated in a special frame while kept somewhat tilted. The flame should not be too high and should be applied until the solution is colorless. Even the slightest tinge of yellow suggests that oxidation is not complete unless iron compounds are present in rather large quantities. As a rule, it takes at the most three hours to complete the oxidation. The mixture is now allowed to cool, and is poured into a distilling flask by means of a funnel. The digestion-flask and the round glass stopper are rinsed with 100 c.c. of water, which is added to the contents of the distillation-flask. Next 40 c.c. of sodium hydroxid solution of specific gravity 1340 are added and (in order to precipitate the mercury, which otherwise would form amido compounds with the ammonia) this is followed by 25 c.c. of potassium sulphid solution (4 gm. to the liter). The flask is now *quickly* connected with the distilling tube. This tube passes through a condenser, and the end is placed in an Erlenmeyer flask of about 200 to 300 c.c. capacity. This latter vessel contains 20 c.c. of the $\frac{N}{2}$ oxalic acid, and just enough water to keep the end of the condenser tube submerged.

Distillation is continued until no more ammonia escapes and the distilled steam no longer develops ammonium chlorid fumes, when a glass rod, moistened with HCl, is held in front of the open end. This condition of affairs usually obtains when about 100 c.c. of fluid have been obtained by distillation. Several drops of a methyl-orange or cyanin solution are now added to the $\frac{N}{2}$ oxalic acid solution to act as indicator, and the amount of contained ammonia is determined by titration with an $\frac{N}{2}$ sodium hydroxid solution. The amount of nitrogen in the quantity of urine employed may then be calculated from the amount of ammonia. The number of cubic centimeters of $\frac{N}{2}$ sodium hydroxid solution used

[1] *Prac. der physiol. u. path. Chemie*, 2d ed., 1900.

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is deducted from 20 c.c., and the difference multiplied by 7.02 gives the number of milligrams of nitrogen in the quantity of urine employed.

This test is best performed with the commercial Kjeldahl apparatus, which permits the oxidation and distillation of a number of specimens at the same time. This shortens very much the amount of time necessary, especially as one is usually obliged to make a series of tests.

In this form the method is perhaps rather too complicated for clinical purposes;

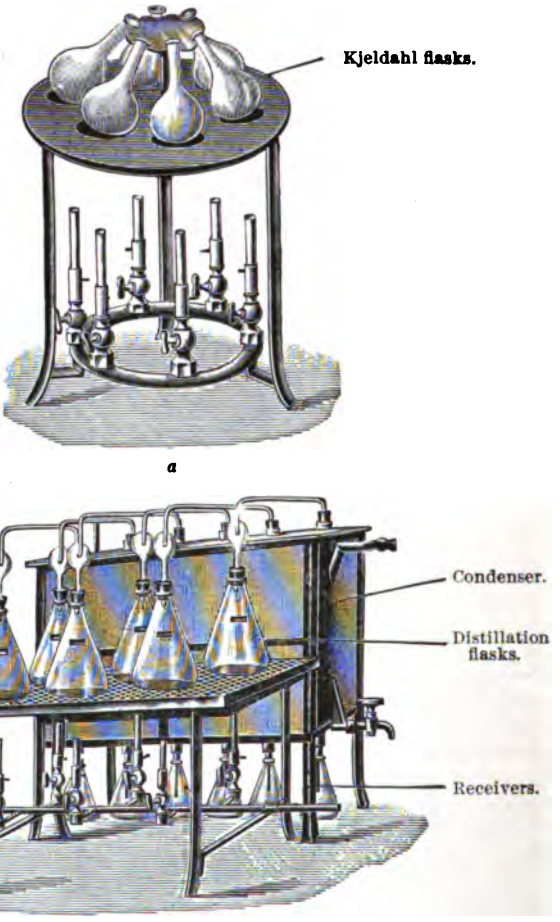

FIG. 183.—Kjeldahl's apparatus: *a*, Apparatus for the simultaneous oxidation of six specimens; *b*, apparatus for the simultaneous distillation of six specimens.

Henninger[1] and Schönherr's modification is more practical. The amount of ammonia produced by the destruction of the organic substances is estimated azotometrically, and the distillation is omitted, as in Knop-Hüfner's method of estimating urea. The ammonia is decomposed by sodium hypobromite solution, and liberated nitrogen is measured volumetrically.

The technic for the procedure is as follows: After the oxidation of 5 to 10

[1] *Compt. rend. de la soc. de biol.*, 1884, p. 474; *Jahresbericht f. Thierchemie*, 1884, p. 205. O. Schönherr, "Chemikerzeitung 12, 217," *Chem. Centralbl.*, 1888, p. 420.

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c.c. of urine according to the method given above, the solution is diluted with a little water, and partially neutralized with potassium or sodium hydroxid until the reaction is only faintly acid, and then the volume made up to 50 c.c. For azometric analysis we use 20 c.c. (corresponding to 2 or 4 c.c. of urine) of this solution and treat it exactly like the urine in Knop-Hüfner's method (p. 520), although, of course, the size of the apparatus must be changed somewhat. With Gerrard's or Dupré's modification this can be accomplished very simply by selecting a tube (Figs. 178 and 179) which will contain 20 c.c. instead of 5 c.c.

The total elimination of nitrogen in the urine generally runs parallel to the urea excretion, which, of course, contains the greater share of the nitrogen. In acute yellow atrophy of the liver, however, the comparison of the total amounts of nitrogen and of urea elimination shows a strikingly different result. In this disease the major portion of nitrogen is eliminated as leucin and tyrosin, and little or no urea is excreted in the urine.

QUANTITATIVE ESTIMATION OF URIC ACID.

A healthy adult excretes 0.2 to 1 gm. of uric acid in the urine during the course of twenty-four hours. The amount increases physiologically with increased ingestion of food, and pathologically with increased metabolism of nitrogen in about the same proportion as urea. The amount of uric acid in normal urine varies with its specific gravity. The last two numbers of the specific gravity (calculated to four places) multiplied by 2 gives approximately the number of centigrams of uric acid in the liter. The daily excretion of uric acid is pathologically increased in fever and in leukemia. Opinions still differ about uric acid excretion before, during, and after attacks of gout. Recent investigations probably disprove the old theory—that in gout more uric acid is permanently produced than normally (so-called uric acid diathesis). According to Haig, the amount of uric acid formed in the body bears a constant ratio to the amount of urea (1 : 35 both under physiologic and under pathologic conditions—*e. g.*, even in gout). He claims that variations in this ratio are due chiefly to irregularities of elimination, which in their turn depend principally upon the varying degree of alkalinity of the blood. Again, the elimination of uric acid will be increased, *ceteris paribus*, by the ingestion of uric acid and other xanthin bodies, as well as by food substances rich in nuclein (rich in cells).

According to Gubler,[1] the amount of uric acid in the urine may be approximately estimated by stratifying upon a layer of nitric acid a specimen of urine in a test tube so that the volume of urine shall be to the volume of acid as 3 : 2. After a short interval uric acid will precipitate out as a cloudy ring at the junction of the two fluids. If the amount of uric acid is increased, the precipitation will be plain within five minutes; if diminished, not until later. Of course, this test applies only when the daily volume of urine is normal. If the volume is diminished, the urine must be diluted up to the normal amount with water. Albuminous urine must first be freed from proteid by boiling, after slight acidification.

Heintz's quantitative method of estimating the amount of uric acid

[1] Laquer, *Schmidt's Jahrbücher*, 1892, vol. 236, No. 10, p. 78.

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has been abandoned entirely. It is not trustworthy. The following two methods are, however, quite accurate:

THE LUDWIG-SALKOWSKI METHOD OF ESTIMATING THE AMOUNT OF URIC ACID.

This method gives the most accurate results in the estimation of uric acid. The principle is as follows:

Uric acid, even if present only as a trace, will be precipitated from the urine as silver urate upon the addition of a mixture of a solution of ammoniacal silver nitrate and of ammoniacal magnesia solution. The precipitate is treated with an alkaline sulphid solution and so decomposed. Silver sulphid and ammonium urate will be formed; the latter is filtered off. The uric acid separates from the solution upon evaporation with hydrochloric acid, and is then dried, and weighed.

The precipitation of uric acid by the addition of ammoniacal magnesia solution has, aside from the advantage of the slight solubility of the double combination of urate, the additional advantage that triple phosphate is precipitated, at the same time mixing with the otherwise gelatinous urate precipitate and making it less tenacious and more easy to be washed.

For the procedure, the following solutions must be prepared:

1. Ammoniacal silver solution: Twenty-six grams of silver nitrate are dissolved in distilled water, and enough ammonia added to the solution to redissolve the precipitated silver oxid. The mixture is then diluted with water up to 1 liter.

2. Ammoniacal magnesia solution: One hundred grams of magnesium chlorid are dissolved in an appropriate amount of water. About 200 c.c. of a cold saturated ammonium chlorid solution are added, and then enough concentrated ammonium hydroxid to give the mixture a strong ammoniacal odor. This mixture contains a double salt of magnesium chlorid and ammonium chlorid, which is not precipitable by ammonia. The mixture is diluted with water up to 1 liter.

The solutions as given in Ludwig's directions are of such concentration that 10 c.c. will suffice for 100 c.c. of urine.

10 c.c.[2] of the silver solution are mixed with 10 c.c. of the magnesia solution, and the resulting precipitate of silver chlorid is redissolved by the addition of hydroxid. If the precipitate does not dissolve perfectly it must contain magnesium hydroxid, which can be dissolved by adding ammonium chlorid. The clear solution is poured, with stirring, into a beaker containing 100 c.c. of urine; the precipitate which forms is allowed to settle a little, then filtered off upon a suction filter, and washed two or three times with water to which a few drops of ammonia have been added. Any of the precipitate remaining in the beaker is washed out into the filter with wash-water. Traces of the precipitate may, however, be allowed to remain in the beaker, because all of the precipitate on the filter is to be removed, by the aid of a glass rod, to the beaker again, after it has become partly dry on the paper. The filter paper must be kept intact.

The precipitate is now placed in about 200 c.c. of water, according to Salkowski, acidulated with hydrochloric acid, and the mixture saturated with hydrogen sulphid. Shaffer and Folin recommend the previous addition of 5 to 10 c.c. of a 1 per cent. solution of cupric sulphate, after which the fluid is brought to the boiling-point. After the saturation with hydrogen sulphid, the fluid is again boiled for several minutes, and the hot solution of uric acid is then filtered from the silver sulphid. The precipitate should be thoroughly washed with hot water. The filtrate is perfectly clear and throws down no silver sulphate upon evaporation, just as in Ludwig's method, in which the silver is removed by an alkaline sulphid solution. The fluid is now placed in a porcelain dish upon a water bath, and evaporated to 10 to 15 c.c. From 5 to 10 drops of hydrochloric acid are now added, the mixture is allowed to stand for twelve hours, and the uric acid crystals are collected upon a weighed filter which has been dried at 110° C. The crystals upon the filter are then washed with a small quantity of water until they

[2] The following description is partly quoted from Huppert: Anleitung zur qualitativen und quantitativen Harnanalyse von Neubauer, neu bearbeitet durch Huppert, 1890.

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are free from chlorin; the quantity of the filtrate together with that of the wash water should not exceed 50 to 60 c.c. The crystals are again washed twice with absolute alcohol, then with ether, dried, and weighed.

Any sediment of urates must be dissolved by heating before measuring off the urine. Any precipitated uric acid can be dissolved in as little sodium hydroxid as possible and the solution then mixed with the urine. Too much sodium hydroxid should not be used, for it would cause excessive precipitation of phosphates, which would not allow any phosphate to remain for the production of a triple phosphate sediment with the silver magnesium precipitation. Adding sodium phosphate to the urine before the silver precipitation would, however, prevent this difficulty.

Stadthagen claims that peptones and proteoses do not influence the uric acid precipitation, but the urine must first be freed from proteid.

THE HOPKIN-WÖRNER METHOD OF URIC ACID ESTIMATION.[1]

This method is, according to all observers, simple and perfectly accurate. It depends upon the following principle: Ammonium chlorid precipitates uric acid quantitatively from the urine as ammonium urate. The determination is carried out as follows: 150 c.c. of urine are warmed in a beaker to 40° to 45° C., and 30 gm. of ammonium chlorid allowed to dissolve in it. The precipitate of ammonium urate which results after one-half to one hour's standing is filtered off, and washed free from chlorin with a 10 per cent. solution of ammonium sulphate. It is then dissolved upon the filter by a warm 1 to 2 per cent. solution of sodium hydroxid, the filter washed afterward with hot water, and the filtrate and wash-water, collected in a porcelain dish, heated upon a water bath long enough to drive off all the ammonia. The alkaline solution of uric acid is then decomposed with 15 c.c. of concentrated sulphuric acid and some copper sulphate.[2] After adding sodium hydroxid the free ammonia is estimated in the usual manner. Such a small amount of ammonia is more accurately measured by employing a $\frac{N}{10}$ oxalic acid instead of a $\frac{N}{2}$, as is usual, and also by retitration. One cubic centimeter $\frac{N}{10}$ oxalic acid corresponds to 0.0042 gm. of uric acid.

Strongly acid urines under some conditions prevent the precipitation of ammonium urate, or make such a precipitation incomplete; hence, Levandowski[3] recommends that the urine be neutralized before the precipitation. If performed in this way the method should be very exact and trustworthy.

TITRATION OF URIC ACID, ACCORDING TO HOPKIN, MODIFIED BY O. FOLIN AND TH. A. SHAFFER.[4]

This method differs from the preceding in that the ammonium urate is not dissolved by a solution of sodium hydroxid, but is washed from the filter by 100 c.c. of water. After the addition of 15 c.c. of concentrated sulphuric acid, the solution is at once titrated with a $\frac{N}{20}$ potassium permanganate solution (1.6 gm. of crystalline potassium permanganate dissolved in water and the volume made up to 1 liter). Toward the end of the titration the permanganate solution must be cautiously added drop by drop. The end reaction is the first slight permanent reddish tinge obtained by stirring the fluid. The permanganate solution may be tested by a normal oxalic acid solution. One cubic centimeter of a $\frac{N}{20}$ permanganate solution corresponds to 0.00375 gm. of uric acid.

[1] Wörner, *Zeits. f. physiol. Chemie.*, vol. xxix., p. 1.

[2] This takes the place of the oxid of mercury (p. 527). It acts catalytically, and has the advantage that the subsequent treatment with alkalin sulphid becomes unnecessary.

[3] *Zeits. f. klin. Med.*, 1900, vol. xl., pp. 3 and 4.

[4] E. Späth, *Die chemische und mikroskopische Untersuchung des Harnes*, 1903.

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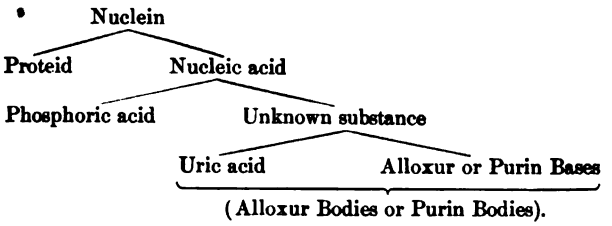
ESTIMATION OF THE ALLOXURIC OR PURIN BODIES IN THE URINE.

Recent investigations concerning the relation of purin bodies (this word is now used to include uric acid and alloxuric or xanthin bases) to the nucleins—*i. e.*, to the cell nuclei—have been mainly conducted by Kossel, Nencki and Sieber, Stadthagen, Ebstein, and Horbaczewsky. These authors have shown the clinical importance of these bases. According to our present knowledge, their formation depends upon the destruction of cellular elements, especially of the cell nuclei rich in nuclein. The following scheme may help the reader to understand the derivation of the purin bodies more clearly:



Kolish considers that gout is due to an increased formation of the purin bodies, but other investigators do not yet agree with him. However, in further work upon the subject of gout the significance of the purin bodies of the urine will be of the highest interest. Moreover, since we already know the relation of the purin bodies to the cell nuclei, the quantitative estimation of these substances will be the source of much interest in the different conditions of disease.

Until lately, Kruger and Wulff's [1] method has been generally used to estimate the total amount of the purin bodies. But it has been proved inaccurate, and so will not be described.

Salkowski's method is exact. It depends upon the precipitation of the purin bases by an ammoniacal silver solution. It determines the purin bases alone, and is analogous to the Ludwig-Salkowski method for estimating uric acid. Dénigès' method is employed at the Bern Clinic quite successfully, and will also be described.

SALKOWSKI'S METHOD OF ESTIMATING THE PURIN BASES.[2]

The precipitate obtained from the urine (at least 500 to 1000 c.c.) by precipitating with the silver magnesia solution is, after carefully washing, decomposed by hydrogen sulphid (just as in the Ludwig-Salkowski uric acid method, p. 530 *et seq.*). The filtrate is evaporated to dryness, and the residue extracted with 2 to 3 per cent. sulphuric acid. The latter dissolves the purin bases and leaves the uric acid undissolved. To be absolutely sure that at the most only traces of uric acid become dissolved, Salkowski delays filtering until the following day. The filtrate is now rendered alkaline with ammonia, and precipitated again with the silver solution. The amount of silver present in the precipitate is determined (after washing) by titration with ammonium sulphocyanate. From the amount of silver present in the precipitate can be calculated the quantity of xanthin or purin bases present.

The amount of xanthin is either figured out or the xanthin bases are separated directly from the second silver precipitate and weighed. Salkowski believes that the larger part of the purin bases does not consist of xanthin, but rather of bodies similar to hypoxanthin, as described by him in *Virchow's Archiv.*, vol. l.

[1] *Zeits. f. physiol. Chemie*, vol. xx., p. 176.

[2] *Deutsch. med. Woch.*, 1897, No. 14, and *Centralbl. f. d. med. Wissenschaften*, 1894, No. 30.

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METHOD FOR THE DETERMINATION OF PURIN BODIES, INCLUDING URIC ACID, ACCORDING TO DÉNIGÈS.[1]

This method is based on the fact that the purin bases and uric acid are precipitated quantitatively by an ammoniacal solution of silver nitrate, ammonium chlorid, and magnesium chlorid, and that a permanent precipitate of silver iodid is produced by silver nitrate in an ammoniacal solution of potassium cyanid and potassium iodid only after all potassium cyanid has been changed to potassium silver cyanid. This test furnishes the quantity of purin bodies, including uric acid.

The following solutions are necessary:

1. A $\frac{N}{20}$ ammoniacal silver magnesia solution. In a graduate of 1 liter capacity are placed 150 gm. of pure ammonium chlorid and 100 gm. of pure magnesium chlorid; it is then filled three-quarters full with ammonium hydroxid. This is gently treated on the water bath until the salts are dissolved. After cooling, 500 c.c. of this fluid are mixed with 500 c.c. of a $\frac{N}{10}$ solution of silver nitrate.

2. A $\frac{N}{10}$ solution of potassium cyanid. Ten grams of KCN are dissolved in about 1 liter of water, to which are added 10 c.c. of ammonium hydroxid, and the whole is then filtered. This solution is too concentrated. It is so diluted with a $\frac{N}{10}$ solution of silver that 10 c.c. of the latter correspond exactly to 20 c.c. of the potassium cyanid solution, since 2 molecules of potassium cyanid react with one molecule of silver. (Formation of potassium silver cyanid, $KAgCN_2$.) To standardize the potassium cyanid solution 20 c.c. are placed in a beaker, and 100 c.c. of water, 10 c.c. of ammonia, and a few drops of potassium iodid solution are then added. Next a sufficient quantity of the $\frac{N}{10}$ silver solution is added to produce a slight but persistent cloudiness. If we assume that for this purpose it is necessary to use $10 + n$ c.c. of the silver solution, then there must be added to the potassium cyanid solution water in the proportion $2\ n$ c.c. to each 20 c.c. A potassium cyanid solution so prepared will correspond to an equal volume of the $\frac{N}{20}$ silver solution.

3. A solution of potassium iodid prepared by dissolving 20 c.c. of potassium iodid in 100 c.c. of water and adding 2 c.c. of ammonium hydroxid.

4. A $\frac{N}{10}$ silver solution. Seventeen grams of pure and dry silver nitrate are dissolved in 1 liter of water.

These solutions are utilized in the following manner to determine the purin bodies of the urine. To 100 c.c. of urine there are added 25 c.c. of the ammoniacal $\frac{N}{20}$ silver magnesia solution. This is well shaken and the precipitate (consisting of silver combinations of the purin bases) filtered off. To 100 c.c. of the filtrate, which correspond to 80 c.c. of urine and 20 c.c. of the silver solution, are added 20 c.c. of the potassium cyanid solution. This volume of potassium cyanid solution would react exactly with 20 c.c. of the silver solution if a part of the silver had not been used up by the purin bases, and thus been removed by filtration Inasmuch as the quantity of silver is less, there will be an excess of potassium cyanid. This excess is estimated by adding as an indicator a few drops of potassium iodid solution, and then from a buret $\frac{N}{10}$ silver solution until a permanent cloudiness is produced. The amount of silver solution added will correspond exactly to the amount of silver held in combination by the purin bodies. By far the greater part of the purin precipitate is made up of uric acid, so that the final result, so far as uric acic is concerned, may be figured as follows:

[1] After C. Vieillard, *L'urine humaine*, Paris, 1898; original in *Bull. de la soc. de pharm. de Bordeaux*, 1884, p. 137.

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Each cubic centimeter of the $\frac{N}{10}$ silver solution corresponds to 0.0168 gm. of uric acid. If n c.c. silver solution have been used, these 80 c.c. of urine will contain $n \times 0.0168$ and 1 liter of urine $\frac{0.0168 \times 1000 \times n}{80} = 0.21 \times n$ purin bodies in terms of uric acid.

ESTIMATION OF THE KREATININ IN THE URINE.

Kreatinin in the urine is probably derived from the kreatin of the muscles. Kreatinin is the anhydrid of kreatin. Normal urine probably does not contain kreatin. The excretion of kreatinin generally runs parallel to the excretion of urea. An increased ingestion of meat, and in all probability any augmentation of muscular metabolism, increases the excretion of kreatinin. Here lies the clinical interest in the quantity of kreatinin in the urine. The various results of experiment as to whether the kreatinin increases in amount with increased muscular work differ. It is generally assumed that the excretion of kreatinin is not increased by muscular exertion. According to Thomas and Spath,[1] the daily excretion of kreatinin of healthy men is about 1 gm. The excretion of kreatinin in disease (in spite of its theoretic interest) has not been followed clinically very closely. A pronounced increase has as yet been noticed only during the fever of acute diseases.

An accurate quantitative determination of kreatinin may be got (Neubauer)[2] by preparing and weighing the kreatinin zinc chlorid precipitated from the urine. The original article must be consulted for the technic. (Compare Neubauer and Vogel.)[3]

Kreatinin can be demonstrated qualitatively in the urine by either of the following two simple methods, which may also be used for a quantitative estimation:

Jaffé's Reaction.[4]—The addition of a little aqueous solution of picric acid and a few drops of diluted sodium or potassium hydroxid to a solution of kreatinin will immediately produce an intense red color even when the amount of kreatinin in the urine is only 1 : 5000. The color will increase for a few moments, and then persist unchanged for hours. Acetone also gives a red color, although a much less intense one; hence, it is wise first to remove by boiling any acetone that may be present in the urine.

Th. Weyl's Reaction.[5]—A few drops of a very dilute solution of sodium nitroprussid is added to the urine, and then some diluted sodium hydroxid. If kreatinin is present a ruby-red color will result, which soon changes to a yellow. Up to this point the reaction is identical with Legal's acetone test (p. 495 *et seq.*). If, however, acetone is present, the fluid turns red again when acidified with acetic acid; this is not the case with kreatinin. Salkowski claims that if kreatinin is present the addition of acetic acid and subsequent heating produce a greenish and then bluish coloration (Prussian blue). Before attempting Weyl's test, the acetone may be expelled by boiling the urine.

QUANTITATIVE ESTIMATION OF THE CHLORIDS IN THE URINE.

The amount of chlorid eliminated in the urine of a healthy adult in twenty-four hours varies between 11 and 15 gm., calculated as sodium chlorid. Chlorids of the urine are diminished in hunger and diarrhea,

[1] *Harnanalyse von Neubauer und Vogel, Neu bearbeitet von Hüppert und Thomas.* Kreidel, Wiesbaden, 1890.

[2] *Ann. der Chemie. u. Pharm.*, 1861, vol. cxix., p. 33. Modification der Methode durch Salkowski, *Zeits. f. phys. Chemie*, 1886, vol. x., p. 113.

[3] *Die chem. u. mikroskopische Untersuchung des Harnes*, 1903.

[4] *Zeits. f. phys. Chemie.*, 1886, vol. x., p. 399.

[5] Th. Weyl, *Ber. d. chem. Gesellschaft*, vol. xi., p. 2175.

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from the formation of exudates or transudates rich in sodium chlorid, and in fever.

It has been assumed that the diminution of chlorids eliminated in the urine in fever depends entirely upon the diminished ingestion of food. This is certainly wrong, because in some other cases, when much less food is ingested, no such marked diminution of the chlorids appears in the urine—*e. g.*, in pneumonia. Again, in pneumonia the amount of chlorin eliminated increases very markedly immediately after the crisis without any increased ingestion of food. No absolutely correct explanation of the diminution of chlorin in fever has yet been advanced. One theory suggests that in fever the cell-proteids, which are relatively poor in chlorin, are disintegrated much more extensively than the circulating proteids. Another theory is that the formation of inflammatory exudates deprives the urine of a considerable amount of chlorin. This agrees very well with the fact that in no other disease is there so great a diminution in the amount of chlorin as in croupous pneumonia, where exudation is very acute. A third assumption is that the chlorids are kept back as a sequence of the supposed retention of water in fever. Probably several of the above-mentioned factors act together in febrile disturbances to diminish the chlorids in the urine.

It is practically important, however, to remember that in febrile diseases any improvement in the condition will increase the amount of chlorids in the urine, and often before any improvement is indicated by the thermometer—*e. g.*, in pneumonia. Any great diminution in the amount of chlorids in non-febrile disorders always points to a serious condition. On the other hand, there are some very severe general conditions (the author has paid particular attention to this in circulatory disorders) in which no diminution of chlorids is observed.

A pronounced diminution or absence of chlorids in the urine in a febrile disorder will always suggest pneumonia, because, as has already been mentioned, the greatest degree of diminution is reached in this disease.

For practical purposes an approximate estimation of the chlorids, made as follows, will suffice: A specimen of urine in a test tube is acidified with about 10 drops of pure nitric acid. Then a single drop of silver nitrate solution (1 : 10) is added. If the chlorids are normal a solid ring, a ball, or one or more compact lumps of silver chlorid form and settle to the bottom; if diminished, only a more or less intense cloudiness arises. With a little practice the relative amount of chlorids may be judged by this simple test. Nitric acid is employed because it precipitates any proteid present, which otherwise would be precipitated by the silver solution, and might thus lead to deception; and also because it prevents the precipitation of silver phosphate, which might be mistaken for the silver chlorid. If a large amount of proteid appears after the addition of the nitric acid, it must first be removed by filtering or by letting it settle to the bottom before performing the chlorid test.

Volhard's procedure (titration with silver and ammonium sulphocyanate solution) is the most reliable method. It is described in con-

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nection with the Lütke-Martius method of estimating the hydrochloric acid of the gastric juice, and can also be employed for the urine (see p. 380 *et seq.*). In employing this method it is best to use the urinary ash, since, if the silver solution is added to the urine, it precipitates not only the chlorids, but also the purin bodies, which would consequently give rise to a slight error.

QUANTITATIVE ESTIMATION OF THE PHOSPHATES IN THE URINE.

The daily excretion of phosphoric acid in the urine, in the form of phosphates, amounts normally to 2 to 3 gm. The ratio of the excreted phosphoric acid to the amount of excreted nitrogen is normally 0.18 (15 urea = 7 nitrogen). This ratio, $\frac{P_2O_5}{N}$, is termed the relative value of the phosphoric acid excreted. In febrile conditions the absolute value of phosphoric acid elimination may be diminished or increased (more frequently the first), but Zülzer claims that the relative value is constantly diminished. In convalescence the absolute and relative values immediately increase to normal or sometimes above. According to Fleischer, a considerable diminution in the absolute, as well as the relative, phosphoric acid values seems to be constant in nephritis.

Quantitative estimation of phosphates in so-called phosphaturia—*i. e.*, voiding of a urine clouded with phosphates—has shown that this condition does not by any means signify an increase of the daily quantity of phosphates, but only a precipitation of the phosphates in consequence of the diminished acidity or even alkalinity of the urine or increase in the quantity of the alkaline earths present (see p. 557, *et seq.*).

The quantitative estimation of phosphates is accomplished by titration with uranium nitrate in a solution of acetic acid, which precipitates all the phosphates.

The end reaction is either the brown color which is produced by an excess of uranium nitrate in the presence of potassium ferrocyanid, or the green color formed by tincture of cochineal with a surplus of uranium.

The following solutions are required:

1. A solution of uranium nitrate, which must be standarized with a solution of disodium phosphate of known strength. About 35 gm. of uranium nitrate are dissolved in a liter of water.

2. A solution of disodium phosphate, so prepared that 50 c.c. shall contain 0.1 gm. P_2O_5. This solution is prepared from the ordinary pure disodium phosphate; but since this salt contains a varying quantity of water, and consequently a varying amount of P_2O_5, the solution must be standardized. This is done as follows: Twelve grams of pure disodic phosphate are dissolved in a liter of water, and 40 c.c. of the solution are titrated with $\frac{N}{10}$ hydrochloric or sulphuric acid, alizarin red being employed as an indicator. The end reaction corresponds to the moment when all of the disodium phosphate is transformed into the monosodium phosphate, and is indicated by the red solution turning yellow. If the solution is correct—*i. e.*, if 50 c.c. contain 0.1 P_2O_5—the 40 c.c. should require 11.25 c.c. $\frac{N}{10}$ hydrochloric acid before the end reaction occurs. As a rule, more is required, and the original solution must consequently be diluted. For example, if 13 c.c. $\frac{N}{10}$ hydrochloric acid are required, and x represents the amount of fluid to which the 40 c.c. must be diluted, we have the proportion:

$$40 : 11.25 = x : 13$$

$$\text{and } x = \frac{40 \times 13}{11.25}$$

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The sodium phosphate solution must then be so diluted that every 40 c.c. is brought up to the volume x. If the solution contains too little phosphate, it must be concentrated by evaporation according to a corresponding proportion.

3. Acetic-acid sodium acetate solution. One hundred grams of sodium acetate are dissolved in 800 gm. of water, 100 c.c. of a 30 per cent. acetic acid solution are added, and the mixture is diluted to 1 liter.

4. Potassium ferrocyanid solution. 10 : 100 water or tincture of cochineal. The latter is prepared by dissolving 6 gm. of powdered good cochineal in a mixture of 300 c.c. of distilled water and 200 c.c. of alcohol. The mixture is kept at an ordinary temperature for several hours, during which time it is frequently shaken, and is then filtered.

Preparation of the Uranium Nitrate Solution.—Fifty cubic centimeters of the prepared solution of sodium phosphate are placed in an Erlenmaier flask, treated with 5 c.c. of the acetic-acid sodium acetate solution, and heated to the boiling-point. A solution of uranium nitrate (35 : 1000) is now added from a buret as long as there is a distinct precipitate formed. The solution should now be tested after the addition of every ½ c.c. by taking a drop of the fluid out of the flask by means of a glass rod, and mixing it upon a porcelain plate with a drop of a potassium ferrocyanid solution. The end reaction is the appearance of a reddish-brown color. A simpler method of attaining the same end is to add several drops of the tincture of cochineal to the fluid in the flask, bring it to the boiling-point, and add the uranium nitrate solution until a permanent pale-green color is obtained after mixing and reheating. It is now known how much uranium nitrate solution is necessary for the precipitation of 50 c.c. of the sodium phosphate solution, and the uranium solution is to be diluted so that exactly 20 c.c. will precipitate 50 c.c. of the sodium phosphate solution. The 20 c.c. of uranium solution are then equivalent to 0.1 P_2O_5, or 1 c.c. is equivalent to 0.005 gm. P_2O_5.

Estimation of the Total Phosphates in the Urine.—If proteids be present they must be removed; sugar does not interfere with the reaction. We proceed with the urine exactly as we did in the preparation of the uranium nitrate solution—*i. e.*, we titrate 50 c.c. of the urine and 5 c.c. of the acetic-acid sodium acetate solution at the boiling-point with the standardized uranium solution until the end reaction is obtained with potassium ferrocyanid or cochineal. Every cubic centimeter of the uranium solution employed then corresponds to 0.005 P_2O_5.

Separate Estimation of the Earthy Phosphates.—In reference to so-called phosphaturia (see pp. 536 and 557), it is of interest to make a separate estimation of the earthy phosphates. The difference between the total phosphoric acid and the earthy phosphates then gives the alkaline phosphates. To estimate the earthy phosphates we proceed as follows: One hundred cubic centimeters of urine are rendered alkaline with ammonia, the mixture is allowed to stand for twelve hours, the precipitate removed by filtration and washed with ammonia water. The filter is now perforated with a glass rod, and the precipitate is washed into a beaker. After dissolving the precipitate by heat in as small a quantity of acetic acid as possible, the solution is diluted to 50 c.c. with water, 5 c.c. of the acetic-acid sodium acetate solution are added, and the mixture is titrated with the uranium solution, as in the estimation of the total phosphates.

QUANTITATIVE ESTIMATION OF SULPHURIC ACID AND OF THE COMBINED SULPHURIC ACID.

Although the total amount of sulphuric acid eliminated is as yet of no particular clinical importance,[1] the amount of the combined sulphuric acid—*i. e.*, sulphuric acid united with organic substances—*e. g.*, phenol, indol, etc.—to form the so-called ethereal sulphates is of much greater interest.

[1] The so-called total sulphuric acid of the urine consists of sulphuric acid in the ordinary sense of the word, and of sulphuric acid in combination—*i. e.*, with various organic substances.

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We can estimate sulphuric acid proper separately from combined sulphuric acid by titration, because in a urine which either is not acidified, or is acidified entirely by acetic acid, barium solutions will precipitate only the sulphuric acid proper as barium sulphate; whereas in the presence of hydrochloric acid the ethereal sulphuric acid will be freed from its combinations and will be precipitated along with the inorganic sulphuric acid. By subtracting the value of the sulphuric acid proper from that of the total sulphuric acid, the amount of combined sulphuric acid may be obtained.

The normal daily excretion of total sulphates in the adult amounts to 1.5 to 3 gm. of SO_3, and parallels the quantity of proteid decomposed. The normal ratio of the daily amount of the total sulphates to the daily quantity of urea is about 1 : 5, while the ratio of the quantity of the ethereal sulphates to that of the remaining sulphates is about 1 : 10.

Many processes of decomposition within the body, particularly increased intestinal putrefaction, show an increase in the amount of combined sulphuric acid at the expense of the inorganic sulphuric acid. An abundant administration of phenol (carbolic acid poisoning) produces the same effect.

QUANTITATIVE ESTIMATION OF AMMONIA IN URINE.

The normal amount of ammonia contained in adult urine, according to Neubauer, varies in twenty-four hours between 0.3 and 1.2 gm., and averages 0.7 gm. A meat diet, the ingestion of radishes, the breathing of air saturated with tobacco smoke, and the taking of ammonium salts and mineral acids as medicaments increase the daily amount of ammonia excreted.

The quantitative estimation of ammonia in the urine may be of clinical interest under certain circumstances. We know that the human organism, like that of carnivora in general, reacts to an increased ingestion of acid or increased acid formation by an increased production of ammonia, which serves the purpose of holding the otherwise injurious acids in combination. Consequently, the amount of ammonia salts contained in urine is an important indicator of acid metabolism.

The daily quantity of ammonia excreted is increased pathologically in diseases of the liver and in acute febrile diseases, such as pneumonia and typhoid fever. The increase is usually at the expense of the urea. In affections of the liver there is a specific impairment of its urea-forming function; in febrile diseases the increased quantity of ammonia is the effect of the increased formation of acids in consequence of the augmented decomposition of proteid. The increase in the amount of excreted ammonia is most striking in diabetic acidosis.

This is of practical importance in diabetes mellitus. The increased elimination of ammonia led in this case to the discovery that certain diabetic urines contain β-oxybutyric acid (compare p. 539), and that the amount of ammonia eliminated indicates in an approximate manner the degree of acidosis.[1]

To be sure, the amount of ammonia in the urine is not exactly parallel to the acid formation in the organism, because the ammonia becomes serviceable for the neutralization of acids only when the available fixed alkalies are no longer sufficient.

Magnus-Levy makes the following estimation: About 1 to 2 gm. of the ammonia excreted by a diabetic are for the purpose of neutralizing the fixed excess of acid resulting from the proteid diet, and only the remainder is the measure for the pathologic excretion of acid. A diabetic with 4 gm. of ammonia in the urine is consequently excreting not twice, but nearly three times, as much pathologic acid as one with 2 gm., since 1 gm. or more of the 2 or 4 respectively is combined with the acids normally excreted. According to this supposition, when 2 gm. of ammonia are excreted, about 1 gm. is utilized for combination with the two pathologic acids of diabetes (oxybutyric and diacetic acid); while an elimination of 5 gm. leaves 3 to 4 gm. for this purpose. An excretion of 8 gm., which is but rarely observed, gives 6 to 7 gm. of ammonia as

[1] *Cf.* Naunyn, "Diabetes Mellitus," in *Nothnagel's Sammelwerk*, 1898, p. 179; and Magnus-Levy, *Arch. f. exp. Path.*, 1899, vol. xlii., p. 1981.

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the measure of the pathologic amount of acids. These figures correspond to 6, 20, and 36 to 40 gm. of β-oxybutyric acid. Magnus Levy also points out that such an estimation of the acids from the ammonia is of value only when an average is taken from the observations extending over several days, since the amount of ammonia formed in any twenty-four hours will vary considerably as the result of the temporary retention or excretion of alkalies. The large quantity last mentioned, of 40 gm. of oxybutyric acid, he says, represents the maximum quantity of acid which the body can neutralize by producing ammonia and without the administration of sodium bicarbonate. By administering 40 gm. of this substance, however, the organism may eliminate 60 gm. of oxybutyric acid daily in the urine. Magnus Levy believes that this latter quantity is exceeded only in diabetic coma.

Schlösing has described the simplest method for estimating the amount of ammonia. It depends upon the fact that aqueous solutions of ammonia readily part with their ammonia when exposed to the air, and that when dilute sulphuric acid is placed in a closed chamber near the ammonia, the acid quickly and completely absorbs the ammonia. Therefore, if we free the ammonia from a known quantity of urine by the addition of lime water, and then allow it to become absorbed in a closed space by a known amount of sulphuric acid, the amount of absorbed ammonia can easily be determined by a simple acidity titration. Neubauer proceeds as follows: A shallow dish with vertical sides, containing 25 c.c. of filtered urine, is placed upon an exsiccator plate. A triangular glass is set over the top of the dish and holds another vessel containing 10 c.c. of normal sulphuric acid. At least 10 c.c. of lime water are now added to the urine, and the whole is then quickly covered with the glass bell of the exsiccator, whose margin is greased with tallow. All the ammonia in the urine will be absorbed by the acid within three or four days. Then, using methyl orange as an indicator, the acid is all titrated with $\frac{N}{4}$ sodium hydroxid till the red color turns to yellow. Every cubic centimeter less than 40 of the sodium solution employed corresponds to $\frac{17}{4}$ mg. of NH_3.

The objection to Schlösing's method is the length of time required for the estimation. Folin[1] tried to shorten the period by forcing the freed ammonia into the acids by a current of air. Nenski and Zaleski,[2] as well as Söldner,[3] distil the freed ammonia in a vacuum into the titrated acids. These methods require carefully made apparatus, and for their details the writer must refer the reader to the original communications.

Schmiedeberg's[4] platinum chlorid method may be more quickly carried out than Schlösing's, but it is also more complicated.

QUANTITATIVE ESTIMATION OF THE β-OXYBUTYRIC ACID IN THE URINE.

The quantitative estimation of β-oxybutyric acid is of great interest in diabetic acidosis. Polarization is a practical chemical method. β-oxybutyric acid is levogyrate, and has a specific rotating power of 23.4° or of 20.6°, according to different authorities. Fermented urine is freed from proteid and decolorized with lead acetate and ammonia or mercuric chlorid, and its rotatory power is then determined (p. 516 *et seq.*). If the 100 mm. tube of the polaristrobometer is used (p. 517), Minkowski states that a rotation of 1° will correspond to $\frac{100}{20.6}$ per cent. of β-oxybutyric acid (about 5 per cent.). In this estimation we must naturally assume that the urine does not contain any other non-fermentable substances which are levogyrate,

[1] *Zeits. f. phys. Chem.*, 1902, vol. xxxvii., p. 161.
[2] *Ibid.*, 1901, vol. xxxiii,, p. 237.
[3] *Zeits. f. Biol.*, vol. xxxviii., p. 237.
[4] *Arch. f. exp. Path.*, vol. vii., p. 166.

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such as the combined glycuronates, etc. A positive Trommer's test with the fermented urine would point to their presence. The normal constituents of the urine which turn the plane of polarized light to the left, such as uric acid and kreatinin, do not disturb the test, because the rotation caused by them is very slight, and, besides, there is only a very small percentage of them in diabetic polyuria.

Magnus Levy[1] regards the quantities obtained by this method inaccurate, and thinks they are too large, but is unable to give a satisfactory explanation of the marked levogyration found in these cases.

For clinical purposes the quantitative estimation of the excretion of ammonia is the best indirect means for determining the amount of β-oxybutyric acid excreted.

Darmstädter's[2] method for the direct estimation of the β-oxybutyric acid seems worthy of recommendation. It depends upon the fact that β-oxybutyric acid is broken up into crotonic acid and water by concentrated mineral acids. The method is carried out as follows: 100 c.c. of urine are rendered alkaline with sodium carbonate and evaporated almost to dryness; the residue is dissolved in 150 to 200 c.c. of 50 to 55 per cent. sulphuric acid; 300 to 350 c.c. are now distilled from this mixture within two to two and one-half hours, the fluid removed being replaced by water; the distillate is now shaken two or three times with ether, the ether evaporated, and the residue heated to 160° C., in order to remove any volatile fatty acids which may be present. The residue is then dissolved in 50 c.c. of water, the solution filtered, and titrated with tenth-normal sodium hydroxid solution: 1 c.c. tenth-normal sodium hydroxid solution equals 0.0086 crotonic acid; 1 part crotonic acid equals 1.21 β-oxybutyric acid.

According to Magnus Levy, 60 gm. of β-oxybutyric acid may be excreted when diabetic coma is not present, while in coma daily amounts of over 150 gm. have been observed. The percentage found by Magnus Levy in diabetic urine rarely exceeded 0.5 to 1 per cent.

QUANTITATIVE ESTIMATION OF THE AMOUNT OF ACETONE IN THE URINE.

Acetonuria is an accompanying phenomenon of diabetic acidosis; hence, in addition to the β-oxybutyric acid estimation, a quantitative acetone estimation is of some importance. The simplest method is to weigh the iodoform which is produced from acetone by Lieben's reaction. One hundred cubic centimeters of urine are concentrated *in vacuo* to 10 c.c., and well cooled, a large excess of sodium hydroxid and a supersaturated iodopotassium iodid solution (p. 495, Lieben's Reaction) are then added to the distillate. The resulting iodoform is allowed to stand twenty-four hours in the solution, and is then filtered through a weighed filter (glass-wool); the precipitate is washed with a little cold water, and then allowed to dry for a short time over sulphuric acid. One gram of iodoform corresponds to 0.147 gm. of acetone. It is simpler to extract the iodoform by means of ether. For this purpose 20 c.c. of ether free from alcohol are placed in the vessel in which the iodoform is formed, the mixture is well shaken and 10 c.c. of the ether are removed by means of a pipet and evaporated in a weighed dish. The residue is dried over sulphuric acid and weighed.

Acetone and diacetic acid usually occur together in the urine; hence, the iodoform obtained includes not only that derived from the preformed acetone, but also that derived from the acetone formed from the diacetic acid by the distillation. Acetone and diacetic acid are about equally valuable in diagnosis (see p. 493), so this is no particular disadvantage. Besides, as we have no exact method of determining the amount of diacetic acid, the estimation of the acetone is the only way in which we can obtain an approximate idea of the amount of diacetic acid.

[1] *Arch. f. exp. Path.*, vol. xlii., p. 167 *et seq.*
[2] *Zeits. f. physiol. Chem.*, 1902–3, vol. xxxvii., p. 355.

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ESTIMATION OF THE TOTAL DRIED RESIDUE OF URINE.

Sometimes this is of clinical interest. The author has demonstrated that a watery diuresis—*i. e.*, diuresis produced by an increased consumption of water, more especially from a subcutaneous or intravenous infusion of salt solution—will increase the twenty-four-hour excretion of solids in the urine. The method of determination is very simple. Fifty cubic centimeters of urine in a crucible are weighed, evaporated to a syrupy consistence over a water bath at a very moderate temperature (not over 60° C.) after adding 2 or 3 drops of acetic acid, then dried further in a vacuum over sulphuric acid to a uniform weight. The urine must not be heated above 60° C., as otherwise ammonia will be given off from the urea. The addition of acetic acid is necessary to combine with any ammonia which may have been liberated in spite of the care taken when evaporating.

It is still safer to dry entirely in a vacuum without heating, but then a much smaller amount of urine (at most 5 c.c.) should be employed. A drop of acetic acid is added to the urine in a very shallow dish, along with a generous amount of sulphuric acid, and it is then allowed to dry in the vacuum to an absolute weight. If necessary, the sulphuric acid can be replenished and the vacuum restored by repeated exhaustion.

An approximate estimation of the total amount of solids in 1 liter of urine can be obtained from the specific gravity of the urine (see p. 451).

URINARY ACIDIMETRY AND ALKALIMETRY.

(Determination of Acidity and Alkalinity. Estimation of the Acidic and Basic Points.)

The conditions which affect the reaction of the urine have already been discussed upon p. 455 *et seq.* The reaction of individual specimens of urine varies; hence, the degree of acidity must be determined from a specimen of the mixed twenty-four-hour urine. The addition of a few drops of chloroform, or keeping the urine upon ice, will prevent any change in the reaction due to decomposition.

The quantitative estimation of the reaction by means of acidimetry or alkalimetry is accomplished by titration. Certain objections have been constantly raised in reference to the possibility of titrating the acidity of the urine, and these objections have also been fully considered in Nägeli's[1] experiments upon this subject, which were undertaken at the writer's request. The writer fully realizes the theoretic justification of all these objections, which arise from the fact that the end reactions of the titration of the phosphoric acid are theoretically never distinct, and not always so, even from a practical standpoint. The reasons for this fact have been clearly explained by T. Heffter.[2] So long as the chemist gives us nothing better, however, we are forced to use these methods in practice, even though they are not strictly accurate, and Nägeli has at least shown that it is possible to obtain approximate values for the acidity of the urine by means of titration.

In view of the undeniable difficulties of titrating the urine acidimetrically the writer thinks it is entirely beside the mark to attempt to replace the classic conception of acidity by an entirely different one—namely, the concentration of the hydrogen ions—instead of trying to improve the method of titration. The acidity is not due to the concentration of the hydrogen ions, but to the number of hydrogen atoms capable of being replaced by metals and contained in a given quantity of a fluid. These are entirely different conceptions, and, since the old conception of acidity is still upheld by the teachings of physical chemistry, nothing but confusion can result from the substitution of a new conception. For this reason the writer believes that L. v. Rohrer's[3] estimation of the acidity

[1] Zur Aciditätsbestimmung des Urines, *Zeits. f. physiol. Chemie*, vol. xxx., parts 3, 4, and 5, p. 366.

[2] *Ergebnisse der Physiol.*, I. Jahrg., 1902.

[3] *Pflüger's Archiv*, vol. lxxxvi.

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of the urine by determining the concentration of the hydrogen ions tends to lead us into a hopeless tangle.

Nägeli tested a large number of indicators in reference to their behavior toward the more important salts occurring in the urine, and ascertained how much information could be obtained from them by any change of color they might undergo upon titration with an alkali or an acid, in reference to the chemical saturation of the acidic or basic affinities of the urine. The following discussion gives the essential results and conclusions obtained by Nägeli.

Only two reagents, phenolphthalein and alizarin-red,[1] are of any value for the purpose under discussion. Litmus is absolutely useless; it never shows any sharp change of color when titrating phosphates. When phenolphthalein is used the end reaction is indicated by the bright-red color shown by this indicator when the acid solution becomes alkaline on the addition of $\frac{N}{10}$ sodium hydroxid solution. (For preparation see p. 378). When alizarin-red is employed, the change from red to yellow indicates the end reaction which takes place upon adding $\frac{N}{10}$ hydrochloric acid to an alkaline solution, the moment free acid is present. Alizarin-red does not react to acid salts nor to CO_2. The following table indicates best how the most important constituents of the urine react on titration toward the above indicators:

Titration with phenolphthalein and $\frac{N}{10}$ NaOH.		Yellow as soon as free HCl is present.
		Acidic point.
Colorless as long as are present:	NaH_2PO_4, KH_2PO_4. $NH_4H_2PO_4$, $Ca(H_2PO_4)_2$, H_2CO_3 (carbonic acid)	Red.
Neutral point:		
Red, as soon as NaOH changes all to:	Na_2HPO_4, Ka_2HPO_4, $(NH_4)_2HPO_4$, $CaHPO_4$, $NaHCO_3$ $KaHCO_3$	Titration with alizarin-red and $\frac{N}{10}$ HCl.
Basic point: (free alkali) not determinable.		

Urea, chlorides, sulphates, urates, oxalates, kreatinin, *et al.*, remain indifferent to titration, but, on account of their small quantity, they are unimportant.

The above table shows that titration of urine with sodium hydrate and phenolphthalein to determine the point of neutralization gives valuable information as to acid affinities and acid equivalents. The value found, usually expressed in cubic centimeters of $\frac{N}{10}$ sodium hydroxid, is usually termed the acidity of urine. This does not, of course, indicate the amount of acid excretion, but only how much the acid equivalents contained in the urine exceed the basic equivalents. The amount of acid in the form of neutral salts (NaCl) or salts reacting alkaline to phenolphthalein (Na_2HPO_4) is, of course, not included in the acidity. Besides this, it should be remembered that, when titrating with phenolphthalein, the neutral point is placed where all phosphates are transformed into secondary salts corresponding to the formula Na_2HPO_4, and the H_2CO_3 is changed to $NaHCO_3$. In this sense the acidity is not identical with the basic capacity, because phosphates with the formula Na_2HPO_4 can still take up one more basic atom. The basic point—*i. e.*, the moment Na_2HPO_4 is changed to Na_3PO_4 by the addition of NaOH and free alkali becomes present—cannot be determined by any indicator.

[1] Alizarin-red of Merck (Alizarin sodium sulphonate).

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By titration with $\frac{N}{10}$ HCl and alizarin-red one obtains in a similar manner information as to the basic equivalents in the urine. Just as the acidity is measured with $\frac{N}{10}$ sodium hydroxid, the basidity is measured with $\frac{N}{10}$ HCl. The acidity can be ascertained only to the neutral point, not to the basic point, whereas the total basicity may be determined, because the moment when free acid appears, following the addition of $\frac{N}{10}$ HCl (acid point), is indicated sharply by alizarin-red.

Leaving out of consideration the urates and oxalates, the sum of the values according to alizarin and phenolphthalein corresponds approximately to the contents of carbonic and phosphoric acid molecules in the urine. Finally, the two titrations give an insight as to the amount of the bases which are excreted combined with phosphoric acid. If the urates, oxalates, and carbonates are not considered, this amount is approximately obtained when the value of the phenolphthalein titration is added to double that of the alizarin titration. In the titration with phenolphthalein, as much alkali equivalent is added to complete the reaction as there is in the phosphates corresponding to the formula NaH_2PO_4 and appearing in the reaction; while in the alizarin reaction one-half as much acid equivalent must be added in order to bring about the end reaction. This calculation contains considerable errors only when there are present in the urine relatively large quantities of carbonates, oxalates, or urates, which may enter into the reaction. This is seldom the case, however.

Urines which reacted alkaline to phenolphthalein could be titrated by means of $\frac{N}{10}$ HCl with the indicator and the neutralization point accurately determined if the alkalinity were caused by *fixed* alkalies. This condition, however, has never been observed. In general, a urine which reacts alkaline to phenolphthalein contains ammonium carbonate, and in this case the addition of $\frac{N}{10}$ HCl causes the formation of acid ammonium carbonate, which immediately decomposes and liberates CO_2 and NH_3. The indicator, being much more sensitive to CO_2 than to NH_3, becomes decolorized (by the CO_2) long before the NH_3 in the urine is completely neutralized—*i. e.*, before all the $(NH_4)_2NCO_3$ is transformed into $(NH_4)HCO_3$. The obtaining of an accurate end reaction with phenolphthalein under these conditions is therefore impossible. Actually, however, this difficulty presents no serious disadvantage, because the ammoniacal character of urine, with the exception of the presence of ammonium carbamic acid as the result of calcium-feeding, is caused exclusively by the bacterial decomposition of urine. In such cases quantitative determinations have no interest. On the other hand, it is to be emphasized that the determination of the acid point—*i. e.*, the point at which all the NH_4Cl is transformed into HCl—presents no difficulties. Such determinations acquire under certain conditions clinical interest in the consideration of the degree of bacterial decomposition of freshly voided urine in cystitis.

On the other hand, it is conceivable that titration with HCl in the presence of alizarin does not lead to accurate results if the urine contains large amounts of ammonium salts, as is the case in diabetic acidosis. Nägeli found in the titration of the ammonium salts of dibasic acids—*e. g.*, ammonium oxalate with HCl—that the change of color of the alizarin into yellow does not take place at the moment of the appearance of free acid, but as soon as the acid salts are formed. The color-change is also too gradual to allow of sharp reading. Nevertheless, this disturbance of the reaction does not come into play in the case of a monobasic acid, such as β-oxybutyric. In the normal condition of the excreted ammonium salts, these hardly effect the determination of the acid point. In cases where the ammonium content of the urine is excessive, the determination of the acid point can be performed as follows: In a portion of the urine the ammonium is quantitatively determined according to the Schlösing method (p. 539), and then to another portion is added the equivalent amount of NaOH,

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sufficient to set the NH_3 free. This ammonia can be driven off by heat, and the solution then titrated in the usual way.

In practice the titration is carried out in the following manner: To 2 portions of 10 c.c. of the urine are added respectively phenolphthalein and alizarin. The first is titrated with $\frac{N}{10}$ NaOH to the appearance of a permanent red color. To the second, $\frac{N}{10}$ HCl is added until the red color has changed to yellow. The color of the urine itself does not interfere in the titration to any noticeable extent, especially if there is placed alongside of the beaker in which the titration is being performed a second, for control, with equal amounts of urine and indicator to which no acid or alkali is added. Both solutions should be diluted to an equal extent. Strongly colored urine can be decolorized by animal charcoal which has been tested and found to be neutral. If the end reaction is not sharp and the cause is to be ascribed to the presence of ammonium salts, then the latter may be removed by NaOH in equivalent amounts, as before described. For the technic of titration and preparation of normal solutions see pp. 378 and 381. Proteid must be removed by heat and acetic acid, in which case a known amount of the acid should be added, and this taken into consideration in the titration. The values found in these titrations may be expressed in cubic centimeters of $\frac{N}{10}$ NaOH, or calculated in grams of NaOH (a $\frac{N}{10}$ NaOH solution contains in a liter 4 gm. of NaOH). Haig found that the average acidity of the daily urines of healthy persons equalled 5.5 gm. of oxalic acid or 3.5 gm. of NaOH. Späth, however, gives a daily acidity of 1.45 gm. of HCl, corresponding to 1.60 gm. of NaOH.

Freund has lately suggested, and Lieblein[1] has agreed with him, that the acidity of the urine can be estimated from the amount of acid phosphates, since the acid reaction of urine is due mainly to these salts.

Lieblein estimates in the usual way the total amount of phosphoric acid in a definite volume of urine by means of uranium nitrate (compare p. 536). He then removes the secondary acid phosphate from an equal amount of urine by precipitation with barium chlorid, and titrates again the acid phosphates in the filtrate. Nägeli's researches have raised certain objections to this method, however.

Nemmeister[2] suggests a method to prevent the phosphate from interfering with all acidity titrations. It is a modification of Maly's[3] old procedure. Fifty cubic centimeters of urine are rendered strongly alkaline by adding 25 c.c. $\frac{N}{10}$ sodium hydroxid, the mixture is heated to boiling, 25 c.c. of a barium chlorid solution sufficiently concentrated to precipitate all the phosphoric acid are then added. The resulting mixture is shaken, and 50 c.c. are filtered through a dry filter (equal to 25 c.c. of the urine); the filtrate is colored with phenolphthalein and then titrated with $\frac{N}{10}$ sulphuric acid to the appearance of a neutral reaction. The less sulphuric acid required, the stronger the acidity of the specimen of urine.

Nägeli considers this modification of Maly's original method to be entirely unreliable (*loc. cit.*).

[1] Freund, *Centralbl. f. d. med. Wissenschaft*, 1892, p. 689; Lieblein, *Zeits. f. physiol. Chemie.*, vol. xx., parts 1 and 2.

[2] *Lehrbuch d. physiol. Chemie*, 1895, vol. ii., p. 225.

[3] *Zeits. f. anal Chemie*, vol. xv., p. 417.

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CRYOSCOPY OF THE URINE; OSMOTIC PRESSURE OR THE MOLECULAR CONCENTRATION OF THE URINE.

PREFATORY NOTES.

According to van t'Hoff, substances held in solution act like gases, and this observer has shown that the laws of Boyle-Mariotte, Gay-Lussac, and Avogadro apply to these substances as well as they do to gases. The dissolved molecules behave exactly like the molecules of a gas, which exert a pressure upon the wall of the containing vessel by their endeavor to diffuse themselves through the greatest possible space. This pressure of dissolved molecules is the osmotic pressure, and this pressure, according to van t'Hoff, corresponds to the pressure which the dissolved substance would exert as gas or vapor of the same molecular concentration and in the same space. From this it follows that osmotic pressure shows the same dependence upon the temperature as does the pressure of a gas, according to the law of Gay-Lussac, and also that solutions which contain the same number of molecules in the same volume of fluid possess the same osmotic pressure, just as gases, according to Avogadro's law, exert the same pressure when they contain the same number of molecules in the same space. If we apply the law of gases to solutions, it will also be found that just as every gram molecule or "mol" of a gas—*i. e.*, the quantity which weighs as many grams as there are units in the molecular weight—in a space equivalent to 22.4 liters at 0° C. exerts a pressure of one atmosphere, so also that solutions which contain 1 gram molecule of substance dissolved in 22.4 liters of fluid possess an osmotic pressure of one atmosphere. *Vice versâ*, if 1 gram molecule be dissolved in a liter of water, the osmotic pressure, corresponding to Mariotte's law, will be 22.4 atmospheres. From this it will also be observed that the osmotic pressure is directly proportional to the concentration. A 1 per cent. sugar solution, for example, possesses double the osmotic pressure of a 0.5 per cent. solution. These laws, however, apply only to solutions of non-electrolytes. Electrolytic substances, such as acids, bases, and salts, when in solution are partly dissociated into ions, and a comparison of the osmotic pressure with the concentration of the ions shows that in such solutions the osmotic pressure is affected by the ions just as it is by the molecules (Arrhenius, van t'Hoff). The osmotic pressure of a partly dissociated solution is consequently proportional to the sum of the undissociated molecules and of the ions. The osmotic pressure of a 0.5 per cent. solution of sodium chlorid, unlike that of a sugar solution of corresponding strength, is more than half of the osmotic pressure of a 1 per cent. solution, because dilute solutions are more strongly dissociated and contain proportionately more ions than do concentrated solutions.

Under ordinary conditions, osmotic pressure has as little visible effect as has the pressure of a gas confined within a vessel. We can, however, demonstrate the effect of osmotic pressure by carrying out certain experiments. For example, if we place a watery solution of a substance in a vessel with membranous, yielding walls which will allow the passage to water but not to the dissolved substance, and place this closed vessel in water, the dissolved molecules will become separated in proportion to the amount of water which penetrates the membrane from without. By this means the osmotic pressure becomes manifest, and there is a distention of the elastic vessel, an effect of pressure in the ordinary sense of the word. The latter phenomenon has also been designated as a result of the "attraction of the solution for water"; this is readily understood, but it does not indicate the theoretic point involved. The amount of this "attraction" is equal to the osmotic pressure, since equilibrium is not established in any experiment until the hydraulic pressure has become equal to the osmotic pressure. The osmotic pressure of a watery solution may consequently be measured hydraulically by placing the particular solution in a tube, the lower end of which is closed by a membrane that allows the water, but not the dissolved substance, to pass through it, and then placing the lower portion of this tube in a vessel of distilled water. The water passes into the tube through the pores of the membrane, and the solu-

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tion continues to be diluted until the hydrostatic pressure in the tube becomes equal to the osmotic pressure of the original solution. In this experiment the final height of the column of fluid is consequently the measure of the osmotic pressure, and the latter may be correspondingly expressed in atmospheres. This method, however, is not very practical, and the osmotic pressure of solutions is usually determined in another manner, as will presently be shown.

METHOD OF DETERMINING THE FREEZING-POINT.

In medicine we are chiefly concerned with the determination of the osmotic pressure of the blood and of the urine, more rarely with that of other fluids of the body. The best method for this purpose, and the one which is almost exclusivly employed, is the method of determining the freezing-point, or cryoscopy. The method depends upon the fact discovered by Raoult, that the lowering of the freezing-point of a watery solution, as compared with the freezing-point of distilled water, is proportional to the molecular concentration of the solution—*i. e.*, to the number of molecules contained in a unit of volume, and consequently (see above) to the osmotic pressure. We must, nevertheless, remember the fact pointed out by Arrhenius and van t'Hoff, that in partly dissociated solutions the ions have the same values as the undivided molecules in determining osmotic pressure, and consequently in the lowering of the freezing-point. It will be seen from what has been said that if one watery solution has a freezing-point of — 1° C., and another has one of — 0.5° C., then the first solution possesses double the osmotic pressure of the second. The lowering of the freezing-point is usually designated by Δ, and the minus sign is omitted. A solution which freezes at — 1° C. consequently has a lowering of the freezing-point, Δ = 1. From the previously stated fact, that a gram molecule dissolved in a liter of water has an osmotic pressure of 22.4 atmospheres, it is easy to estimate the osmotic pressure in atmospheres from the lowering of the freezing-point. Since a gram molecule dissolved in a liter of water produces a lowering of the freezing-point indicated by Δ = 1.85, it follows that a lowering of the freezing-point of 1.85 corresponds to an osmotic pressure of 22.4 atmospheres. Moreover, since the lowering of the freezing-point and the osmotic pressure vary proportionately, the correct osmotic pressure of any solution may be estimated in atmospheres from the freezing-point of the solution. The blood, for example, has an osmotic pressure of about 7 atmospheres. From a medical standpoint, however, we are more interested in the deviation of the fluid examined from the normal—*i. e.*, in a relative value—and consequently the freezing-point is not usually calculated in pressure values, but the osmotic pressure is simply indicated by the lowering of the freezing-point.

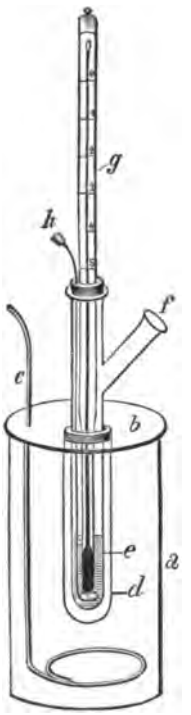

FIG. 184.—Beckmann-Heidenhain's apparatus for determining the freezing-point of a solution.

The freezing-point is usually determined by means of Beckmann's apparatus, Heidenhain's modification of which is shown in Fig. 184. The instrument is composed of the following parts: *a* is a battery jar, which has a metal cover (*b*), perforated in the middle. This jar holds the freezing-mixture, which may be stirred by the strong wire loop (*c*). The opening in the lid gives passage to the wide reagent glass (*d*), in which the actual freezing-vessel (*e*) is held in place by a perforated cork. This freezing-vessel has a lateral prolongation (*f*), the importance of which for the "inoculation of the fluid" will subsequently be pointed out. Between the vessels *d* and *e* there is an enclosed layer of air, which is for

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the purpose of allowing the cold to act gradually upon the fluid contained in *e*. The thermometer (*g*), divided into hundredths of a degree, is held in the freezing-vessel by means of a perforated cork, which also gives passage to the platinum-wire stirrer (*h*). The thermometer of the original apparatus does not possess a fixed zero, since in purely physical experiments it must be used not only for watery, but also for other solutions having an entirely different freezing-point. By a special manipulation, the details of which may be omitted, the amount of mercury in the thermometer may be increased or diminished from a specially constructed reservoir at the top of the thermometer, so that the freezing-point of the particular solvent can be brought within the scale. The freezing-point of the pure solvent is first determined, and then that of the solution. The difference obtained is the lowering of the freezing-point in hundredths of a degree.

This procedure with the original instrument of Beckmann is too complicated for clinical purposes, and, as we have to do with watery solutions only, we now usually employ a thermometer with a fixed zero (Heidenhain), which is found at the upper end of the scale, and corresponds to the freezing-point of pure distilled water (see figure). Below this point the scale is divided into hundredths of a degree. The upper end of the capillary thermometer tube is expanded, so that when the instrument is exposed to higher external temperatures the mercury may accumulate in this expansion and not break the tube. An accurately made instrument of excellent quality and adapted for clinical purposes may be obtained from the glass-instrument factory of Götze, Hertelstrasse 4, Leipsic. At the writer's request this firm has supplied an instrument with which only 5 c.c. of fluid are necessary in order to determine the freezing-point. This is a matter of great importance, since we frequently have to do with a very small quantity of fluid, particularly in the examination of the blood.

Since, in time, all thermometers vary, it is important to test the accuracy of the zero mark occasionally, even though the scale were originally absolutely correct. This is done by determining the freezing-point of distilled water with the instrument, and, when it does not agree exactly with the zero mark, adding the correction to all subsequent readings. In doing this, however, we cannot employ any distilled water at random. After a time distilled water absorbs gases, and it also takes up a certain amount of alkali from the glass container; this is sufficient to cause a distinct lowering of the freezing-point. For this reason the distilled water employed must, at least, be freshly boiled or, better still, freshly distilled. In this distillation the receiver should consist of a so-called steamed flask—*i. e.*, one which has been freed from soluble alkali by means of a current of steam.[1] If it be desired to verify not only the zero mark, which may change through no fault of the maker of the instrument, but also the accuracy of the scale, this may be done by also determining the freezing-point of a pure 1 per cent. sodium chlorid solution. According to Hamburger, the freezing-point of such a solution is 0.589. If the instrument be correct, this is the value which should be found after making the necessary correction for the zero mark.

In the determination of every freezing-point, care must be exercised to see that small particles of mercury are not sticking to the sides of the upper expanded portion of the thermometer, since, if this were the case, an error would arise from the exclusion of this part of the mercury. For this reason the expansion should be examined with a lens before every determination, and if separate particles of mercury are found they must be united with the mercurial column, either by shaking or by beating the instrument.

In determining a freezing-point the jar (*a*) is first filled with a freezing-mixture. In order to obtain correct results, it is important that the freezing-mixture be correctly prepared. It should not be too cold. The best mixture, according to Hamburger,[2] is one of 3 parts of finely cracked ice or snow with 1 part of sodium chlorid; this is placed in the jar, and sufficient water added to make the temperature about — 3° C. If the temperature rises in the

[1] See Cohen, *Vorträge über physikalische Chemie*, 1901, p. 10.
[2] *Osmotischer Druck und Jonenlehre*, 1902.

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course of the experiment, a fresh amount of ice and sodium chlorid in the given proportion should be added. The fluid to be examined is now placed in the freezing-tube, and the thermometer and the platinum stirrer subsequently introduced. Care must be taken that the mercury reservoir of the thermometer is completely surrounded by the fluid, so that it is entirely submerged, and does not come in contact with any portion of the freezing-tube. The freezing-tube is then inserted into the reagent glass in the cover of the battery jar so that it is surrounded by a uniform layer of air. The fluid in the freezing-tube must be kept in constant motion by gentle, but interrupted, movements of the platinum stirrer (*h*). The fluid must not splash. The mercury soon commences to sink in the thermometer, leaves the upper expanded portion, enters the capillary tube, and quickly falls to a point more or less below the freezing-point (excessive cooling), and then suddenly rises to a definite point at which it remains stationary for some time. This point is the freezing-point of the fluid examined, and is read off. At this time needles of ice commence to form in the fluid, and it gradually becomes solidly frozen. When this has happened, the mercury commences to descend again. This complete freezing should not be waited for, however, but the determination should be repeated immediately after the mercury has ceased to ascend, by taking out the freezing-vessel, holding it in the hand and stirring the fluid constantly until the mercury commences to ascend, when the freezing vessel is again introduced into the reagent glass (*d*), and the freezing-point again determined as before. In warming the freezing-vessel for the purpose of repeating the determination all of the crystals should not be melted—*i. e.*, the temperature must not reach 0° (since the crystals consist of pure water)—because errors would arise if the fluid were subsequently "inoculated" with small pieces of ice (see below). When three determinations have been made, one immediately after the other, the average of the three is taken as the final result.

In order to determine correctly the freezing-point it is important to avoid excessive cooling (see above), since large masses of ice will form, and as these consist of pure water a more concentrated solution remains, which gives a lower freezing-point, corresponding to its higher osmotic pressure. The error of excessive cooling is avoided, first of all, by compounding the freezing-mixture so that its temperature is not too low. The safest way would be to compound it so that its temperature would be but a few tenths of a degree below the expected freezing-point, which could be approximately determined in a preliminary experiment. This takes up too much time, however, since the cooling would proceed so slowly, and in addition it makes the necessary recognition of the descent and ascent of the mercury much more difficult. If the temperature of the freezing-mixture be — 3° C., instead of the very low temperatures so commonly employed previously, the chances of excessive cooling are considerably reduced. An additional means of avoiding excessive cooling consists of the introduction of a minute piece of ice into the fluid through the lateral prolongation at the moment when the mercury falls below the zero-point. In physical chemistry this is referred to as "inoculation" of the fluid with a piece of ice. This accelerates the formation of ice, and consequently diminishes the danger of excessive cooling. This would seem to dilute the solution, and consequently lead to false results; but this is not the case when the manipulation is correctly performed, because the piece of ice cannot melt if the temperature be below zero. This would be possible, however, if the fluid were warmed in the hand to the zero point in making the second and third determinations. This must be avoided, as has been previously pointed out, and no error will then result if the fluid be repeatedly "inoculated." It will readily be understood that the fluid is useless for subsequent determinations after it has become thoroughly melted. In the writer's experience he has found that "inoculation" is unnecessary if the temperature of the freezing-mixture be not more than 3° C. below the expected freezing-point, since in this manner the excessive cooling is avoided.

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EMPLOYMENT OF CRYOSCOPY FOR THE STUDY OF THE RENAL FUNCTIONS.

Since the determination of the freezing-point furnishes us with a simple and clinically applicable method of estimating the osmotic pressure, and consequently the molecular concentration of a fluid, this method of examination has been applied to the urine and to the study of the functions of the kidney. By this method it has been attempted to determine not only the total amount of work done by both kidneys, but also, by the practice of ureteral catheterization or by Luys' method of separation of the urines (see p. 458 *et seq.*), to compare the functions of the two kidneys. Disregarding the excretion of water, which is doubtless a renal function and not to be regarded simply as a filtration, it is clear that the greater part of the work of the kidney consists of excreting a fluid of high osmotic pressure from the blood, which has a much lower molecular concentration. An expenditure of energy is necessary for this purpose, and the production of a urine of high osmotic pressure is consequently to be regarded as an accumulation of potential energy. Should we assume, as is frequently done, that this production of a fluid of higher osmotic pressure from the blood represents the entire work of the kidney, the daily amount of work done by both kidneys would be calculated by $W=Q(\Delta-\delta)$, in which Q is the daily excretion of urine, Δ the freezing-point of the urine, and δ that of the blood. The result may be designated as the daily osmotic capability of the kidney. It should be remembered, however, that this measure of the renal functions is neither complete nor exact. It is not complete, because it neglects the work done by the kidneys in excreting water, and also because it is evident that by the excretion of a diluted urine, which has a lower osmotic pressure than that of the blood, and in which the kidneys create no osmotic pressure whatever, the kidneys still perform work that is not apparent as osmotic pressure.[1] It is not exact, since we do not know that different molecules require the same amount of energy for their excretion. Although the difference in the molecules is immaterial for the calculation of the potential energy stored up in the urine, it is not only plausible, but also possible, that the excretion of a molecule of sodium chlorid requires a different expenditure of energy than is necessary for the excretion of a molecule of urea. The difference probably exists in variations in the amount of heat evolved, and is not at all apparent in the osmotic pressure of the urine. The

[1] Should we assume that the production of an osmotic pressure in the urine exceeding that of the blood is the only work done by the kidneys, as is frequently stated, we should arrive at the paradoxic and ridiculous conclusion that kidneys which furnish a diluted urine perform no work at all, or even a negative amount of work (the latter if $\Delta-\delta$ is negative), while, on account of the markedly increased quantity of water in the urine in these cases, a particularly large amount of work is perhaps represented.

The writer's opinion, that the excretion of water by the kidney is a specific function of this organ, will possibly be objected to on the ground that this excretion can be accounted for by assuming that the secreting membranes of the kidney are extremely permeable to water, and that it is not at all necessary for the kidney to supply energy for a simple filtration. Such a permeability to water, however, must be something quite specific, since a similar permeability is found nowhere else in the body. We also have reason to believe that this excretion of water is an active function, since under physiologic conditions it is always accurately adapted to the needs of the organism. A further proof of the activity of the kidneys in excreting water is furnished by the hypertrophied kidneys of beer drinkers, which, by their differences from the kidneys of wine drinkers, demonstrate that the hypertrophy is due to the effect of water and not to that of alcohol. The same thing is still more strikingly shown by the renal hypertrophy acquired in diabetes insipidus. If the activity of the kidneys were not increased under these conditions, they would not become hypertrophic.

Another reason for the writer's opinion that the so-called osmotic energy of the kidneys is no accurate measure for the work done by these organs is the fact that the ionization of the urinary constituents varies with the concentration of the urine. Since part of this ionization occurs after passing through the renal epithelium (in case a concentrated urine becomes more dilute), this portion of the osmotic pressure dependent upon ions, or at least some of it, cannot be reckoned as work done by the kidney.

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majority of the works upon cryoscopy of the urine do not regard this point at all, and consequently deduce far-reaching conclusions from the determination of the freezing-point of the urine which are by no means justified.

The utilization of the results of cryoscopy for the determination of the functional activity of the kidneys is also complicated by difficulties which are encountered in the study both of the mixed twenty-four-hour urine and of the separately collected urine from each kidney. It is difficult to deduce conclusions from the freezing-point of the mixed twenty-four-hour urine, since this freezing-point is subject to great variations under normal conditions. It is not unusual for the lowering of the freezing-point of the twenty-four-hour urine to exhibit a physiologic variation of from 0.9 to 2.7° C., and still greater variations may be encountered under perfectly normal conditions, dependent upon the ingestion of food and water. If an individual drink large quantities of water, the difference between the freezing-point of the urine and that of distilled water may amount only to 0.11° C. (Koppe). In nursing infants, the osmotic pressure of the urine is always less than that of the blood, and the lowering of the freezing-point may vary between 0.087 and 0.455° (Koppe). In any individual case the freezing-point may vary from day to day. Since there is no normal freezing-point for normal urine, we can draw conclusions in reference to the renal function in pathologic cases only when repeated examinations have shown the freezing-point to be constantly too high or too low, and when this extreme variation cannot be accounted for by anomalies in the ingestion of food or water. These difficulties are by no means overcome by basing the calculation upon the daily osmotic energy of the kidneys determined by $W = Q\ (\Delta - \delta)$ instead of upon the lowering of the freezing-point (Δ) or by employing the product of the daily excretion and the freezing-point of the urine ($Q\ \Delta$). The formula $W = Q\ (\Delta - \delta)$ is more exact than the freezing-point alone, since it not only indicates the molecular concentration, but also measures the osmotic energy expended by the kidneys within twenty-four hours, while the product $Q\ \Delta$ is a measure for the number of molecules excreted in a similar period of time. The previously emphasized objections to utilizing the freezing-point also exist here, since under normal conditions the number of excreted molecules and the product $Q\ \Delta$ vary just as much as does the molecular concentration or the freezing-point. These results may consequently be employed for diagnostic purposes only when they are extreme, when they extend over long periods of time, and when they cannot be explained by an abnormal diet.

The most pronounced pathologic changes in the molecular concentration of the urine, in the daily osmotic energy of the kidneys, and in the product $Q\ \Delta$ are encountered in nephritis, and particularly in the uremia which frequently results. In these cases the retention of urinary solids is very frequently manifested by an abnormally low molecular concentration and a consequent abnormally low daily excretion of molecules. Every improvement of the condition is indicated by an increase of both. According to Strauss, the chronic parenchymatous nephritis cases produce a more marked diminution of the daily excretion of molecules than do the interstitial, since in the latter cases the marked lowering of the freezing-point is more than compensated for by the increased quantity of urine excreted. In diabetes mellitus the osmotic pressure of the urine and the daily excretion of molecules are, of course, greatly increased by the amount of sugar in the urine.

The utilization of cryoscopy in determining the functional activity of a single kidney, the urine from which is obtained either by ureteral catheterization or by Luys' urinary separator, is accompanied by difficulties equally as great as those encountered in studying the mixed twenty-four-hour urine. The object of such examinations is to determine whether one kidney is functionating normally, and whether it will be able to do all the work after the removal of the diseased kidney. The conclusions drawn from the separate cryoscopic examinations of the urines of both kidneys in such cases, which have given rise to a large amount of literature contain a great many sources of error, only the most important of which will be given. First of all, it by no means follows that the kidney

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remaining after the extirpation will do the same amount of work as before. Rather, if the diseased kidney still functionated to a certain extent, it is quite possible that the remaining kidney will respond to the increased excretory stimulus and compensate for the defect, even if it apparently did too little work before the extirpation. Upon the other hand, if the function of the healthier kidney be impaired, and yet sufficient to meet the needs of the organism when aided by the diseased kidney, it is possible that it may be insufficient when called upon to do the entire amount of work. These possibilities are not even mentioned in many of the publications upon the subject. Another frequent error is the false supposition that the freezing-point of the urine is the only measure of the functional activity of the kidney. As we have already seen, this is not the case, and the supposition of Kümmel, that a freezing-point of the urine below —1° C. indicates insufficiency of the viscus, is purely arbitrary.[1] Again, in order to reach more accurate conclusions, the quantity of urine excreted must also be considered. This cannot be correctly done, however, since it is not possible to collect the twenty-four hours' urine from a single kidney. Great caution is also necessary when the results of the cryoscopic examination of the separate urines are employed for the purpose of comparing the functions of the two kidneys. This examination is usually made by catheterizing first one and then the other ureter, and then comparing the freezing-points of the two urines. Although the excretion of the urines is approximately simultaneous, it is not exactly so; and, since the excretion of the urine is subject to sudden temporary variations, the comparison is not accurate. Even if the two urines were excreted simultaneously, as is the case when Luys' separator or simultaneous catheterization of the ureters is employed, we should be by no means sure of obtaining comparative values, since experiments upon animals prove that the excretions of the two kidneys are not always alike. It is therefore justifiable to assume a difference in function only when repeated examinations always demonstrate similar variations. The fact that, in the customary utilization of the cryoscopic findings, the surgery of the kidney is usually correct, in spite of these errors, may be partly due to the fact that the indication for the operation is not based exclusively upon these results, but also the chemical and microscopic examination of the separate urines.

In view of the objections to the utilization of the cryoscopic findings, we are justified in raising the question as to whether this new method of urinary examination is of more value than a very old procedure dealing with similar questions —namely, the determination of the specific gravity. It is quite clear that the determination of the osmotic pressure or of the freezing-point is not equivalent to the determination of the specific gravity. The determination of the freezing-point furnishes a relative idea of the number of molecules excreted, irrespective of their quality, and the result of such a determination is theoretically independent of the specific gravity of the urine, since molecules of low and high specific gravity exert exactly the same influence upon the freezing-point. The specific gravity, on the contrary, is dependent not only upon the number of molecules dissolved in a given volume of urine, but also upon their chemical composition or weight. With a single exception, presently to be mentioned, this difference, however, is more theoretic than practical. The quality of the excreted molecules in different urines is subject to such slight variation that a marked parallel usually exists between the lowering of the freezing-point and the specific gravity; and if the specific gravity of the urine of a patient rises or falls we may rest assured that the lowering of the freezing-point or the osmotic pressure has undergone a similar variation. We are, consequently, just as much justified in drawing conclusions in reference to the work done by the kidney from the specific gravity of the urine as from the height of the osmotic pressure. One exception, however, must be borne in mind: If the urine contain proteid, this substance may be responsible for a considerable part of the specific gravity,

[1] Such conclusions would be more justifiable if all fluids were interdicted before the examination of the patient, in which case the approximately healthy kidney would always excrete a concentrated urine with a marked lowering of its freezing-point.

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since it is heavier than water, but it has no perceptible influence upon the osmotic pressure or the freeezing-point. Since the proteid molecule is very large and heavy, quite a small number of such molecules may cause a rise in the specific gravity without appreciably influencing the freezing-point. With an albuminous urine we should consequently not be justified in attributing a high specific gravity to increased renal activity, particularly since the proteid is not secreted by the kidney, but is an indication that the filtering action of the kidney has been impaired by inflammation. With this exception, however, the writer maintains that the determination of the specific gravity gives us the same indication of the work done by the kidney as does the determination of the freezing-point. In order to draw similar conclusions from the specific gravity of albuminous urine, it is necessary simply to remove the albumin and then utilize the specific gravity of the remaining fluid. Just as in the estimation of the total work done by the kidneys from the determination of the freezing-point, the urine examined must be a portion of the mixed twenty-four hours' elimination if correct conclusions are to be obtained. In doing this we may calculate a product whose significance completely corresponds to the significance of the cryoscopic product Q Δ (see above), by multiplying the last two figures of the specific gravity, carried to the third decimal place, by the amount of urine secreted in twenty-four hours. The number so obtained gives a relative idea of the amount of urinary solids secreted in twenty-four hours. We may, however, easily obtain absolute quantities, as stated upon page 452, by multiplying the last two figures of the specific gravity by 2.33, and then multiplying this product by the daily urinary excretion expressed in liters. The resulting figure is the approximate number of grams of urinary solids excreted in twenty-four hours. The writer believes that this number is just as good an indication of renal activity as is the number of molecules excreted daily, provided that any proteid present be removed before the specific gravity is determined. This number is, of course, markedly influenced by the quantity of ingested food and water, so that the normal is hard to determine, and the same difficulties are encountered in drawing accurate conclusions as have been previously mentioned in reference to the determination of the number of excreted molecules. In an entirely analogous manner the specific gravity of the urines (after the removal of proteid, if present) obtained by ureteral catheterization or Luys' separator (see p. 458 *et seq.*) may be utilized in place of the osmotic pressure for the critical study of the urine of each kidney; but, owing to the impossibility of obtaining the total twenty-four hours' urine, this procedure is open to the same objection previously mentioned in our consideration of osmotic pressure. The writer must also take this occasion to claim that cryoscopy has not materially aided our diagnostic ability in reference to the critical study of renal activity, since all of the facts learned by ureteral catheterization could also have been discovered by means of the specific gravity. Cryoscopy accidentally became popular just at that time and was given preference over the older method from an exaggerated idea of its value, just as all new methods are usually supposed to be an advance over those formerly employed. In addition to the fact that it is not necessary to remove proteid, it must nevertheless be admitted that cryoscopy possesses one distinct advantage, particularly in the study of the separate urines, in that it requires a smaller amount of urine than is necessary for the determination of the specific gravity. Nevertheless, the chief advantage of the introduction of the new method has been that it has caused us to study these questions more carefully than was done by means of the specific gravity, since the latter method had not the charm of novelty. We shall subsequently learn that the case is quite different with reference to the cryoscopy of the blood, which is of special significance for general pathology and diagnosis.

How closely the conclusions obtained by means of the determination of the freezing-point of the urine correspond to those which the practical physician may deduce much more readily from the specific gravity is best shown by the statement of G. Fuchs,[1] that in a urine free from proteid and sugar the lowering of the

[1] *Zeits. f. angewandte Chemie*, 1902, p. 1072.

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freezing-point may be empirically calculated by multiplying the last two figures of the specific gravity, carried to the third decimal place, by 0.075° C.

The far-reaching calculations and conclusions (based upon the freezing-point of the urine) which Koranyi [1] and others have formulated in regard to the freezing-point of the blood, as well as to the amount of sodium chlorid contained in both fluids, will not be discussed, since the writer regards their fundamental principles as hypothetic, and in many respects erroneous.

SEDIMENTS AND TURBIDITY OF THE URINE.

EXAMINATION OF URINARY SEDIMENTS; SEDIMENTATION; FILTRATION; CENTRIFUGATION; MICROCHEMICAL REACTIONS.

Both urinary sediments and turbidity may be frequently enough recognized and characterized, without any microscopic examination, by their physical and chemical behavior. But a microscopic examination is necessary in most cases, especially for the differentiation of organized sediment. If the sediment is very abundant, a drop of the cloudy urine under the microscope will be sufficient; but generally the sediment is so scant that it is advisable to isolate and collect it by sedimentation, filtration, or centrifugation.

In concentrated urines with uratic sediments the microscopic examination of other constituents, and particularly of the organized elements, is frequently rendered very difficult by the presence of urates. In such cases the urates should be dissolved by heating the urine slightly and adding enough water to hold the urates in solution upon cooling. If this cannot be accomplished, which may be the case if uric acid has also been thrown down, both substances may be brought into solution by rendering the urine alkaline by the addition of a concentrated solution of soda. A borax solution may also be employed for the same purpose (see below). Such a solution of the precipitated urates and uric acid may be necessary not only for the direct microscopic inspection of the remaining deposits, but also as a preliminary to sedimentation, filtration, or centrifugation. In certain cases sediment of phosphates or of carbonates may similarly interfere with the microscopic examination. These sediments may be readily dissolved by acidulating the urine with hydrochloric acid. A reference to the important experiments of Moritz will be found in the supplement of this work.

For Sedimentation.—A tall pointed glass is filled with the urine, and the sediment is examined under the microscope just as soon as enough has settled to be plainly visible at the bottom of the glass. This usually requires a few hours. The sediment is picked up with a pipet while the upper end is closed with the finger. By slightly decreasing the pressure of the finger a certain amount of sediment is allowed to enter the pipet. It is then closed again, removed, and carefully dried, while the finger pressure is kept constant. A drop of its contents is then allowed to flow upon a glass slide by slightly releasing the finger.

Strassburger has recently recommended diluting the urine with two parts of alcohol, in order to hasten sedimentation by diminishing the specific gravity of the fluid. But alcohol will precipitate proteids, so that this procedure can be

[1] *Zeits. f. klin. Med.*, 1897

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recommended only where the amount of these bodies is very slight or where the presence of an amorphous precipitate will not produce confusion, especially for the microscopic demonstration of tubercle bacilli or other bacteria.

The urinary sediment changes so quickly that it is important to examine it as soon after settling as possible.

The various constituents of a sediment differ in their specific gravities; hence, in many cases it is advisable to select specimens for examination at varying depths of a sediment. With practice and care this selection can be made while picking up one specimen.

To prevent bacterial decomposition, so prone to occur during the summer months, it is a good plan to add to the urine one-fifth its volume of chloroform water (1:200) or a small piece of camphor, and to select a cool place for the sedimentation. To avoid obscuring the examination of organized sediments by the formation of heavy urate sediments in very concentrated or very acid urines, we may add to the urine one-fifth to one-third its volume of a saturated (1:17) borax solution. This solution will not coagulate proteids, and at the same time will act antiseptically. If the sediment is very scanty the filtration method may be employed. As large an amount of the urine as possible is filtered, and the residue upon the filter is picked up with the ordinary pipet while only a few drops of the urine remain.

FIG. 185.—Electrical centrifuge.

The centrifuge[1] is now employed in practically all laboratories for obtaining the sediment. With it the sediment may be obtained for examination within a few minutes and from very small specimens of perfectly fresh urine. [Fig. 185 represents a very satisfactory form of electrical centrifuge, to be connected to the ordinary street current available in all large towns. There are numerous other electrical centrifuges upon the market, some of which are thoroughly trustworthy, others unreliable. Where there is no street current available, practitioners may find the ordinary hand or water centrifuge a very convenient addition to their laboratory equipment. They are both less expensive.—ED.]

Many chemical reactions which reveal the nature of certain sediments can be performed macroscopically and also observed under the microscope. In the latter case—*e. g.*, to determine the nature of unfamiliar crystals—a drop or two of the agent is allowed to run under one edge of the cover-glass, or perhaps sucked under with the help of a piece of filter paper at the other edge.

[1] Stenbeck, *Zeits. f. klin. Med.*, vol. xx., 457, 1892. Litten, *Deutsch. med. Woch.*, 1891, p. 23.

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The amount of the sediment is best estimated by v. Posner's method of estimating the amount of pus present (p. 568). (See p. 564 *et seq.* in regard to the preservation of organized sediment.)

NON-ORGANIC CRYSTALLINE AND AMORPHOUS SEDIMENTS AND MIXTURES.

THE URATES.

When the urine cools, urates may settle to the bottom, especially if the sample is concentrated, scanty, or strongly acid—*e. g.*, the urine of congestion and of fever, less often the urine of nephritis. The sediment will then present a fairly characteristic appearance, clay-colored, reddish yellow, brick red, or rose red (sedimentum lateritium). Sometimes it adheres to the wall of the urine glass as a thin coating. Unlike all other sediments, it dissolves extremely readily even with very gentle heating, and so can be accurately recognized. The addition of acid will also dissolve urate sediments with a gradual separation of uric acid crystals. The addition of alkalies also dissolves the precipitate particularly easily, and often with the separation of phosphates. The ordinary urate sediments consist of a mixture of sodium, potassium, calcium, magnesium, and ammonium urates. Sodium urate predominates. With the exception of ammonium urates, the urates appear only in acid urine. They are formed by a double rearrangement between acid sodium phosphate and the neutral urates held in solution, from which acid urates are formed, which are not very soluble and become precipitated. Aside from these chemical processes, the cooling of the urine favors the separation of urates. That the mere cooling of the urine alone will not, however, produce the separation is shown by the fact that the sediment forms sometimes only a considerable time after the cooling, and also by the fact that a urate sediment usually does not entirely dissolve until the urine is heated to a temperature above that of the body.

Urate sediments often contain crystals of pure uric acid, which also arise from the double rearrangement mentioned above.

The peculiar pronounced brick- or rose-red color of some urate sediments arises from uroerythrin, a pigment as yet little understood (compare p. 478). It seems to be formed to excess in certain febrile affections (acute rheumatism, pneumonia).

If a urine with a urate sediment is decomposed by ammoniacal fermentation, the sediment will be partly changed to acid ammonium urate. The latter is the only urate sediment which occurs in alkaline urine (compare p. 558).

Under the microscope urate sediment appears as fine amorphous granules. If acetic acid is added, characteristic crystals of uric acid may be seen to shoot out from these granules (Fig. 186). Ammonium urate presents characteristically shaped crystals (Figs. 189, *b*). Uratic sediments respond to the murexid test. (See p. 556, under Uric Acid.)

As already mentioned, the concentration of the urine and its acidity are particularly important in the formation of urate sediments. If, therefore,, despite a normal specific gravity, a urine very readily deposits

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a sediment of amorphous urates, it does not signify, as was formerly supposed, an increased production of uric acid, but usually merely an increase in the acidity of the urine—*i. e.*, an increased amount of the acid sodium phosphates.

URIC ACID IN THE SEDIMENT.

Crystals of uric acid occur in the urine at the same time with amorphous urate sediments, and sometimes without any separation of urates, mostly as a fine crystalline precipitate, often adherent to the walls of the vessels (Fig. 186). The beautifully shaped, sparkling, shining crystals of red-brown color can often be recognized by the naked eye. Their appearance is usually very characteristic. In case of doubt the *murexid* test may be performed with a few small crystals. The latter are heated with dilute nitric acid upon a porcelain plate or dish. Solution takes place with effervescence. After evaporation there remains a reddish residue, which turns a beautiful purple red upon the addition of a little diluted ammonia, due to the formation of ammonium purpurate

FIG. 186.—The more common forms of uric acid crystals (after Bizzozero and Scheube).

(murexid). The addition of potassium hydroxid will change the color to violet. The urates also react to the murexid test. The most frequent shapes which the uric acid crystals present under the microscope are depicted in Fig. 186.

If uric acid crystals are observed in the sediment at the same time as amorphous urates, they have the same significance as the latter. They may be found in any concentrated urine. Rapid precipitation of purely crystalline uric acid without any amorphous urate in a comparatively abundant urine shows that it is strongly acid. Such a condition has nothing to do with the so-called uric acid diathesis (gout and uric acid calculus). Moritz has demonstrated that every individual crystal of uric acid has an albuminous ground-substance.

CALCIUM OXALATE IN THE SEDIMENT.

These crystals occur both in pathologic and in normal urine. The sediment is usually scant, and recognized only when examined microscopically. An abundant oxalate sediment is often noticed after the ingestion of foodstuffs rich in oxalic acid (fruit, especially tomatoes,

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etc.), sometimes in diabetes mellitus, and finally in *oxaluria*. The last-named disorder occurs chiefly in dyspepsia and nervous disorders. It is questionable if it is an independent anomaly of metabolism (Fig. 187).

The formation of oxalate calculi will, of course, be favored by such a condition.

According to Fürbringer, calcium oxalate is in all probability a normal urinary constituent, held in solution in the urine by the acid sodium phosphate. If for any reason the acid reaction is reduced so that the acid phosphate becomes converted to a neutral salt, calcium oxalate becomes precipitated. Ordinarily the double change between neutral urate and acid phosphate mentioned in connection with the urate sediments is the cause of the reduction of the acid reaction; and then, besides the urates, calcium oxalate precipitates. The separation of the oxalate usually takes place very slowly, and it therefore generally occurs as well-formed crystals (Fig. 187). They usually present a very characteristic octahedral type (envelopes); but other varieties also occur (see Fig. 187, note to Fig. 188), which cannot, however, be recognized immediately from their shape.

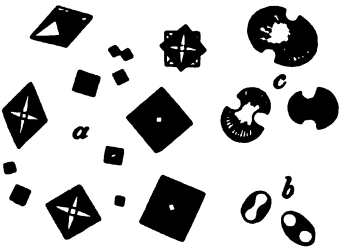

FIG. 187.—Crystals of calcium oxalate: *a*, So-called envelopes; *b* and *c*, rarer forms (after Scheube).

From the way in which calcium oxalate crystals separate, it is plain that they may occur in faintly acid, in neutral, and in faintly alkaline urine. Calcium oxalate crystals are characterized not only by their shape and insolubility in acetic acid, but by the fact that they are soluble in hydrochloric acid.

The foregoing explanation of the separation of calcium oxalate crystals makes it evident that their appearance in the sediment does not justify the assumption of oxalic acid any more than urate sediment necessarily implies increased uric acid. "Oxaluria" is often diagnosed without sufficient reasons. An increase of the oxalic acid elimination can be determined only by a quantitative urinalysis.

SEDIMENTS OF THE EARTHY PHOSPHATES AND CARBONATES AND OF AMMONIUM URATE.

They are usually light in color, because they are less inclined than the urates to absorb the urinary pigments, and because their separation does not depend so much upon the degree of concentration as upon the reaction of the urine.

1. **Amorphous Earthy Phosphates and Carbonates.**—Normal and basic phosphate and carbonate of calcium and magesium occur as granular amorphous masses, and may be present in every alkaline urine, particularly if the alkalinity is due to a fixed alkali. These salts are also precipitated if the urine is artificially rendered alkaline, or if a faintly acid, neutral, or faintly alkaline urine is boiled, the acid combinations in which calcium and magnesium phosphate and carbonate

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are dissolved in the urine being changed to basic combinations by boiling. Their precipitation produces a turbidity which, unlike the precipitate of proteid from heating, is soluble in dilute acids. The carbonates when dissolved by dilute acids give off CO_2, a peculiarity which will distinguish them from the phosphates.

Amorphous phosphates and carbonates compose the chief part of the urinary sediment after the diminution of acidity following gastric lavage or vomiting. Sometimes they are separate even in the bladder. Hypersecretion of the acid gastric juice with delayed gastric motility causes sometimes a permanent turbidity, and sometimes a turbidity only after meals. In either case it is due to a precipitation of the phosphates and carbonates, probably because the secreted hydrochloric acid is not completely or, at least, not quickly enough reabsorbed and the urinary acidity is proportionately diminished. This is an explanation of the so-called phosphaturia in nervous individuals. The name is incorrectly applied, of course, and should be limited strictly to those cases where the elimination of phosphates is quantitatively increased. In very mild cases of such hyperchlorhydria, turbidity appears only after the urine has been boiled.

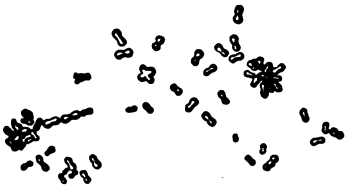

FIG. 188.—Indistinct crystalline sediment (dumbbell crystals) of calcium carbonate. Similar crystals are formed by calcium oxalate and calcium sulphate (after Funke).

FIG. 189.—*a*, Crystals of ammoniomagnesium phosphate (triple phosphate); *b*, crystals of ammonium urate (after Neubauer and Vogel).

A crystalline precipitate of calcium carbonate sometimes appears as a sandy powder mixed with the amorphous phosphate sediments. Microscopically, it consists of spheric or dumbbell-shaped formations (Fig. 188), which dissolve in acetic acid with the evolution of gas, and so can be distinguished from similar formations of calcium oxalate (see p. 557).

2. **Ammoniomagnesium Phosphate (Triple Phosphate) and Ammonium Urate.**—These are combined with the amorphous phosphate and carbonate sediment if the alkaline reaction of the urine is entirely or partially due to the formation of ammonium carbonate within the urinary passages from the urea, or, after voiding the urine, from bacteriologic fermentation. In such a condition triple phosphate will compose the major part of the sediment. The large characteristically shaped prisms, the so-called "coffin lids" (Fig. 189, *a*), are readily recognized under the microscope. The ammonium urate which so frequently accom-

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panies the triple phosphate crystals (Fig. 189, *b*) is the only urate precipitated in an alkaline urine. This salt forms dark-colored spheres with projecting spines, "morning-star or thorn-apple balls." Crystals of ammonium urate dissolve in acetic acid with a gradual formation of uric acid. Ammoniomagnesium phosphate crystals are also readily soluble in acetic acid. Although both these crystalline forms occur chiefly in ammoniacal urine, they also appear in faintly acid or amphoteric urine if an ammoniacal fermentation has commenced.

FIG. 190.—Other forms of triple phosphate crystals (after Peyer).

3. **Dicalcium phosphate** (neutral or simple acid calcium phosphate) is a rather rare sediment formed in faintly acid or amphoteric urine as microscopic prismatic or wedge-shaped prisms grouped as rosets (Fig. 191). These crystals are soluble in acetic acid. The rosets are frequently only indistinctly developed.

4. **Crystalline Trimagnesium Phosphate.**—In very rare cases there have been observed in alkaline urine large, flat, strongly refractive masses which consist of elongated rhombic plates with angles of 60° and 120° (chiefly when the alkalinity of the urine is due to removal of the gastric contents). These are trimagnesium phosphate (normal magnesium phosphate).

OTHER INORGANIC SEDIMENTS OR TURBIDITIES (RARE).

Gypsum (calcium sulphate) occurs very rarely, and only in the sediment of strongly acid urine. Its crystals are represented in Fig. 192 (compare Fig. 188).

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They are insoluble in ammonia, alcohol, and acetic acid; slightly soluble in hydrochloric acid, nitric acid, and hot water. Their aqueous solutions may be precipitated with barium chlorid, and the resulting precipitate is insoluble in hydrochloric or nitric acid. Ammonium oxalate also removes them from their aqueous solutions, and the resulting precipitate is insoluble in acetic acid, but soluble in nitric and hydrochloric acids.

Cystin.—Cystin occurs in the urine only very rarely (so-called cystinuria).[1]

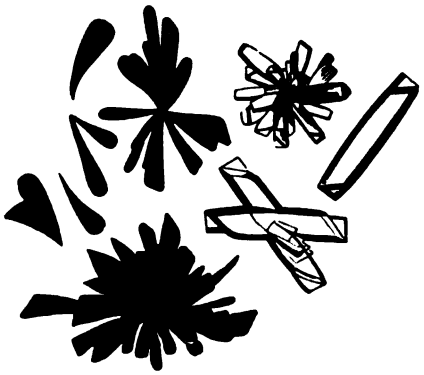

FIG. 191.—Crystals of dicalcium phosphate from amphoteric urine (after Neubauer-Vogel and Ultzmann-Hofmann).

This is a peculiar metabolic anomaly which leads to the formation of cystin and diamins. According to recent investigation, it is probably due to some peculiar intestinal mycosis, because these substances are also found in the intestinal contents. Cystin usually separates from an acid urine within the urinary passages in characteristic hexagonal plate-like, colorless crystals (Fig. 193) (cause of cystin calculi).

Uric acid also separates in similar crystals, although quite exceptionally;

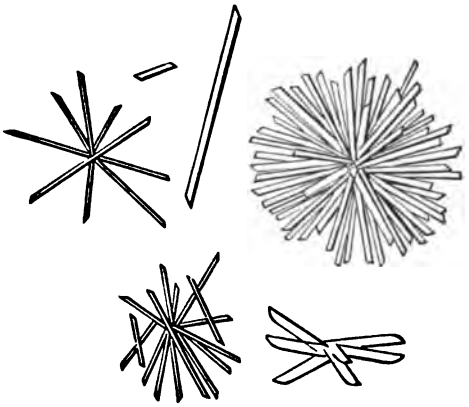

FIG. 192.—Crystals of gypsum (after Neubauer and Vogel).

hence cystin and uric acid may be confounded. The crystals of cystin are, however, perfectly colorless. To differentiate doubtful crystals chemically, ammonium hydroxid is added to the sediment. Cystin will be dissolved. The filtrate is

[1] See Stadthagen and Brieger, *Berlin. klin. Woch.*, vol. xxvi., p. 344, 1889, and Udránsky and Baumann, *Zeits. f. physiol. Chemie*, 1890.

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then acidulated with acetic acid or, better still (Salkowski, Drechsel), allowed to stand exposed to the air for some time until the ammonia has escaped. Cystin will then be again precipitated in hexagonal disks. Uric acid is only very slightly soluble in ammonia, and also differs from cystin in being only slightly soluble in hydrochloric acid.

Tyrosin and Leucin.—Tyrosin very rarely appears as a sediment. It is recognized by its characteristic crystals (compare p. 498 and Fig. 173, *b*).

Leucin is a still rarer sediment than tyrosin, and is then always associated with the latter (compare p. 498 and Fig. 173, *a*).

Xanthin, although a normal urinary constituent, is very rarely observed as a sediment, and then under entirely unknown conditions. It forms whetstone-shaped crystals, which are differentiated from those of uric acid by being readily soluble in ammonia. Further, if xanthin crystals are evaporated with moderately concentrated nitric acid over a water-bath, they leave a yellowish white residue, which turns to an intense yellow after careful heating over a small flame; then the addition of potassium hydroxid will change the color to a yellowish red, which will become more deeply colored when freshly heated, and which will finally turn to a violet red when the potassium hydroxid is evaporated (Strecker). This test must not be confused with the murexid reaction (p. 556).

Cholesterin.—Cholesterin crystals have been found occasionally, and sometimes in considerable quantity (cholesterinuria), in the urine in diseases of the

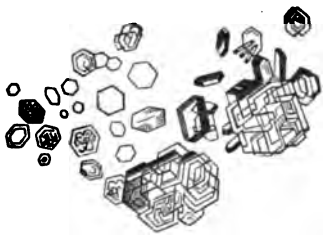

FIG. 193.—Crystals of cystin (after Neubauer and Vogel).

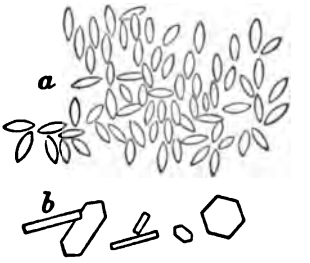

FIG. 194.—*a*, Xanthin crystals; *b*, crystals from the solution of xanthin in hydrochloric acid (after Neubauer and Vogel).

urinary passages (inflammation of the bladder, pyelitis, echinococci, chyluria, and nephritis). This occurrence is, however, extremely rare.[1] Cholesterin is supposed to be derived from the epithelium (Fig. 211, *b*).

Hematoidin Crystals (*Bilirubin*, compare Fig. 211, *d*.[2]).—Hematoidin crystals occur, although very rarely, in the urine of hemorrhagic nephritis. Bilirubin crystals, on the contrary, are not infrequently precipitated in sediment of markedly icteric urine when cool, particularly if it is strongly acid or artificially acidulated. They can be easily recognized by their yellowish-red color, by their solubility in alkalies and chloroform, and by their reaction to Gmelin's test (p. 472 *et seq.*), under the microscope.

Indigo.—If the indican is increased in the urine, indigo may separate as pointed or rhombic crystals. These may either be found in the sediment or form a scum on the surface. They dissolve readily with a blue color in chloroform (pp. 454 and 475).

Melanin.—Melanin separates in rare cases as fine amorphous granules. It usually remains, however, in solution (p. 477).

Hemoglobin.—This may be precipitated, while hemoglobin is still in solution, as a sediment of amorphous cakes or cylinders in hemoglobinuria (compare pp. 453 and 469).

Fat.—A large amount of fat in the urine almost always signifies *chyluria*

[1] *Cf.* Hirschlaff, *D. Arch. f. klin. Med.*, vol. lxii., p. 531.

[2] Hematoidin and bilirubin are usually considered identical.

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(lipuria). The urine is then albuminous, of a milk-white to a cloudy-yellow appearance, sometimes even slightly blood-tinged, neutral or faintly acid, forms a cream-like layer, and often contains small coagula. The latter may form within the body as well as after the urine is voided. (Fibrin and Fibrinogen Contained in the Urine, see p. 464.) A microscopic examination shows that the fat is subdivided much more freely in the urine than in the milk. No distinct fat-drops can be seen, but extremely finely divided, almost invisible, fat-granules. These granules furnish the cloudiness and the cream-like layers. The other characteristics of this fatty admixture are the same as those that will be described later on (see p. 709 in reference to the chylous fluids sometimes found in the serous cavities. Chylous urine does not contain sugar, since this substance is not removed from the intestine by the lacteals, but by the veins. Chyluria is well known to be a tropic disease caused by a threadworm, Filaria sanguinis, which inhabits the blood. The urine occasionally contains the embryos of the filariæ (see Fig. 205).

The occurrence of tropic chyluria is due to the presence of adult filariæ in the thoracic duct, producing a stasis of the chyle, which extends not only to the lymphatics of the intestines, but also to those of the urinary apparatus. In such cases chyle may become admixed with the urine by drapedesis or by rupture of those lymphatics which do not ordinarily contain chyle. In one case Havelburg succeeded in definitely proving that the escape of chyle into the urine took place in the bladder. The frequent simultaneous admixture of blood is to be explained, according to Scheube, partly by the coincident rupture of blood-vessels and varicose lymphatics, and partly by the disappearance of the tissue between the venous and lymphatic vessels, due to the lymph stasis, whereby the contents of the lymphatics become mixed with blood. A similar stasis of chyle, produced in different ways in individual cases, may probably be the cause of the rare chyluria observed in this climate.

If the amount of fat contained in the blood is abnormal (lipemia), the urine also contains an abnormal amount (bone fractures, fat-embolism, diabetes mellitus, alcoholism, and acute phosphorus-poisoning). (See Examination of the Blood.)

A small amount of fat is also found in the urine in Bright's disease when the kidney elements (casts and epithelium) are eliminated in a fatty degenerated condition. The fat is then ordinarily enclosed within cells or casts, but exceptionally it may appear as fat-drops floating upon the surface, as a result of the degeneration of the cells or casts. Crystalline needles of fat have in a few cases been found in the urine. Such crystals are sometimes formed within the body, sometimes only outside from the fluid fat. (Fig. 211, *a*).

The examiner must, of course, be careful not to confound contamination from dirty vessels or catheter grease with *lipuria*.

If the macro- or microscopic appearance of the urine does not suffice for the demonstration of fat, then the urine should be extracted with ether. After evaporating the ether the physical characteristics of the fat are more easily recognized (grease spots), or an odor of acrolein (like smoking tallow candles) can be demonstrated if the residue is heated on a platinum foil, or, if the fat contains oleic acid the addition of a 1 per cent. solution of osmic acid will turn the residue black.

According to Späth, *lipuric acid* has been observed in urine, in rare cases, in the shape of prisms or needles. The factors governing its occurrence are not yet definitely known, although it seems to have been more frequently observed after the administration of salicylic acid.

MUCOUS SEDIMENTS.

If the urine contains considerable nucleo-albumin (compare p. 468 *et seq.*), it will separate spontaneously as a sediment of mucus-like consistence. Such a sediment consists microscopically of a cloud of clotted, transparent, indistinct masses. The addition of acetic acid will make their contours more distinct. They sometimes enclose various morpho-

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logic elements, white blood-corpuscles, epithelium, crystals, etc. Epithelial elements and white blood-corpuscles are almost always found in mucous sediments, because the increased nucleo-albumin is the product of a catarrhal disintegration of the mucous membrane.

ANALYTIC SCHEME OF THE PRINCIPAL INORGANIC URINE SEDIMENTS.

Readily soluble upon heating: Urates.

Insoluble or soluble only with difficulty upon heating.

- Soluble in acetic acid:
  - Phosphates, no effervescence.
  - Calcium carbonate with development of carbonic acid.
  - Ammonium urate with microscopic precipitation of uric acid.
- Insoluble in acetic acid:
  - Calcium oxalate. — Soluble in hydrochloric acid, the last three soluble in ammonia.
  - Leucin, tyrosin, xanthin, cystin. — Soluble in hydrochloric acid, the last three soluble in ammonia.
  - Uric acid. — Insoluble in hydrochloric acid.
  - Gypsum. — Insoluble in hydrochloric acid.

APPENDIX TO DISCUSSION OF INORGANIC SEDIMENTS. URINARY CALCULI.

Many of the substances enumerated as inorganic sediments are under certain conditions eliminated in the urine in the shape of concretions or urinary calculi.

Only the most important points in recognizing the various forms of urinary calculi will be mentioned here. The earthy phosphatic calculi are distinguished by their friability. The commonest calculi, those of uric acid and urates (urate calculi), are far more firm. The hardest of all are the calculi of calcium oxalate (so-called oxalate calculi).

The rare cystin calculi are usually yellow, smooth, small, and soft as wax; the still rarer xanthin calculi are clearer, rather hard, and when rubbed exhibit a wax-like polish. Calculi of cholesterin are also very rare and resemble the cystin concretions. The concrements, consisting of fat and of fatty soaps of the alkaline earths and designated as urostealiths, are extremely rare; they are characterized by their light color, soft consistence, and hardness when dried. Mention should also be made of a single recorded instance of a calculus consisting of indigo, which was sufficiently characterized by its color. Urinary calculi frequently consist of different substances arranged in layers, each of which may be more or less distinctly recognized by the previously mentioned characteristics.

For qualitative analysis a finely powdered specimen of the stone is first heated upon platinum foil. If the specimen burns up entirely or nearly so, it is composed of uric acid, xanthin, or cystin. Cystin and xanthin dissolve in dilute hydrochloric acid; uric acid does not. Uric acid may also be recognized by the *murexid test* (p. 556). Xanthin can be distinguished from cystin, as shown on page 561. To identify cystin, Salkowski digested the powder with ammonium hydroxid, filtered, and evaporated the filtrate in a watch glass. Cystin crystallized out in its characteristic hexagonal plates (Fig. 561). Cholesterin is characterized by its solubility in ether and by the beautiful rhombic plates which are formed by evaporating this ethereal solution.

If the powdered calculus does not burn up entirely upon the platinum foil, it must consist either of lime or magnesia. If in such a case the powdered specimen completely dissolves in dilute hydrochloric acid, it contains no uric acid along with the alkaline earths. What dissolves must consist of phosphates, carbonates, or calcium oxalate. Carbonates can be recognized by the development of gas.

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Calcium oxalate can be recognized by the fact that (by acetic acid) it is precipitated gradually, as a flaky cloudiness, from the dilute HCl solution neutralized with clear ammonium hydroxid. If any residue remain after the treatment with dilute hydrochloric acid, it generally consists of uric acid, which can easily be identified by means of the murexid test (p. 556).

ORGANIC ADMIXTURES AND SEDIMENTS OF URINE

As to the method of isolating these, see page 558 *et seq.*, at which place will be found the necessary information in reference to the elimination of interfering sediments composed of urates, phosphates, or carbonates.

PRESERVATION OF THE ORGANIC SEDIMENT.

If it is impossible to examine an organic sediment immediately after settling or after centrifuging, it may be preserved by washing several times with a normal salt solution and then kept in 1 per cent. osmic acid. The fat-drops of the cellular elements will be colored black if they contain any oleic acid. Instead of this procedure[1] the sediment, which has been washed with physiologic salt solution, may be hardened in a 1 : 20 sublimate solution for five minutes and then preserved in a 2 to 10 per cent. formalin solution. Formalin will destroy the red blood-corpuscles if they have not been previously hardened in the sublimate solution; hence the hardening by sublimate may be omitted if there are no red cells. May[2] calls attention to the fact that even in a case of this sort the urinary sediment must be first washed, otherwise considerable sediment of spheric crystals of diformaldehyd urea readily forms.

STAINING OF THE ORGANIC SEDIMENT.

There is no perfectly satisfactory method for staining a urine sediment. Most pigments produce precipitates after being added to the urine, so that the sediment is disfigured by granular masses. Dry specimens are unsatisfactory, because urine with an organic sediment is always more or less albuminous, and dried proteid stains strongly; hence the dry preparations are never neat. These disadvantages may be partially obviated, although in a rather cumbersome way, by washing the sediment repeatedly with normal salt solution, repeating the sedimentation, then drawing off the fluid repeatedly with a pipet, thus separating it from the urinary constituents which are held in solution, especially from albumin, and then finally adding the staining pigment. A cover-glass preparation may then be made in the same manner as with sputum (see pp. 593 and 596). The sediment must not be washed for too long a time, however, or sufficient material will not be obtained for the cover-glass. The same rules apply to the staining of the dry preparation as obtain in the examination of the sputum. If the attempt to make a dry preparation be unsuccessful, the moist sediment may be best stained according to the method of T. Liebmann:[3] The urine is centrifuged, the supernatant fluid poured off, and the sediment treated with 2 to 4 drops of a solution of 2 gm. of methylene-blue (Merck) in 100 c.c. of a 10 per cent. solution of formalin. The sediment and the staining solution are then well shaken in the centrifuge tube and allowed to stand for several minutes. The tube is then filled with water to remove the salts and the excess of stain, the solution is again centrifuged, and the sediment placed beneath the microscope. Hyaline casts are stained light blue, waxy casts dark blue, nuclei and bacteri dark blue, and red blood-corpuscles grayish blue. This method may also be employed for specimens preserved with formol.

If it be desired to demonstrate the fatty elements, a good method of staining the urinary sediment is that of Cohn[4]: The dry slide is hardened in a 10 per cent. solution of formalin for about ten minutes, then washed with water, and

[1] Gumprecht, *Centralbl. f. inn. Med.*, 1896, No. 30.
[2] *Arch. f. klin. Med.*, vol. lxviii., p. 425.
[3] *Münch. med. Woch.*, 1904, vol. xlix., p. 1768.
[4] *Zeits. f. klin. Med.*, vol. xxxviii., 1899, parts 1, 2, and 3. This article also contains good illustrations of urinary sediments stained by this method.

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placed for ten minutes in a concentrated solution of Sudan stain in 70 per cent. alcohol.[1] Hematoxylin is used as a counterstain, and the specimen mounted in glycerin. Fat is stained red and the nuclei violet.

Posner recommends hardening the dry preparation with osmic acid. He proceeds as follows:[2] Several crystals of osmic acid are placed in a wide-mouthed dark-glass bottle provided with an accurately ground glass stopper. The moist, smeared cover-glass is now placed over the mouth of the open bottle, with the smeared side down. This is accomplished by making the clean side of the cover-glass adhere to a slide by means of a drop of water. The slide is then held in position by the dark-colored stopper so that light is excluded. Fixation is completed in forty seconds. The cover-glass is then dried in the air, and stained without previous washing. Posner found that this treatment adapted the preparation for all the finer staining methods, and that it was also applicable for blood-preparations.

EPITHELIUM.

Scattered epithelium cells may be found even in normal urine. The desquamation of epithelium is a normal physiologic process. If they are present in large numbers, however, they point to inflammatory and destructive changes of the urinary apparatus. The following kinds may be distinguished:

Renal Epithelium.—They are generally spheric or cubic, a little larger than white blood-corpuscles, often distinguishable from the latter only by their larger size and by their large single nucleus. The nucleus is readily seen, and looks like a bubble, especially when stained (compare p. 567). They may show any degree of fatty degeneration, and may eventually become disintegrated to form a conglomeration of fat-drops. These cells are very uncommon in normal urine, but in some forms of nephritis are very plentiful (Fig. 195).

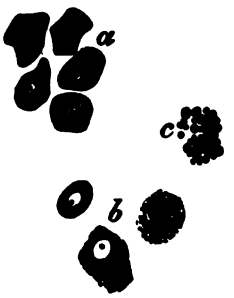

FIG. 195.—Renal epithelium from nephritic urine: *a*, Polyhedral renal epithelium in nephritis of scarlet fever; *b* and *c*, different grades of fatty degeneration in renal epithelium in chronic nephritis (× 400) (after Bizzozero).

Epithelium of the Urinary Tract.—These present extremely varied forms. The cells of the upper layers are generally flat, round, or polygonal; those of the deeper layers are elongated and provided with processes. Fig. 196 illustrates a number of these cells. A considerable quantity of the epithelium of the urinary passages is thrown off in all inflammatory processes. It was formerly believed that all cells provided with prolongations always came from the pelvis of the kidney. Unfortunately for the differential diagnosis of pyelitis and cystitis, this old idea is not true. No absolute point of differentiation between epithelium of the pelvis of the kidney and that of the bladder has yet been found. Nevertheless a very great predominance of caudate epithelium, as compared with the permanent variety, suggests pyelitis rather than cystitis. Fig. 197 represents the sediment taken directly from the pelvis of the kidney in a case of pyelitis.

[1] The solution is prepared by Dr. Grübler, Leipzig, Bayrischestrasse 63.
[2] *Berlin. klin. Woch.*, 1903, vol. xxxii.

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Vaginal and **preputial epithelia** are typical large pavement cells. Their shape should be familiar, because their accidental occurrence in the urinary sediment may lead to the erroneous presumption of

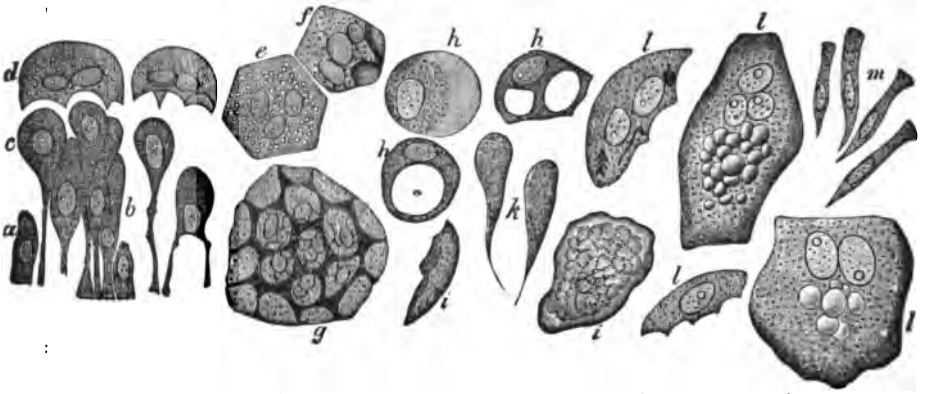

FIG. 196.—Epithelium of urinary passages: *a*, *b*, *c*, *d*, Profile of cells in their normal position: *a*, cell of deep layer; *b*, long cell of second layer; *c*, simple- and double-caudate cells; *d*, flat surface cell; *e*, surface appearance of superficial flat cell with three nuclei; *f*, surface appearance of superficial flat cell with one nucleus and 4 impressions (Nischen); *g*, surface appearance of superficial flat cell with many nuclei and many impressions; *h*, *i*, *k*, epithelium of bladder modified by action of urine; *h*, from alkaline urine, one cell swollen, the other two with vacuoles; *i*, surface cells; *k*, caudate cells; *l*, bladder epithelium, granular and stained yellow by blood-pigment, from a case of cystitis and nephritis; *m*, cylindric epithelium of male urethra (× 370 to 400) (after Bizzozero).

desquamative affections of the urinary tract. If a catheterized specimen is examined, no such mistake is possible.

PUS CELLS.

Pus cells may also appear in the urine in all inflammatory processes of the urinary tract or kidneys or when an abscess breaks into the uri-

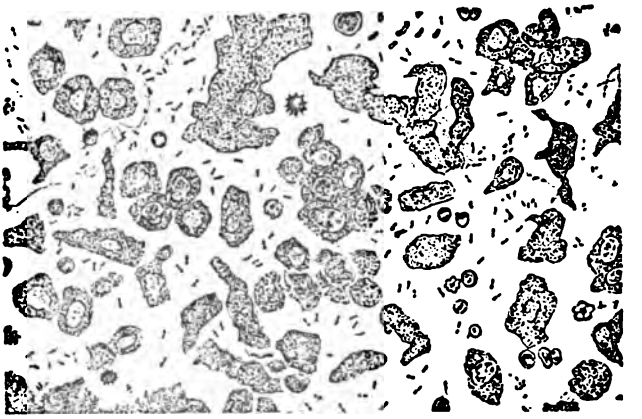

FIG. 197.—Sediment from a case of pyelitis, taken directly from the pelvis of the kidney in post-mortem examination.

nary tract. Their number may vary greatly. Sometimes they form the chief part of an abundant sediment, and sometimes they are only found

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scattered. When the urinary tract is diseased the amount of pus in the urine may be great, but in disease of the renal parenchyma proper the pus cells are usually very few. The origin of the pus cells can be determined with some degree of certainty only when the presence of other morphologic elements (epithelium, casts) serves as an indicator. The sudden appearance of a pus sediment in the urine suggests the rupture of an abscess into the urinary tract, provided that the other clinical signs correspond with this assumption. The occurrence of thread-like formations is very characteristic of gonorrhea and gonorrheal sediment (gonorrheal threads, compare p. 573). They consist of pus cells glued together with mucus.

In females the pus sediment may come from the vagina. To exclude such a possibility, either the vagina must be thoroughly irrigated before the urine is voided or the urine must be drawn with a catheter.

With alkaline fermentation a pus sediment is oftentimes converted

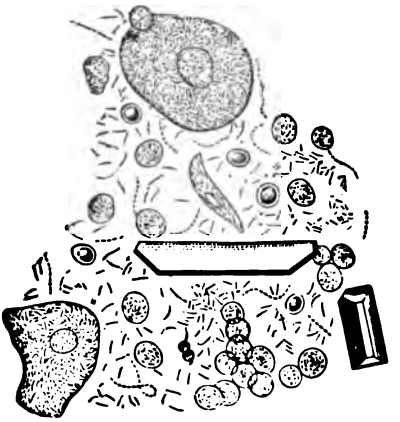

FIG. 198.—Sediment in alkaline inflammation of the bladder: Pus, epithelium, and triple phosphate crystals (after Peyer).

into a ropy, gelatinous mass by the swelling of the pus corpuscles. Purulent urinary sediments are often of a slimy consistence, even if the urine is not alkaline, because the urine in inflammatory affections usually contains nucleo-albumin (compare p. 468).

The pus corpuscles voided in the urine vary in their microscopic appearance, which depends partly upon the length of time since their escape from the blood-current, and partly upon the consistence of the urine, or upon the nature of the affection in question. Sometimes they are very cloudy and shrunken, so that the nuclei can be seen only after the addition of acetic acid (usually polynuclear or with nuclei irregularly crumpled); sometimes in an alkaline urine they are much swollen and glossy, and in this case also the nuclei are not easily seen. In faintly alkaline, neutral, or faintly acid urine, on the contrary, the pus corpuscles are often well preserved and sometimes even show active ameboid movements, especially when the slide is slightly warmed. The most important point of differentiation between pus corpuscles and

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epithelium, especially renal epithelium, is the shape or number of the nuclei. In the pus cells the nuclei are usually multiple or very irregularly shaped, never bubble-shaped. This can be best recognized in a stained specimen (p. 565). The size must also be considered. The pus corpuscles are usually of a diameter of 7 to 10 μ, corresponding to the polynuclear leukocytes from which they arise, whereas the epithelium cells are usually much larger. Figs. 198 and 199 represent the purulent sediment of alkaline and acid inflammation of the bladder or of pyelitis.

Of course, pus-containing urine always contains proteid in solution. It is a part of the quantitative estimation (see below) to decide whether the albuminuria, when pus is present, can be explained by the admixture of pus plasma only, or whether we must assume the simultaneous occurrence of a true renal albuminuria. In the latter case the amount of proteid is much greater. According to Posner, 100,000 pus corpuscles

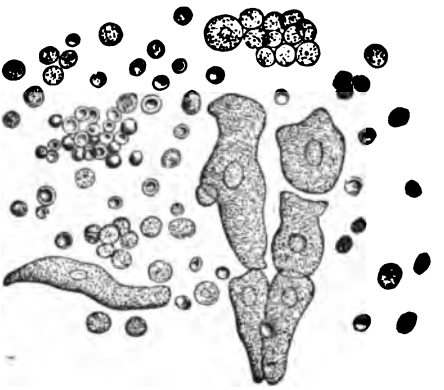

FIG. 199.—Sediment in acid inflammation of the bladder: Pus, red blood-corpuscles, and epithelium (after Peyer).

in a cubic centimeter correspond to about 1 per cent. of proteid. Morphologic elements characteristic of true albuminuria (casts, renal epithelium) will, if present, decide this question. Filtration does not remove the proteid of pus plasma, although this erroneous view has so often been maintained.

Posner formerly suggested that the amount of pus present in the urine could be estimated by counting the number of pus corpuscles in the centrifugalized twenty-four-hour amount of urine (analogous to counting the red blood-corpuscles) by means of the Thoma-Zeiss counting apparatus. This process is too complicated for practical purposes, so Posner, in a later communication,[1] announced another method of estimating the amount of pus mixed with the urine, or of the amount of sediment in general. The procedure is as follows: The transparency of the urine (the specimen must, of course, be taken from the mixed twenty-four-hour urine) is determined by placing a beaker with flat bot-

[1] *Deutsch. med. Woch.*, 1897, No. 40.

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tom upon a piece of paper with ordinary-sized print, and estimating the height in centimeters to which it is necessary to fill the beaker so that in good daylight the print can no longer be read. The degree of transparency is indicated by the thickness of the layer in centimeters. Posner found that a transparency of ½ to 1 cm. corresponded to 40,000 pus corpuscles per centimeter, and a transparency of 6 cm. to about 1000 pus corpuscles. Eight centimeters' thickness and above he assumed to indicate normal conditions. Such a quantitative estimation of the amount of pus present is important for estimating the effect of therapeutic interference in cystitis and pyelitis.

BLOOD.

Red corpuscles are found in the urine in hemorrhagic inflammation and tumors of the urinary tract and of the kidneys, in traumatic hemorrhage, in calculi, in hemorrhage from congestion, and in hemorrhagic diathesis. The blood-corpuscles appear in the urine partly intact, and partly changed in various ways by the action of the urine. They are most frequently both swollen and minus their pigment, exhibiting only the pale shadow-like stromata, sometimes as pale disks and sometimes as peculiar, almost invisible circles. Sometimes the blood-corpuscles break up into small masses of substance containing hemoglobin.

In determining the diagnostic significance of blood-cells in the urine, we meet with the same difficulty as in determining the significance of pus corpuscles—the difficulty of being sure of their source. In this connection also we must determine whether the amount of proteid present in the urine may be explained by the admixture of blood alone, or whether it depends upon a renal albuminuria as well. In the latter case the hemorrhage probably comes from the kidneys. If casts with blood-corpuscles adherent or blood-casts also occur in the sediment, the source is certainly a renal hemorrhage. Hemorrhage which leads to the elimination of larger blood-coagula in the urine usually arises not in the kidney parenchyma, but in some part of the urinary tract lower down, either the pelvis of the kidney or the bladder. It should be remembered, however, that in cases of hemorrhage from the renal parenchyma due to nephritis, blood-clots of considerable size may sometimes be found in the urine. A characteristic shape of the coagulum may point to its source from the pelvis of the kidney or the ureter. A considerable amount of blood at the end of micturition probably indicates bladder hemorrhage.

Gumprecht[1] claims that if most of the blood-corpuscles are fragments—*i. e.*, disintegrated to small masses—the kidney is generally the seat of the blood-extravasation. In hemorrhage of the bladder, on the contrary, but few fragmented blood-corpuscles will be found. As concentrated solutions of urea (as low as 8 per cent.) cause fragmentation of the red blood-corpuscles, Gumprecht maintains that the above-mentioned difference is due to the influence of the urea contained in the renal epithelium upon the extravasated blood. The amount of urea in the urine itself is said to be insufficient to produce the change. An objection to this supposition is that it is probable that the blood-corpuscles never come in

[1] *Deutsch. Arch. f. klin. Med.*, vol. liii., 1894.

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contact with solutions of urea of the above-mentioned concentration in the kidney. In the opinion of the author, it is more likely that the fragmentation is caused mechanically by the contusion of the blood-corpuscles in the urinary tubules.

CASTS.

Urinary casts are characteristic microscopic formations of cylindric form (Fig. 200), which originate in the kidney tubules. They are eliminated in the urine in nearly all cases of renal albuminuria; and infrequently even without albuminuria. Albuminuria may occur without

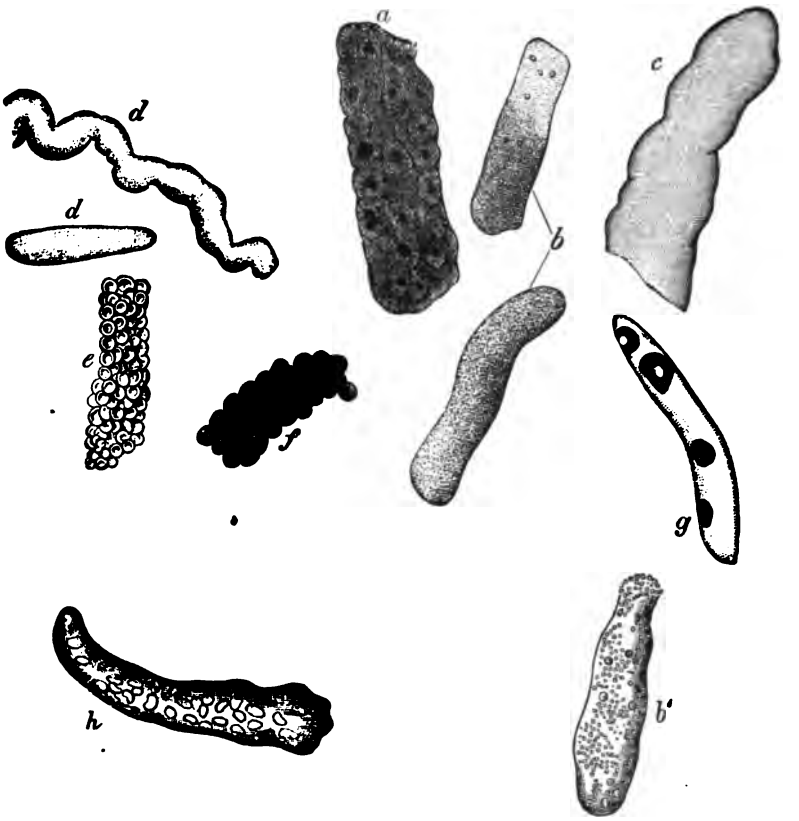

FIG. 200.—Casts in nephritis: *a*, Epithelial; *b* and *b'*, granular; *c*, waxy; *d*, hyaline; *e*, blood-cast; *f*, cast of white blood-corpuscles; *g*, hyaline cast with epithelium and white blood-corpuscles adherent; *h*, waxy cast with epithelium and red blood-corpuscles adherent (*h* and *b'*, after Bizzozero).

casts; but, generally speaking, elimination of casts and albuminuria go together. Casts occur most commonly in the various kinds of nephritis; but they may be present in the albuminuria of passive congestion as well as in other types of renal albuminuria. Nevertheless, casts may be absent in all of these cases. If the renal albuminuria does not depend upon nephritis proper, this is much more apt to be the case. In nephritis the degree of albuminuria and the number of casts eliminated are usually quantitatively proportional one to the other. Exceptionally, however,

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and then only for a short time, casts may be absent even with a considerable amount of renal albuminuria. The occurrence of casts without albuminuria (a rare condition) has been observed in very chronic cases of nephritis, in contracted kidney, and in jaundice.

The principal varieties of casts are the following (Fig. 200):

(1) Epithelial casts (*a*), composed of epithelium.

(2) Granular casts (*b* and *c*), granules partly soluble in acetic acid and partly consisting of fat.

(3) Waxy casts (*c*), strongly refractive, sharply outlined, often of a slightly yellow color.

(4) Hyaline casts (*d*), pale, indistinctly outlined, seen readily only with a slanting light.

(5) Blood-casts (*e*), composed of red blood-corpuscles or casts with red cells adherent, yellowish red to brownish, sometimes decolorized.

(6) Casts of white blood-corpuscles (*f*).

Besides these there are many intervening varieties—*e. g.*, hyaline or waxy casts with red or white blood-corpuscles or epithelium adherent (*g* and *h*).

Fatty casts are casts which contain fat in such large quantities that the ground-substance of the cast is entirely obscured. Casts may be composed of pus bacteria. Such occur in infections—pyelonephritis and in the kidney of pyemia. Other casts may become filled with bacteria in infectious conditions of the urinary apparatus, in the kidney itself or in the urinary tract, and even outside the body in urine containing bacteria. In uric acid infarct of the newborn so-called uric acid casts are found, consisting of spheres of sodium urate. Casts of any variety may become incrusted with urates in a concentrated urine after standing; they then present a peculiar dark granular appearance. They are differentiated from true granular casts by their uneven margins. The urate granulation disappears as soon as the urine is warmed or rendered alkaline.

The length and thickness of casts varies greatly. It is impossible to draw any conclusions as to the source of the casts in the kidney tubules from their diameter, because the diameters of the renal tubules are changed in pathologic affections of the kidney parenchyma. Sometimes the casts present peculiar screw-like twists, but such a peculiarity does not mean that they originate in the convoluted tubules. We require a microscope to see casts properly unless they are very thick and long.

The epithelial casts evidently consist of desquamated epithelial cells which are eliminated adhering to each other. These casts sometimes possess a lumen, and are then spoken of as epithelial "tubules." In a similar way extravasated red corpuscles form the so-called blood-casts, and the leukocytes the white-corpuscle casts. Any of these conglomerations of cellular elements may be so tightly compressed or disintegrated that the cell borders become more and more obliterated. Such changes, the nature of which we do not well understand, may take place in the kidney tubules themselves or in the urine after voiding. All grades of intermediate forms which still have the stamp of their origin merge without any sharp division into granular and waxy casts, in which no nuclei or cell-walls are to be distinguished. The existence of such transitional forms makes it probable that the granular and waxy

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casts originate as cell conglomerations. In these the epithelium and leukocytes appear to play the chief part, the red corpuscles a much smaller one. This view as to the genesis of the waxy and granular casts is supported by many investigations upon the diseased kidney itself. Formerly the casts which contained no cells, the granular and waxy as well as the hyaline, were considered to be coagulated. Whether this genesis can still be considered, in view of what has already been mentioned, has not yet been certainly decided, but it is not improbable that the hyaline casts are really formed by exudation. It is as yet still unknown by what process the hemoglobin which separates from the blood in solution in hemoglobinuria assumes in the kidney tubules the form of solid casts, the so-called hemoglobin casts. It can, however, be easily conceived that these casts do not consist solely of hemoglobin, but only represent hyaline and waxy casts infiltrated with hemoglobin.

We have but little accurate knowledge of the chemical nature of casts. Once in a while some cast (especially a waxy one) will give an amyloid reaction—*i. e.*, stain red with gentian violet and brown with iodin—although such a reaction does not indicate any amyloid degeneration of the kidney itself, for, as a matter of fact, in amyloid disease such a reaction of the casts is hardly ever observed.

The importance of finding casts in the urine is due to the fact that they always indicate some pathologic condition in the kidney, and, if accompanied by albuminuria, make it practically certain that the kidney itself is involved. But the change in the kidney need by no means be of a severe anatomic nature. Even slight disorders which produce but a temporary albuminuria may be accompanied by casts, although in small numbers.

For the distinction between nephritic albuminuria, on the one hand, and febrile or congestive albuminuria, on the other, we may lay down the rule that, if casts do occur in the latter, this is the exception. They are almost always merely hyaline. But both the febrile and congestive albuminuria may change to a nephritis without any very sharp border line.

Little can be claimed for the diagnostic importance of the different varieties of casts. Granular and waxy casts (as opposed to hyaline and epithelial) were formerly supposed to indicate a chronic process; but the idea was wrong. Any and all varieties of casts may be found in every type of nephritis, and even an amyloid kidney does not produce any distinctive type of casts (see above). Generally speaking, granular and waxy casts probably owe their peculiar characteristics to the fact that they have remained in the kidney tubules for some time. But that does not necessarily mean a case of chronic nephritis. As a matter of fact, it would probably mean acute nephritis or the acute exacerbations of chronic nephritis, as in such cases the casts remain especially long in the kidneys because the excretion of urine is most interfered with.

MUCOUS CASTS (Cylindroids).

These are peculiar formations which an inexperienced observer may mistake for true casts. They are, however, less distinctly outlined, shaped more irregu-

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larly, sometimes flat and tape-like, of smaller diameter, and frequently branching. They probably consist of mucus—*i. e.*, of the undissolved portion of nucleoproteid contained in the urine. They are, generally speaking, pale and

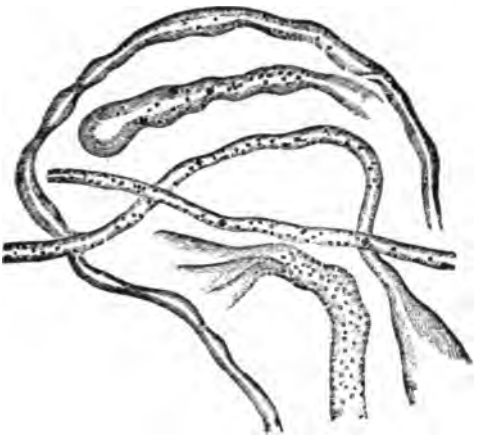

FIG. 201.—Cylindroids (after Peyer).

hyaline, but may be covered with urates, and then appear granular (compare p. 571). They are of no especial diagnostic importance.

TESTICLE CASTS.

In spermatorrhea certain formations are sometimes found in the urine which by themselves can hardly be distinguished from hyaline casts. The chemically normal condition of the urine, and the fact that this type of cast appears only in that portion of the urine leaving the urethra first and is usually accompanied by spermatozoa, will generally suffice to differentiate them from true renal casts.

GONORRHEAL THREADS (Shreds).

In the late stages of acute gonorrhea, when the secretion becomes more of a mucous consistence, and in chronic gonorrhea even when it gives rise to no other distinct symptoms, peculiar thread-like formations are found floating in the urine. They are up to 1 cm. in length, visible to the naked eye, generally pointed at the end, and of a yellowish to whitish color. Under the microscope we can see that they have a mucoid ground-substance, probably consisting of nucleo-albumin, in which pus corpuscles and epithelium are embedded.[1] They are probably caused by accumulation of secretion in the longitudinal folds of the urethra, whence they are torn away by the stream of urine.

SPERMATOZOA.

Spermatozoa occur in the urine after coitus, nocturnal emissions and onanism, in spermatorrhea, and after epileptic and other convulsive attacks.

FRAGMENTS OF NEW GROWTHS AND ELASTIC FIBERS.

We may find in the sediment bits of tissue which have become separated from papillomata and carcinomata of the urinary tract or, more especially, of the bladder. Similar bits may be caught in the catheter

[1] Plate to be found in Peyer, *Atlas der Mikroskopie am Krankenbette*, 1887, Plates 62 and 63.

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when irrigating the bladder. If such fragments are large enough to be sectioned and stained, the microscope will quickly determine their nature.

The demonstration of elastic fibers is sometimes quite important in the diagnosis of ulcerative processes of the urinary tract (Fig. 210). (See Examination of the Sputum.) The urine is first acidulated to prevent the formation of a phosphate precipitate, and then centrifugalized or allowed to settle. The liquid is then poured off from the sediment, and the latter gently heated with an equal quantity of diluted (10 per cent.) potassium hydroxid. This will destroy most of the morphologic constituents except the elastic fibers. The mixture is then diluted with water and centrifugalized again. If the urine contains a great number of elastic fibers, they may be recognized microscopically without further preparation. Elastic fibers must not be confused with vegetable fibers, which may gain access to the urine from the walls of a dirty vessel. The differentiation is discussed at p. 589—Examination of the Sputum.

MICRO-ORGANISMS.

Urine which has been allowed to stand soon furnishes a very favorable medium for the growth of all kinds of bacteria, particularly if the surrounding temperature is raised. The development of these organisms will decompose the urine in various ways. The chemical changes which take place in the urine may lead to the decomposition of any organized admixture and also to deception as to its composition. It is therefore important to preserve the urine in a cool place, and to undertake the qualitative examination as quickly as possible after voiding. For this purpose a twenty-four-hour specimen is not necessary. The decomposition of the urine may be checked by adding chemically indifferent antiseptic substances—*e. g.*, several cubic centimeters of coarsely powdered camphor or one-fifth its volume of chloroform water or the same amount of 0.1 per cent. solution of thymol. For the same reason urine glasses must be kept as aseptic as possible. This is best accomplished by washing them well with water and then with a 0.1 per cent. sublimate solution or a 2 per cent. warm soda solution every time after they have been emptied, and then keeping them covered, so that they are not contaminated by the bacteria in the dust of the air.

As we have seen, bacteria are very frequently found in the urine. The microscope will quickly settle the question of their presence in the sediment. A diffuse contamination by bacteria alone may be responsible for a very pronounced turbidity. Such a cloudiness may be distinguished from one caused by unorganized material, as it is affected neither by heat nor by the addition of acids or alkalies. It differs from sediment composed of pus, epithelial cells, or casts in that the latter sediment (1) quickly settles and so clears the upper layers of urine, and (2) is practically always associated with the presence of proteid. Shaking a urine extensively contaminated with bacteria often produces a peculiar opalescent, wave-like movement of the cloudiness. Also the opalescent

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film which appears upon the surface of a urine which has been kept for some time usually consists of bacteria.

The demonstration of bacteria in the urine is diagnostically important only when they occur in freshly voided urine or in a specimen obtained by catheterization. Then they consist either of great numbers of saprophytic bacteria, which are readily seen under the microscope without staining, and which produce abnormal decomposition of the urine within the urinary tract when the latter is diseased (bacteria of ammoniacal fermentation and bacteriuria), or else of true pathogenic bacteria, which can be recognized microscopically or cultivated upon appropriate media. The latter are found partly in local diseases of the urinary tract, as in certain cases of cystitis and especially in types of acute nephritis, and partly in general infections. They include such organisms as colon bacilli and staphylococci (Fig. 219), streptococci (Fig. 218), gonococci (Fig. 203), pneumococci (Fig. 214), typhoid bacilli, and tubercle bacilli (Fig. 213). In general infections in which bacteria (staphylococci, streptococci, pneumococci) are eliminated in the urine, it is still debatable whether the kidneys and urinary passages are unaffected or whether such elimination is always combined with a lesion of these organs. The microscopic demonstration of these bacteria is really far more important than the culture method of demonstration, because the former excludes the source of error of any incidental contamination, and because it gives more accurate information than the latter as to the quantitative importance of individual species in mixed infections. With a culture it may happen that bacteria of little or no pathologic importance develop inordinately.

The microscopic examination is performed just as with dry-sputum examinations—*i. e.*, dry preparations are prepared by spreading the sediment on cover-glasses (pp. 593 and 596 *et seq.*). If a specimen of urine is swarming with bacteria, a drop of the urine can be dried and examined like an ordinary dry preparation. If, on the contrary, the specimen of urine contains but few bacteria, it is better to examine a freshly and cleanly prepared sediment obtained by means of the centrifuge. Bacterial suspensions are so difficult to centrifugalize, that if other morphologic elements are wanting the examination will be simplified by first diluting the urine with alcohol. If the urine contains proteid or albumoses, the precipitate which results from the addition of the alcohol aids in isolating the bacteria by helping to carry them down in centrifugalizing. Of course, such a precipitate should be merely flaky, otherwise it would interfere with making a good dry preparation. Should the alcohol precipitate large flakes, the urine must first be diluted. Dry preparations are prepared by spreading the sediment thinly upon a cover-glass (just as in sputum) and fixing over a flame. Fixation is, of course, sometimes difficult, because urea is hygroscopic. Hence it is often necessary first to wash the sediment several times with distilled water, centrifugalizing after each washing, or, if necessary, heating for a longer time over the flame, so as to convert the urea into ammonium carbonate. Staining can be accomplished exactly as with sputum

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preparations (pp. 593 *et seq.* and 596 *et seq.*). The bacteria may also be stained by the previously described method of Liebmann (see p. 564).

For cultures we must naturally employ perfectly fresh urine, withdrawn by means of a sterilized catheter into sterilized vessels after careful disinfection of the urinary opening. The first urine drawn in this manner must be thrown away, since it may contain bacteria which have been scraped from the walls of the urethra by the eye of the catheter. Even after observing this precaution the urine may become contaminated, so that positive conclusions should not be drawn unless there are a large number of colonies.

The demonstration of *tubercle bacilli* in the urine is important for the diagnosis of tuberculosis of the urinary tract (Fig. 213). Dry preparations of the sediment are employed in the same way as with the examination of sputum (p. 593). In difficult cases the search for the tubercle bacilli may be facilitated by treating the sediment with sodium

FIG. 202.—Smegma bacilli (× about 800) (after Fränkel).

FIG. 203.—Gonococcus (× about 800) (after Fränkel).

hydroxid (see p. 596), or tuberculosis may be proved to be present by inoculation experiments upon guinea-pigs (see p. 596). A difficulty in such a method is that other pathologic bacteria which occur in the urine may multiply and kill the animal before the tuberculosis can develop. By washing the sediment before inoculation, the danger of such infection is lessened by the removal of toxic urinary substances. The reader should refer to p. 596 and to bacteriologic text-books for the details of performing an inoculation and for the conditions found in the inoculated animals.

The so-called *smegma bacillus* (Fig. 202 *et seq.*) has been so frequently confused with tubercle bacilli as often to lead to an erroneous diagnosis of urogenital tuberculosis. The smegma bacillus, as described by Alvarez and Tavel, occurs very frequently in the preputial fold of the male and in the folds above the female clitoris. In the specific staining for tubercle bacilli they also are stained (p. 595). From their location they can easily contaminate the urine and multiply

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in it outside of the body (?). These parts should therefore be washed very carefully before the urine is passed or before catheterization.[1]

The smegma bacilli differ from the tubercle bacilli in being more slender and not granular, and not exhibiting the characteristic groupings of the tubercle bacilli (see Figs. 202 and 213). Finally, when the stained preparations are afterward treated with HCl alcohol for from five to ten minutes, the smegma bacilli become decolorized, but not the tubercle bacilli. Grette recommended combining the counterstaining effect of methylene-blue with the decolorized action of the

FIG. 204.—Echinococcus elements: 1, Free scolices; *a*, rostellum projected; *b*, rostellum withdrawn; 2, hooklets; 3, membrane (× cross-section) (after Heller).

alcohol.[2] He stains as ordinarily with carbol fuchsin, and after washing with water he treats the specimen with a concentrated alcoholic solution of methylene-blue (without acid). With this method the tubercle bacilli remain red, whereas all the rest of the preparation, including the smegma bacilli, is stained blue. Another difference between them is that Gram's stain decolorizes the smegma but not the tubercle bacillus. Finally, the tubercle but not the smegma bacilli are changed to formations like strings of pearls, resembling streptococci, by excessively heating the dry preparation—*i. e.*, by passing it perhaps ten times through the

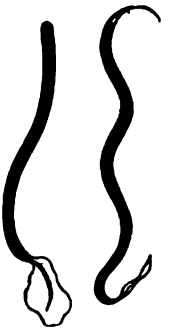

FIG. 205.—Embryos of Filaria sanguinis: Length, 0.0075 to 0.21 mm.; thickness, 0.004 to 0.36 mm. (after Scheube).

FIG. 206.—Eggs of Distomum hæmatobium (Bilharzia hæmatobia): Length, 0.12 mm.; breadth, 0.05 mm. (after Bilharz).

flame. Compare also the staining method recommended by Pappenheim for distinguishing tubercle bacilli from smegma bacilli in the sputum. The employment of Ebner's decolorizing fluid will also prevent the confusion of these bacilli (see p. 595). The presence of gonococci in the urine sediment is also of diagnostic interest.

[1] Runge and Trautenroth have shown that the urine obtained by catheterization after carefully cleansing the external orificium urethræ is always free from smegma bacilli.

[2] *Fortschr. d. Med.*, 1896, No. 9.

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The *bladder catarrh* and *pyelitis* which may complicate gonorrhea never depend upon a pure gonorrheal infection, but upon secondary infections of the urinary tract with other organisms. At the same time, in such cases, if the disease has not been entirely cured, pus corpuscles which contain gonococci are sometimes found in the sediment. They have probably become mixed with the urine from the urethra. Fig. 203 shows the characteristic arrangement of the gonococci in the interior of the pus cells. A diagnosis of gonorrhea is permissible only when this characteristic intercellular arrangement has been recognized, because the same biscuit-like grouping so characteristic of diplococci is also seen with the ordinary staphylococci. Another characteristic of gonococci is that they do not grow upon ordinary culture media.

Braatz[1] found actinomycosis granules in a case of actinomycosis of the urinary tract (Figs. 224 and 225).

In diabetic urine a rapid yeast growth is sometimes produced by the presence of yeast fungi. The sugar becomes fermented, carbon dioxid bubbles are formed, and yeast spores settle in the sediment. Hence, the demonstration of yeast fungi in the urine may suggest the diagnosis of diabetes; but even non-saccharine urine may exhibit yeast-like fungi.

ANIMAL PARASITES.

Echinococcus of the urinary tract and of the kidneys may cause fragments of echinococcus cysts and hooklets or whole daughter cysts to be voided in the urine (Fig. 204).

Embryos of Filaria sanguinis (Fig. 205) are found in the urine in the tropic hematochyluria, and the eggs of Distomum hæmatobium (Bilharzia hæmatobia, Fig. 206) in the Egyptian hematuria.[2] It remains to be mentioned, finally, that in rare cases the trichomonas is found in the urine in disease of the urinary tract. This species is probably identical with that occurring in the intestine (Trichomonas intestinalis; compare p. 449 and Fig. 151).

EXAMINATION OF THE SPUTUM.

EXPECTORATION, or sputum, is what is voided by coughing or clearing the throat. It is composed of the secretion or exudate from the respiratory mucous membrane of the nose, pharynx, and trachea down to the finest bronchi and alveoli; of material which has reached the respiratory tract from neighboring regions (pus of abscesses and empyema); of blood derived from somewhere along the respiratory tract; and, finally, of material from the buccal cavity or from any part of the digestive tract. Macro- and microscopic foreign bodies which have entered the respiratory system from without are usually voided in the expectoration.

On account of its numerous sources of origin, the composition of the sputum is very complex and of great symptomatologic importance.

[1] *Petersburg. med. Woch.*, 1888, No. 13.

[2] Rüttimeyer, *Mittheilungen aus kliniken und medicinischen Instituten der Schweiz*, 1894, vol. i.

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The sputum is not always expectorated. Small children, and occasionally adults, in consequence of bad habits, insufficient practice, or impaired consciousness, swallow their sputum. In such case its help in diagnosis is lost. Some of these adults and most older children can be taught to expectorate. For diagnostic purposes, the expectoration for twenty-four hours is collected most conveniently in a transparent sputum cup.

AMOUNT OF SPUTUM.

The amount of sputum may vary greatly, depending upon what process produces it. Some pulmonary patients, in spite of violent coughing, expectorate only a small quantity, and that usually very tenacious (dry bronchitis, incipient phthisis). Others may expectorate large quantities during the day, or even at one time (in certain types of chronic bronchitis sometimes called bronchorrhea, in advanced pulmonary tuberculosis, in bronchiectasis, in pulmonary edema, in pulmonary hemorrhages, and in perforation of abscesses or an empyema into the air passages). The special characters of these various types of sputum will be discussed later on.

CONSISTENCE OF THE SPUTUM.

The consistence of the sputum bears a certain relation to its amount. If very abundant, a sputum is usually less tenacious than if scanty. Ordinary sputum is "slimy"; it may, however, be either serous, purulent, or bloody. The peculiar slimy consistence of sputum depends largely upon the amount of mucus it contains, since the mucin-like substances excreted from the respiratory mucous membrane form a considerable portion of the expectoration.[1] The stickiness of the sputum also is partly due to mucus and partly, in some cases, to proteid, especially the sputum of croupous pneumonia, the stickiness of which is supposed to be largely due to the amount of nuclein contained.

REACTION OF THE SPUTUM.

The reaction of fresh sputum is generally alkaline. It may become acid, after standing for some time, from decomposition of the sputum by bacterial growth.

COLOR AND TRANSPARENCY OF THE SPUTUM.

The color of the sputum varies decidedly. Pure mucoid or glassy sputum may be perfectly colorless, and so viscid that it might easily be mistaken for saliva. Admixture of blood-corpuscles makes this slimy secretion more and more yellow or greenish, and also cloudy and opaque. It is not clear why the color in one case is more yellow, and in another more greenish, any more than in the case of pus. But as the more

[1] The mucus of the respiratory apparatus appears to be composed mostly of true mucin (Cohnheim, *Chemie der Eiweisskörper*, Braunschweig, Vieweg, 1900; F. Müller, "Beiträge zur Kenntnis des Mucins," etc., *Zeits. f. Biol.*, vol. xlii.).

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intense inflammations are usually associated with the transudation of red blood-corpuscles, it seems fair to assume that the green derivatives of blood-pigment usually produce the greenish pus color. (See later with reference to other causes of green discoloration of the sputum.)

A *mucopurulent sputum* consists of an admixture of masses of pure mucus with masses of pus, or of a moderate but uniform admixture of the two elements, the whole appearing but slightly clouded. *Purulent sputum* is the next step in such admixtures. Its consistence is still mucoid unless composed of pure pus (either thick or thin) derived from an abscess or from an empyema, and expectorated as such without any mucus.

Serous sputum is another type of colorless expectoration. It differs from the pure mucoid or glassy sputum in its very liquid consistence and foamy appearance. It occurs in pulmonary edema, and rarely from the perforation of serous pleural exudates into the lung. Serous sputum is frequently slightly tinged with blood.

Sputum has a slightly reddish color whenever mixed with blood. In some cases the sputum consists of pure blood; in other cases it contains only a slight admixture, producing a salmon color. Between these two extremes all sorts of transitional forms occur. Hemorrhagic sputum is observed in traumatic and tuberculous hemorrhages from the lungs, in hemorrhagic infarctions of the lungs, in pneumonia (especially in croupous pneumonia), in gangrene of the lung, in tumors of the lung, and finally in congestion of the pulmonary circulation.

Certain derivatives of blood-pigment produce in the sputum very similar shades to that of bloody sputum proper. Rusty sputum, the most common type in pneumonia, is an instance. The coloring matter is here partly unchanged blood, partly, however, a yellowish-red derivative of the blood-pigment, about which little is as yet known. Peculiar lemon-colored and grass-green shades are infrequently observed in pneumonia sputum. They are due to further chemical alterations of the blood-pigment. All these variations are analogous to the changes of blood-pigment observed in subcutaneous ecchymoses. Such sputa respond to Gmelin's test for bile pigment (see p. 472 *et seq.*). Lemon-yellow and greenish sputa in pneumonia are frequently regarded as icteric. This supposition is, however, justifiable only when there is general jaundice or, at least, some icteric discoloration of the conjunctivæ, or when bile pigment is contained in the urine. In the remaining cases we cannot speak of icteric sputum, although it must be admitted either that the contained coloring matter is related to bile pigment, because it is derived from hemoglobin, or that it is identical with bile pigment when a distinct Gmelin reaction is obtained. A peculiar light-brown shade characterizes sputum which contains abundant cells of heart disease (see Plate 8, Fig. 3). Here, especially in mitral disease, amorphous blood-pigment is found encapsulated in the pulmonary lung epithelium. The brownish or ochre-like color of the sputum which is occasionally observed in destructive processes of the lung, especially in pulmonary abscesses, or in a liver abscess which has perforated into the

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lung, is due to hematoidin or bilirubin crystals. In the expectoration from a liver abscess the hematoidin or bilirubin crystals are derived not from the blood, but from the admixture of bile with the pus. The bitter taste noticed by patients when expectorating such sputum depends upon the presence of biliary salts. Gmelin's reaction for bile pigment will not differentiate the source of the hematoidin or bilirubin, as they react in the same way, whether derived from blood or from bile.

True icteric sputa, such as has been observed in certain cases of pneumonia or with any other pulmonary affections with jaundice (compare p. 42 *et seq.*), may present various shades of color, due to the oxidizing of bilirubin. Yellow and then dirty-green shades are the most common.

A rather different type of green sputum has been observed in lung tumors; but the nature of the pigment in question is as yet unknown. Certain pulmonary tumors (chloroma) which belong to the sarcomata or to the lymphadenomata are greenish in color. Hence a greenish sputum in a tumor of the lung would suggest a chloroma; but green sputum has been observed in cases of carcinoma. The pigment of chloroma dissolves in alcohol. Too much attention has probably been devoted to the green sputum in tumors of the lung, because, as we shall see, bacteria which may produce pigment may cause a greenish discoloration of sputum outside of the body.

Other noticeable pigmentation in sputum has been observed from the admixture of inhaled dust particles. Black sputum belongs to this class. Its pigment is due to inhaled coal dust or soot, of which, as is well known, normal lung pigment consists. A small portion of the carbon is free in the sputum; the larger part is contained in the interior of round or oval epithelium and white blood-cells (compare p. 209). Under the microscope we can frequently recognize the vegetable structure of the larger particles of carbon. These are usually not contained in the cells, but are found free. The gray or blackish discoloration of such sputa, as well as the degree of lung pigmentation, depends chiefly upon the individual's occupation. The sputa of coal miners are frequently intensely black (anthracosis of the lung). Other *pneumoconioses* —*i. e.*, changes in the lung produced by the inhalation of dust—are associated with peculiar discoloration of the sputum. Workmen—*e. g.*, in polishing mirrors—who breathe the dust of oxid of iron (English-red, caput mortuum), and who have thereby acquired siderosis of the lung, may expectorate an ochre-colored sputum. The reddish particles are for the most part enclosed within the cells. To demonstrate siderosis from the sputum, add hydrochloric acid and a solution of ferrocyanid of potash; Berlin blue will then be formed. Blue sputum has been observed in men working in ultramarine.

These discolorations of the sputum are not due to the pigment which has impregnated the lung, for whatever pigment particles are deposited in the lung probably never leave it; they should rather be considered as coeffects of the same cause—viz., inhalation of dust. Only the dust which has been recently inhaled, and which has not penetrated the interior of the pulmonary tissue, is expectorated.

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Consequently the characteristic discoloration of the sputum will disappear soon after the patient gives up the occupation that produced it, although the pneumoconiosis will persist. Discolored sputum should be considered rather as a sign of the inhalation of dust than of pneumoconiosis. Where the characteristic discoloration of the sputum continues or reappears after the individual has been removed for some length of time from the specific dust atmosphere, it generally means that some destructive process, usually tuberculosis, is added to the pneumoconiosis.

The "gluing" or "smearing" of the respiratory passages, described by Gerhardt and Lublinski,[1] which occurs in bakers and millers should be mentioned. Here paste-like masses are expectorated. It is due to the inhalation of flour.

Hair and teeth are sometimes found in the sputum following the perforation of a dermoid cyst of the mediastinum into the bronchi.

The manifold discolorations of the sputum due to admixtures from without, and not from the air passages, should be mentioned—*e. g.*, when patients take milk, eggs, claret, coffee, chocolate, or some colored medicine before expectorating, the sputum is apt to become discolored in the mouth. A green discoloration of the sputum is sometimes due to the growth of certain chromogenic bacteria, especially the Bacillus virescens.[2] Yellow, bluish, and reddish sputa of probable bacteriologic origin have also been observed. The parasite of "blue pus" (Bacillus pyocyaneus) probably grows in sputum.[3]

AIR CONTENT OF THE SPUTUM.

Sputum is often more or less distinctly foamy or frothy, due to the presence of air. Other things being equal, the amount of air contained in sputum is greater the finer the bronchi from which it is derived. This is because air is most easily incorporated with tiny masses of sputum, as they are being collected into large masses, while passing from the smaller to the larger bronchi. The consistence of the sputum is also of importance. Thin sputum is usually very frothy. Thick, tough sputum is less so. The amount of air contained in the sputum can be readily recognized by its specific gravity. Air-containing sputum will float on the water in a sputum cup; airless sputum will sink. The sinking of sputum (sputa fundum petentia) has been considered a proof of its derivation from a cavity of the lungs. Evidently, sputum derived from the pus of pulmonary cavities will ordinarily contain but a slight amount of air; but so may purely catarrhal secretion from the larger bronchi, so that this test is of slight diagnostic value, and then only in connection with other signs.

SPUTUM STRATA.

Sputa which settle in layers in the cup are observed chiefly in bronchorrhea (chronic bronchitis with abundant secretion), in bronchiectasis,

[1] Gerhardt, *Centralbl. f. inn. Med.*, 1896, No. 20; Lublinski, *Ibid.*, 1896, No. 28.

[2] See Frick, *Virchow's Archiv*, vol. cxvi., 1889, p. 226.

[3] [A bacillus which could not be differentiated from the Bacillus pyocyaneus was isolated from the sputum of a case of chronic bronchitis. This sputum when first expectorated was colorless, but developed an intense greenish color upon standing less than three hours. The literature contains a number of similar instances.—C. N.]

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in putrid bronchitis, and in gangrene of the lung. There are usually three layers—an uppermost, aërated portion; a middle or serous layer, consisting chiefly of pus, serum or mucoid fluid; and a third layer, the sediment, which consists of pus corpuscles, gangrenous shreds of lung tissue, and molecular lung detritus.

ODOR OF THE SPUTUM.

Fresh sputum rarely has any particular odor; but upon standing it may acquire a disagreeable odor, due to the action of bacteria. Freshly expectorated sputum has a very strong odor in purulent bronchitis, in some cases of pulmonary tuberculosis, in bronchiectasis and pulmonary gangrene, and very frequently in pulmonary abscess and in empyema of the lung. The disagreeable odor in these cases arises from the growth of putrefactive bacteria even before the expectoration of the sputum. Stagnation of the secretion in cavities favors the processes of decomposition. The foul odor of the contents of the lung is usually imparted to the exhaled breath, where it may even be more distinct than in the sputum itself. This is frequently noticed in consumptives. The peculiar carrion-like odor of the breath so characteristic of this disease will sometimes point to pulmonary tuberculosis before even any definite pathologic signs have appeared in the chest, and while the sputum may be remarkably free from odor. We should then naturally suspect that the foul odor came from the mouth, which is not by any means always the case. This paradoxical condition is very likely due to the fact that the warm air in the lung takes up odors more readily than the air at room temperature. Besides, sputum may very rapidly lose its odor upon standing. The same is true of the fecal vomitus in intestinal obstruction. It seems to the writer probable, and his idea is confirmed by others, that consumptives with this characteristic odor usually have cavities (even if quite small) in which the secretion stagnates.

The sputum may have a peculiar odor after the administration of myrrh, oil of turpentine, ether, alcoholic drinks, paraldehyd, etc. It has been assumed that these substances are partly eliminated by the lungs; but it has been proved recently that no appreciable quantity of alcohol is expired, and that, except for the slight trace of alcohol which, of course, adheres to the mucous membrane of the mouth, the odor of the expired air after drinking alcoholic beverages is due principally to the esters which all liquors contain. At all events, the odor comes largely from the lungs, for deodorizing mouth washes, such as permanganate of potash, produce no effect.

CHARACTERISTIC GROSS APPEARANCES OF THE SPUTUM.

Many sputa appear to the eye perfectly homogeneous—pure mucous, pure purulent, pure bloody, etc. But sometimes not only may the sputa from one patient vary, but differences can be distinguished in each expectoration. Particularly in mucopurulent sputum the particles of

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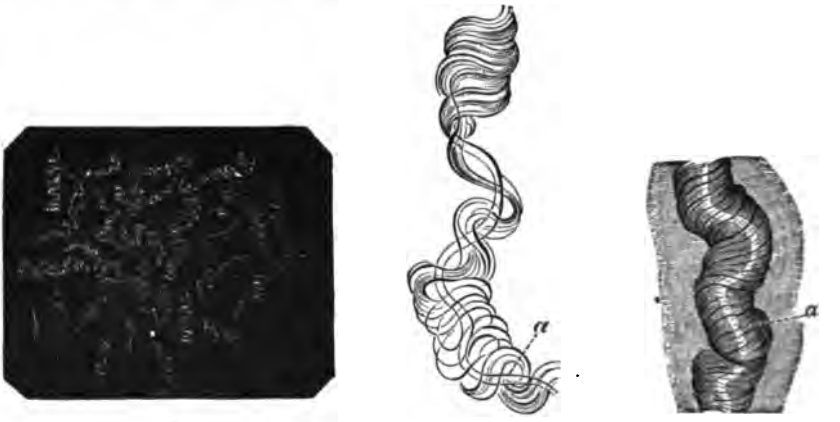
mucus and pus may alternate. From the quantity of these individual constituents we can judge more or less correctly whether the sputum arises from a larger or a smaller bronchus.

A peculiar fine, flocculent appearance of purulent sputum is very characteristic of the slow emptying of a pleural empyema or of a pulmonary abscess. The flocculi can be best seen if the sputum is suspended in water. They are probably due to the fact that the pus is pressed into the shape of shreds or strands in being squeezed through the seat of perforation. These shreds become surrounded by mucus, and are then no longer confluent.

To detect other admixtures which exhibit a microscopic difference from the main bulk of the sputum, it is advisable to examine a small portion of the sputum upon a plate one-half of which has been painted with black enamel paint, so that the background may be either black or



I. II. III.

FIG. 207.—Curschmann's spirals: I., Natural size; II. and III., enlarged: *a*, central fiber (after Curschmann).

white, as desired. In this way it is easy to select ropy, fibrous, generally dirty dark particles or larger grayish-black shreds characteristic of necrotic lung tissue. If elastic fibers (Fig. 210) are detected by the microscope, the diagnosis is confirmed. These, however, may be absent in gangrene of the lungs (p. 590). Bits of necrotic cartilage in ulcerative processes of the bronchi, trachea, or larynx, and tumor fragments may be detected in the same way.

"Dittrich's plugs," yellowish-white bits the size of a mustard seed, are very conspicuous when looked for over a dark background. They come from the smaller bronchi in putrid diseases of the lungs, especially in putrid bronchitis and in pulmonary gangrene. Microscopically, they consist of clumps of bacteria and crystals of fatty acids (compare Fig. 211, *a*). They have a very intense and disagreeable odor.

Similar plugs may be seen mixed in the sputum in follicular tonsillitis, or may even be extruded from the crypts of a normal tonsil.

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"Dittrich's plugs" should not be confused with the spiral formations described by and named after Curschmann. These are represented in Fig. 207; I., natural size; II. and III., slightly magnified. They consist of worm-like formations 1 and 2 cm. long and about 1 mm. thick, more or less opaque, and usually suspended in a glassy menstruum. They are shiny, viscid in consistence, are visible to the naked eye, and are composed of shreds twisted into a spiral. They are only rarely branched. Some of them exhibit a central, more strongly refractive fiber (Fig. 207, II., *a* and III., *a*). Other formations which resemble these central fibers occur in sputum, but they are isolated and not surrounded by spirals. A. Schmidt showed that Curschmann's spirals and the central fibers consist of a mucin-like substance and not of fibrin, as was formerly supposed.[1] They are not so dense as the shreds of fibrin which occur in the sputum. These spirals of Curschmann are portions of the secretion or exudate which forms in the finest bronchi as the product of a so-called *bronchiolitis exudativa*. This affection is frequently the cause of or secondary to *bronchial asthma*. Hence Curschmann's spirals are more or less characteristic of asthmatic sputum. After the end of a typical attack they are sometimes found in extraordinary numbers. We must not, however, suppose that Curschmann's spirals are pathognomonic of asthma, for not only do cases of asthma occur without spirals, but spirals may be found in the sputum of a bronchitis without asthma. They are sometimes found in croupous pneumonia, in which case there is a very good opportunity to distinguish them from bits of fibrin. When strongly magnified the spirals will frequently be found to contain white blood-corpuscles and Charcot's crystals (Fig. 211, *e*). These leukocytes, particularly in cases of bronchial asthma, are strikingly white, mostly mononuclear, and contain eosinophilic granules (see p. 591 *et seq.*).

Curschmann's spirals can be preserved in glycerin. Their mode of origin is by no means definitely known. Doubtless their shape is due to the screw-like movement of the mucus while passing through the bronchi. Personally the author prefers to consider that this spiral motion is caused by the lashing movement of the cilia of the bronchial epithelium, although we do not yet know whether the direction of the motion of the cilia is spiral or not. Senator believes that the screw-like shape of these structures is brought about by physical means, and that it is analogous to the spiral-like mass of a salve which is forced out of a compressible tube by pressure.

Formations consisting of fibrin may be found in the sputum under various conditions. They are easily recognized by their white color, by their tenacious consistence, and sometimes by their shape. In diphtheria of the pharynx, larynx, and trachea, the expectorated fibrinous pseudomembranes consist of uniform masses or casts of these organs. Diphtheria not infrequently reaches as far as the bronchi, and branching bronchial casts may be expectorated. They are readily recognized and are important in diagnosing the extension of the disease to the lungs, and so determining the prognosis. Similar branching bronchial casts are observed quite as frequently in croupous pneumonia, because the

[1] *Zeits. f. klin. Med.*, vol. xx., p. 476, 1892.

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bronchial mucous membrane usually participates in the fibrinous inflammatory process. Fig. 208 represents a cast of this sort. Large numbers of such casts are often contained in the sputum of pneumonia. They look like white strands or shreds. To investigate their nature more accurately, they should be isolated by shaking the portion of sputum which contains them in a test tube with water. All other formations lose their shape, whereas these casts float about in the water.

FIG. 208.—Fibrinous bronchial casts.

These tree-like formations may be solid or hollow. Similar formations are found in the sputum of patients with fibrinous or croupous bronchitis. The etiology of this disease has not been investigated carefully. Its course is usually chronic, very rarely acute. The sputum is more or less blood-tinged, and not infrequently contains beautiful branching casts of very large bronchi. Under the microscope these fibrinous casts show the ordinary coarse structure of fibrin and the same power of refracting light. Like Curschmann's spirals, they often contain Charcot's crystals (Fig. 211, *e*). Fibrin coagula can be distinguished both macroscopically and microscopically from mucous constituents of the sputum by their swelling and becoming more translucent after the addition of acetic acid. If fresh fibrin is submerged in peroxid of hydrogen, gas will develop much more quickly than in the case of mucus (catalytic action).

Foreign bodies are sometimes aspirated. They may even remain in a bronchus for years without causing any symptoms, and finally be expectorated. There are cases reported where foreign bodies, such as teeth, cherry stones, etc., have been expectorated after having been ten and even nineteen years in the lungs.

Calcareous concretions are sometimes, but very rarely, formed in the lungs during chronic infections. They may be finally coughed up (lung stones).

Very rarely daughter cysts of pulmonary or hepatic echinococcus reach the bronchi, and so appear in the sputum (Fig. 204).

MICROSCOPIC EXAMINATION OF THE SPUTUM.

In many cases this purpose can be satisfactorily accomplished by placing a small particle of the sputum upon a slide and then pressing down with a cover-glass. To recognize some of the constituents, the specimen must first be treated with reagents and stains (see later).

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The sputum should, however, first be examined just as expectorated. This process is frequently neglected. The presence of fungi or of crystals in the sputum, the nature of many cellular elements, etc., can be recognized only in such a fresh preparation, or, at least, much more easily recognized there.

Most sputa consist microscopically of a groundwork of mucoid material of indefinite structure in which pus corpuscles are embedded. The number of the latter determine the more or less purulent nature of the sputum. The character of these corpuscles varies considerably. Their size is from 7 to 10 μ. They are more or less granular. The granules consist partly of proteid material (neutrophilic granules, compare Examination of the Blood, Leukocytes), partly of fat, partly of extraneous débris identical with that of so-called lung epithelium (see

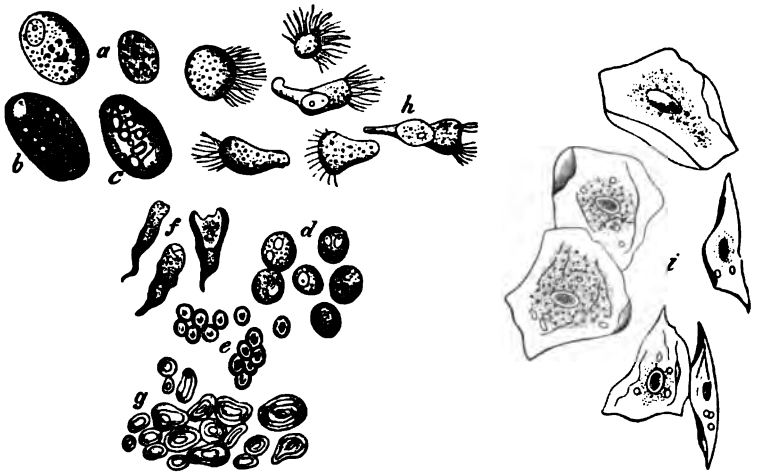

FIG. 209.—Different morphologic elements of the sputum: *a*, *b*, *c*, Pulmonary or alveolar epithelium: *a*, with normal lung pigment (carbon); *b*, with fat-globules; *c*, with myelin granules or drop; *d*, particles of pus; *e*, red blood-corpuscles; *f*, cylindric beaker-shaped bronchial epithelial cells; *g*, free myelin granules; *h*, ciliated epithelium of different kinds from the nose, altered by coryza; *i*, stratified epithelium from the pharynx (after Bizzozero).

below). Like polymorphous leukocytes, these pus corpuscles usually possess one irregular nucleus or several nuclei. In stained preparations (hematoxylin) the nuclear substance never appears as a vesicle, but is compact. (For the presence of eosinophilic cells see p. 591 *et seq.*)

Epithelial cells are also found in sputum. They differ from pus corpuscles in exhibiting a single rather large vesicular nucleus. Various types of epithelium are found in the sputum. Their demonstration and recognition is of especial importance for determining the origin of a catarrhal expectoration. First of all, there is *squamous epithelium*, derived from the mouth, the pharynx, and a portion of the larynx, especially the true vocal cords (Fig. 209, *i*). There is *cylindric epithelium* (Fig. 209, *f*, *h*), derived from the deep-seated bronchi or from the nose, and represented by beaker-shaped and ciliated cells, although, of course, many of the latter have lost their cilia. They are

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rarely very numerous except perhaps in a fresh bronchial catarrh. In the latter stages of catarrh they are almost completely supplanted by leukocytes.

Besides epithelium derived from the upper portion of the respiratory tract, there is the so-called *pulmonary* or *alveolar epithelium* (Fig. 209, *a*, *b*, *c*). These are oval cells 20 to 50 μ in diameter, with one or several nuclei and various inclusions. The latter consist of: (*a*) normal lung pigment—*i. e.*, carbon (Fig. 209, *a*); (*b*) fat; (*c*) so-called myelin granules or drops, which are pale, irregular bodies of considerable size, showing concentric layers, and resembling the myelin globules of the central nervous system (see below as to their origin); (*d*) granules or scales of brown pigment, in all probability derived from blood-pigment (heart cells; Plate 8, Fig. 3); (*e*) hematoidin crystals. The same cell may enclose a variety of inclusions. We do not know whether these cells, as claimed by Bizzozero, are derived exclusively from the epithelium of the pulmonary alveoli, or whether some of them may not be cells from the epithelium of the upper air passages, or phagocytic leukocytes.

The character of the epithelial cells found in the sputum makes it possible to locate the portion of the respiratory tract from which the microscopic specimen was obtained.

The myelin drops or granules above mentioned are often found free in the sputum. According to the investigations of A. Schmidt, they should be considered a product of normal secretion of the mucous membrane of the respiratory system. They make up the larger part of the morning sputum, which is swallowed by the majority of people. Chemical examinations by A. Schmidt and F. Müller have shown that the myelin of sputum consists largely of protagon, with which is mixed a small amount of cholesterin and lecithin.

When "heart cells" occur in large numbers in the sputum, we are justified in assuming definite anatomic changes in the lung. These cells are filled with yellowish-brown pigment granules. They have been called the "cells of heart disease," because they are found in the sputum in brown induration of the lung from cardiac failure, especially with mitral lesions. If found in considerable numbers for a considerable length of time without the existence of an infarction, they are of real diagnostic importance. These cells are also found in the sputum of pulmonary infarctions and after hemoptysis. Their characteristic pigmentation is probably derived from the pigment of red blood-globules which they have ingested. Sputum containing many of these cardiac cells may sometimes appear slightly reddish brown.

Whenever the bloody character of a sputum is doubtful, a microscopic examination will usually determine the presence of the red corpuscles. They may have preserved their normal appearance, or they may be altered in various ways. They may disintegrate into distinct and separate accumulations of hemoglobin and, later on, become amorphous or crystalline hematoidin, or sometimes the coloring matter may be entirely dissolved.

Wherever lung tissue is considerably destroyed by any pathologic process, elastic fibers are apt to be found in the sputum. They form

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the residual portion of the pulmonary parenchyma (Fig. 210). Their presence in the sputum proves beyond a doubt the occurrence of some destructive process in the lungs; hence their importance in the diagnosis of tuberculosis of the lungs before the tubercle bacillus was discovered. Even now the presence of elastic fibers may decide the diagnosis if we are unsuccessful in our search for tubercle bacilli. Elastic fibers are also found in pulmonary abscess and gangrene in greater or less number according to the rate of disintegration.

The fibers can usually be detected if a thin layer of the sputum is examined microscopically. If they are so scarce that they cannot be found in this way, the following procedure is recommended: About 10 c.c. of sputum are boiled in a small porcelain dish with an equal quantity of 10 per cent. KOH. The mixture should be well stirred during the boiling. When it becomes homogeneous it is diluted with about four times as much water, well shaken, and then centrifugated or allowed

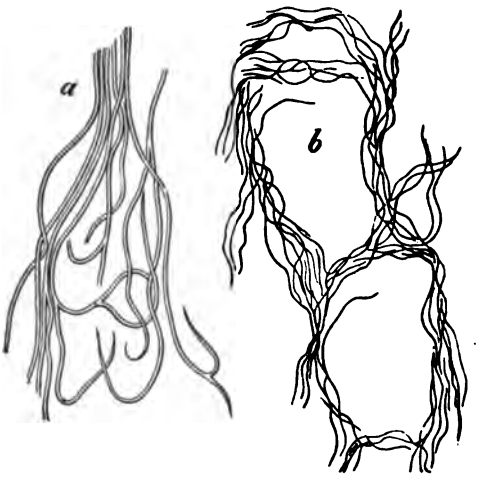

FIG. 210.—Elastic fibers from the sputum (after Bizzozero).

to stand in a pointed glass. From the sediment any elastic fibers which may be present may be picked up with a pipet and then examined under the microscope. After subjecting elastic fibers to KOH, they are usually somewhat less sharply outlined and slightly swollen.

When present in considerable quantity they are easily recognized by their alveolar arrangement (Fig. 210, *b*). Beginners are liable to confuse isolated elastic with vegetable fibers, but most of the latter have a large diameter, and are much less plainly wavy.

R. May recently described a very convenient method of staining elastic fibers with orcin. The method is useful in doubtful cases. The technic will be found in May's original article.[1]

Weigert has also given an excellent method for staining elastic fibers in sections by means of fuchsin. The author does not know whether it has been employed for the demonstration of elastic fibers in the sputum, but it would seem

[1] *Deutsch. Arch. f. klin. Med.*, vol. lxviii., parts 5 and 6, p. 427.

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that it could be made use of, provided that the cover-glass smear were dried, and then fixed with absolute alcohol or formalin. A few minutes will suffice for fixation if alcohol be employed. If formalin is used, the specimen should be left in a 4 per cent. aqueous solution of formaldehyd, and then washed off with absolute alcohol. The staining fluid is prepared as follows: Two hundred cubic centimeters of an aqueous solution of fuchsin (1 per cent.) and resorcin (2 per cent.) are brought to the boiling-point in a porcelain dish; 20 c. c. of liquor ferri sesquichlorati are then added, and the mixture constantly stirred and boiled for three to five minutes, when a muddy precipitate will form. The mixture is then cooled and filtered. The filter and the contained precipitate are dried, and again placed in the same porcelain dish, which will also contain some of the precipitate. Two hundred cubic centimeters of 94 per cent. alcohol should then be added, and the mixture boiled over a water bath and constantly stirred. During

FIG. 211.—Crystals in the sputum: *a*, Fat; *b*, cholesterin; *c*, leucin (balls) and tyrosin (needles); *d*, hematoidin (bilirubin) in rhomboids and needles; *e*, Charcot-Leyden crystals.

this procedure great care must be exercised to avoid ignition of the vapor. The fluid is cooled, and enough alcohol is added to bring the amount up to 200 c.c., after which 4 c.c. of hydrochloric acid are added. The cover-glasses should remain in this fluid from twenty minutes to an hour, after which they are washed with alcohol, cleared in xylol, and finally examined in Canada balsam.

Some cases of incipient phthisis which can hardly be recognized may show the presence of elastic fibers.

It is a peculiar fact that in some cases of gangrene of the lung, but not in all, elastic fibers of the lung are absent in necrotic pieces of lung tissue recognized microscopically as such. In such cases the elastic fibers have been destroyed by a trypsin-like ferment formed by bacteria which grow in the gangrenous tissue.

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Fragments of neoplasms of the lung which are sometimes seen in the sputum (p. 584) are best recognized in microscopic sections.

Crystals Observed in the Sputum (Fig. 211).—They occur in the sputum almost exclusively when the latter has remained in the body for a considerable length of time. Crystals of fat or of fatty acids are most frequently found. They form long needles (*a*), sometimes free and sometimes grouped together in rosets. They may be distinctly bent, and so perhaps may be mistaken for elastic fibers; but may be readily distinguished by the fact that they dissolve in potassium hydrate or ether. Crystals of cholesterin (*b*), of leucin and tyrosin (*c*) are very rarely observed. They are formed chiefly in stagnating, putrid sputum. Crystals of triple phosphate (see Figs. 189 and 190) may be found in the sputum under similar conditions. Hematoidin crystals (Fig. 211, *d*) are found chiefly in the sputum of abscess and of perforating empyema. The blood-pigment in pulmonary hemorrhages is mostly changed in the interior of the cells to amorphous pigment (see Heart-failure Cells), and but very rarely forms hematoidin crystals. Charcot-Leyden crystals (*e*) sometimes occur in the sputum. They are colorless, elongated, double pyramids, and vary considerably in size. They also occur elsewhere in the body during life and post mortem (in tumors, in stools, in leukemic blood, in bone marrow, and in the spleen).

Fr. Müller and Gollasch not long ago discovered a peculiar relationship between Charcot's crystals and the presence of eosinophilic leukocytes in the sputum. According to Müller, 60 per cent. of the leukocytes in asthmatic sputum are sometimes eosinophiles. It therefore seems rational to assume that Charcot's crystals are products of the crystallization of eosinophilic cells. This assumption is supported by the fact that the crystals readily stain with eosin, and that they occur in leukemic blood (see p. 667). Nothing definite is yet known regarding the chemistry of Charcot's crystals. Schreiner demonstrated that the so-called "Böttcher's sperm crystals" could be obtained by drying spermatic fluid. They resemble Charcot's crystals in appearance, and are the phosphate of an organic base (spermin Pöhl). Schreiner's idea is pretty generally accepted that they are identical with Charcot's crystals. But Cohn[1] claims that these crystals differ considerably crystallographically, and that they belong to a different system. Charcot's crystals, as shown by their transverse surfaces, belong to the hexagonal system. They are doubly refracting, like Böttcher's crystals, but, unlike the latter, have a hexagonal transverse section.

These crystals are chiefly but not exclusively found in the sputum in bronchial asthma, so that for some time they were regarded as the exciting cause of such an attack. The bronchial nerves were supposed to be irritated by the pointed crystals. The relation, however, is not constant enough to justify any such conclusion. Asthma frequently occurs without crystals, and crystals are often found in the sputum without asthma. It seems much more rational to assume that the crystals are formed during the attack of asthma from eosinophilic cells, owing to the stagnation of the secretion products of the bronchial mucous membrane. *Curschmann's spirals* are to be included among these same products. The fact that Charcot's crystals are found in asthmatic sputum chiefly after the expectoration has been interrupted for a considerable length of time would seem to favor this theory. When expectoration is free and abundant, the crystals are usually absent. Furthermore, Charcot's crystals are found very abundantly in the interior of Curschmann's spirals; and, when the latter are preserved in a moist chamber, crystals may form in spirals which were previously free from them.

[1] *Deutsch. Arch. f. klin. Med.*, vol. liv., p. 514, 1895.

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Animal parasites or fragments of them are very rarely found in the sputum. Infusoria are very uncommon in the sputa, and of little importance clinically. Hooks, scolices, and bits of membrane of echinococcus may be found in the sputum in the rare cases where echinococci cysts of the lung or liver perforate the bronchi. There is a disease in eastern Asia which usually manifests itself by the occurrence of hemoptysis. It is due to the presence of a worm (Distomum pulmonale) in the lungs. Its eggs (Fig. 212) can always be demonstrated in the expectoration. They are oval, of a brownish-red color, 0.08 mm. long and 0.056 mm. broad, and are surrounded by a cuticle. They are easily identified under the microscope.

FIG. 212.—Distomum pulmonale with embryo (× 225) (after Nakahama-Leuckart).

Vegetable parasites are of greater interest and importance, especially the bacteria; the fungi play a more subordinate part. In examining the sputum for vegetable organisms, we should always bear in mind that by far the majority are saprophytic in nature—*i. e.*, they develop in the sputum either outside of the body or, if within the air passages, only in the secretion, and exist there in a more or less harmless way. To determine whether vegetable organisms in doubtful cases are expectorated as such, or are due to contamination of the sputum outside the body, we should examine the sputum immediately after it has been expectorated. There are, however, a number of bacteria which can be distinguished with so much certainty from accidental contaminations that their presence, even in an old sputum, is of diagnostic importance. This is especially true of tubercle bacilli and pneumococci.

In obtaining sputum for bacteriologic examination a number of precautions must be observed. Since it is usually desired to examine the secretion originating in the deeper parts of the respiratory tract rather than that coming from the mouth, the mucopurulent masses should be selected instead of the thin secretion, which largely consists of saliva. It is well to examine only such clumps as are expectorated in the presence of the physician. If this be done, it is easy to determine from the character of the expectoration whether the mass has originated in the bronchi, in the larynx, in the pharynx, or in the nose. In order to reduce bacterial contamination from the mouth to a minimum, the oral cavity should be thoroughly washed with water just before the expectoration. The sputum obtained after this precaution is received in a clean glass; if cultures are to be made the glass receptacle should be sterile and provided with a lid, so that the sputum may be immediately covered.

DETERMINATION OF TUBERCLE BACILLI IN THE SPUTUM.

Tubercle bacilli are often so numerous in the sputum that they may be found with ease in any particle examined (Fig. 213); but in cases which are doubtful clinically and where the diagnosis depends upon the examination of the sputum, tubercle bacilli may be found only

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after prolonged search. If the sputum is absolutely homogeneous, we must prepare a large number of slides without any particular selection. If mucopurulent, we can save time by spreading the expectoration upon a black background, and then selecting for examination the larger purulent clumps and any very cloudy, friable, cheesy-looking particles. Tubercle bacilli may be found in the initial hemoptysis, although the patient seemed perfectly healthy beforehand. It is, of course, advisable in such a case to select particles which are not pure blood, but which have a certain amount of mucus and purulent material admixed. In a fresh hemorrhage from the lung, the more profuse the bleeding the less chance of finding bacilli.

The recognition of the tubercle bacillus depends upon a special procedure by which they alone are stained. If unstained, they cannot be distinguished from other bacteria. The ordinary methods of staining bacteria are not suitable. A specimen is prepared by teasing a portion of the sputum to be examined, placing a small fragment of it or of the sediment (provided centrifugalization has been performed, see p. 596) upon a cover-slip, and then carefully spreading it. To obtain a uniform distribution, a second cover-slip is placed over the first, and the two then drawn apart with the fingers or forceps. Unless too much material has been used, a thin layer will be left upon each cover-slip, and this may be dried over an alcohol or gas flame with a moderate amount of heat. It must then be *fixed* so that it will not wash off in the staining fluid. This is done by passing the slip rapidly through a flame three times. The principle of the special stain for tubercle bacilli depends upon the fact that these bacteria do not stain readily with the ordinary anilin colors, but that when they are once stained they do not readily decolorize. An anilin dye which acts very intensely is selected; and for this purpose the ordinary basic anilin colors are the best, such as fuchsin or gentian violet, mixed with carbolic acid or with anilin. The directions for preparing this stain are given below (see p. 594). A few drops of the stain are dropped upon the cover-glass, and the latter held over the flame with a pair of forceps till the fluid steams. This process will stain the tubercle bacilli and all other organisms present.

FIG. 218.—Tubercle bacilli (after a photograph by Günther) (× 500).

The next step is to decolorize all the other organisms and any other morphologic constituents of the preparation. This is easily accomplished, because the tubercle bacilli holds the anilin colors with great tenacity. Some mineral acid is generally employed for decolorizing, most frequently nitric acid in some dilution or other, as mentioned

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below. The acid is allowed to work until the specimen appears entirely decolorized. This occupies a few seconds to a minute, according to the thickness of the preparation. If parts of specimens are too thick, we select only the thin places for examination.

After decolorizing, the preparation is washed in water. If any color remains the acid must be renewed. It is sometimes necessary to alternate these two processes several times. The specimen is then mounted upon a slide with water or balsam, and examined with an oil-immersion lens. A much larger specimen can be prepared and examined directly with the oil-immersion lens by dropping some cedar oil upon the slide. Although this method is a very good one, it is not to be recommended, on account of the danger of scratching the objective by particles of mineral matter contained in the sputum. Such a dry preparation may be made upon the slide, but it should be covered by one large or several cover-glasses of the usual dimensions.

A counterstain after decolorizing aids in recognizing other microorganisms or tissues in the specimen, and makes them form a contrast to the tubercle bacilli. Methylene-blue is well adapted for this purpose if the tubercle bacilli have been stained red. If gentian violet has been employed, Bismarck-brown or fuchsin are suitable counterstains. This second staining may be accomplished without heating. The counterstain should not be intense enough to obscure the tubercle bacilli.

Details in reference to the counting of tubercle bacilli will not be given, since the author can impute neither a diagnostic nor a prognostic value to such a procedure. The number of tubercle bacilli in a single preparation is dependent upon chance in the selection of the particle examined; and even when such chance is eliminated by the study of a large number of specimens, the prognosis and the gravity of the case are entirely independent of the number of tubercle bacilli in the sputum. For this reason he regards Gaffky's scale for the quantitative designation of the bacilli as valueless, and employs simply the terms "a few," "many," or "very many" tubercle bacilli.

Solutions for Staining Tubercle Bacilli.—1. A solution of fuchsin or gentian violet in saturated anilin water (Ehrlich). This is best prepared fresh for each examination by pouring 2 c.c. of anilin oil into a test tube, filling with distilled water, and shaking. The mixture is filtered into a watch glass, and a saturated alcoholic solution of fuchsin or gentian violet is added drop by drop to the filtrate until a metallic sheen appears upon the surface of the mixture, indicating that the anilin water is saturated with the stain.

2. A solution of fuchsin or gentian violet in carbolic water (Ziehl-Neelsen). One gram of fuchsin or gentian violet is mixed with 10 c.c. of absolute alcohol and 100 c.c. of a 5 per cent. solution of carbolic water; or a saturated alcoholic solution of fuchsin or gentian violet may be dropped into a 5 per cent. solution of carbolic water until saturation takes place (formation of metallic sheen on the surface). These carbolic solutions have the advantage of keeping for a considerable length of time.

3. Czaplewsky[1] recommends the use of the following solution: One

[1] *Hyg. Rundschau*, 1896, No. 21.

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gram of fuchsin in 5 c.c. acid. carbol. liquefact. in a dish. Fifty cubic centimeters of glycerin are added, stirring constantly, and then diluted with 100 c.c. of water. This solution keeps extremely well and does not need to be filtered.

Ordinary aqueous solutions without anilin or carbolic acid may be used (concentrated alcoholic stain dropped into water and prepared fresh each time). The stain is somewhat less intense. The other mixtures are preferable for diagnostic purposes.

4. The best decolorizing method is that of Czaplewsky, who employs Ebner's fluid, consisting of:

℞ Acidi hydrochlorici,		
Sodii chloridi,	*āā*	2.5
Aquæ destillatæ,		100.0
Alcoholis,		500.0

Before the employment of this fluid the specimen is washed with water; the decolorization may be accelerated by treating the specimen alternately with Ebner's fluid and water. The advantage of this fluid is that the acid-resisting bacilli are decolorized by the contained alcohol and cannot be confused with tubercle bacilli, as was the case with the purely aqueous solutions of acids previously employed.

Gabbet's method of decolorizing and counterstaining simultaneously, which was formerly almost universally employed, is not to be recommended, since Gabbet's solution contains no alcohol, and consequently gives rise to the previously mentioned confusion.

ISOLATING TUBERCLE BACILLI FROM OTHER ACID-STAINING BACILLI IN THE SPUTUM.

(Occurrence of Smegma Bacilli in the Sputum.)

Pappenheim,[1] in Lichtheim's Clinic, recently found smegma or nearly related bacilli in a non-tubercular affection of the lung. They closely resemble tubercle bacilli in their affinity for acid stains. Laab found similar bacilli on the tonsils, on the tongue, and in the coating about the teeth. They are so rare, however, that probably no serious mistakes have occurred. In examining urine for tubercle bacilli, these smegma bacilli are of considerable importance. To avoid any mistake, Pappenheim's method of staining should be employed. This colors the tubercle bacilli red and the smegma bacilli blue. Pappenheim recommends the following procedure: 1. Stain in carbol fuchsin, heat to boiling-point for a few moments; 2, pour off the excess of carbol fuchsin; 3, decolorize and counterstain, without washing, with the following solution, pouring it slowly three to five times over the preparation and allowing it to run off: One part of corallin is dissolved in 100 parts of absolute alcohol, and methylene-blue added till saturation (a considerable amount is necessary for this purpose); 20 parts of glycerin are then added; 4, wash with water, dry, and mount. Duration of entire procedure, three minutes.

Confusion may be avoided by decolorizing the specimen stained in carbol fuchsin with Ebner's fluid, as described above.

SEDIMENTATION OF TUBERCLE BACILLI.

If the tubercle bacilli in a specimen of sputum are scarce, the examination may be facilitated by diluting the sputum and then centrifugating the mixture.

[1] *Berlin. klin. Woch.*, 1898, No. 37, p. 809.

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This can best be accomplished as recommended by Viedert. One to 2 c.c. of sputum are placed in a test tube with six to eight times as much 0.2 per cent. solution of sodium hydrate, and then boiled once. The flocculent residue obtained (after about forty-eight hours' standing, if centrifuged) is used for making a dry preparation, after adding some of the original sputum, so as to make the layer stick. To determine the other micro-organisms which may be contained in the sputum, an antiseptic—for instance, one-fifth volume of saturated chloroform water—should be added, so as to prevent the growth of contaminating bacteria. Tubercle bacilli and other bacteria in the dilute sodium hydrate above mentioned survive, and the tubercle bacilli will often be found, where they would be missed in the ordinary dry preparation.

Spengler's[1] method of making the sputum homogeneous by pancreatic digestion or, so as to prevent decomposition, by peptic digestion in acid solution has no particular advantage over Hiedert's original method, according to the data the author has been able to collect.

Ilkewitsch[2] recommends another method which is very useful for collecting tubercle bacilli in the sediment. This consists in stirring the sputum for quite a while with twenty times as much water, precipitating with acetic acid (mucin, nucleo-albumin), and then centrifuging.

EMPLOYMENT OF ANIMAL EXPERIMENTS FOR DEMONSTRATING TUBERCLE BACILLI IN SPUTUM.[3]

When no tubercle bacilli can be found in the sputum microscopically even after sedimenting, animal experiments may be resorted to for diagnostic purposes. A suspicious portion is washed repeatedly and rubbed up in normal saline solution, and 0.5 to 1.5 c.c. of the fluid injected into the peritoneal cavity of a guinea-pig.

The weight of the animal is recorded, and it is killed in four to six or ten weeks, according to the diminution in its weight. An examination is then made to ascertain whether tuberculosis of the abdominal organs has developed. Sometimes the animals die in twenty-four to seventy-two hours, from an infection with pneumococci or streptococci. The experiment should then be repeated, after heating the sputum for ten minutes at 60° C., a temperature which will, according to the investigations of Forster and his pupils, kill all inflammatory organisms except the tubercle bacilli.

DEMONSTRATION OF OTHER MICRO-ORGANISMS.

These may be demonstrated in dry preparations made very much in the same way as those for tubercle bacilli. They are stained with the same solutions and according to the same methods, but the acid is omitted. After staining, the preparations are washed in water only. If the stain is then too intense, the preparation may be subjected for a very few moments to the action of alcohol. The latter will remove any color which has been precipitated and render the bacteria more distinctly visible. Most bacteria possess so great an affinity for fuchsin and gentian violet that heating is unnecessary. Cold preparations will stain bacteria sufficiently inside of a few seconds to a minute.

Gram's Method.—Some bacteria stain very intensely with Gram's method, while the other elements of the sputum are decolorized. A specimen is stained with anilin gentian violet and then washed with

[1] *Zeits. f. Hyg.*, 1894, vol. xviii., part 2.
[2] *Baumgarten's Jahresbericht*, 1892, vol. viii., p. 664.
[3] See Levy and H. Bruno, *Deutsch. med. Woch.*, 1900, No. 9, p. 141.

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water; Lugol solution[1] is then added drop by drop. The iodin solution is allowed to act upon the preparation from one to three minutes; it is then washed with absolute alcohol until entirely decolorized microscopically. When no further color can be washed away the specimen is dried, and then examined in cedar oil. The nuclei and the body of the preparation will appear entirely decolorized or very slightly yellow, but the micro-organisms will be stained intensely blue or almost black. By subsequent counterstaining (Bismarck-brown or fuchsin ten times diluted, Ziehl-Neelsen's or Czaplewsky's solution) a capital contrast stain is obtained.

Beautiful specimens may also be obtained, according to Czaplewsky, if Weigert's modification of Gram's method be combined with a fuchsin counterstain. Czaplewsky describes the procedure as follows: Stain for one minute in carbolic gentian violet (11 c.c. of a concentrated alcoholic solution of gentian violet, 10 c.c. of alcohol, 50 c.c. of a 5 per cent. solution of carbolic acid, 50 c.c. of distilled water); wash thirty to sixty seconds. Lugol's solution (1 iodin, 8

FIG. 214.—Fränkel's pneumococci (from a photograph by Fränkel) (× about 800).

potassium iodid, 200 aqua); wash, dry, differentiate with anilin-xylol 2 : 1, to which has been added 1.5 per cent. of acetone; wash with xylol, dry, counterstain with diluted carbol fuchsin, 1 : 10 (see p. 594), for about one minute, during which time the specimen is warmed slightly. The specimen is then washed, dried, embedded in Canada balsam, and examined with the oil-immersion lens.

Staphylococci, streptococci, diphtheria bacilli, tubercle bacilli (staining with heat), anthrax bacillli, bacilli of tetanus, and Fränkel's pneumococci stain with Gram's stain; whereas typhoid, influenza and cholera bacilli and Friedländer's bacilli of pneumonia decolorize with Gram's stain.

This property of decolorizing is only a relative one, for individuals of certain species which, as a rule, are not stained by Gram's stain may retain their stain, and *vice versâ*. A great deal depends upon the intensity of action of the decolor-

[1] This solution consists of 1 part of iodin, 2 parts of potassic iodid, and 300 parts of water. These are the original directions; but according to the author's experience, a solution of gentian violet in 5 per cent. carbolic water, as recommended by Ziehl-Neelsen for staining tubercle bacilli, is quite as good.

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izing method. Where mixtures of bacteria and not pure cultures are being treated, as in the examination of sputa, the question whether certain bacteria decolorize after Gram's stain can best be decided by subsequently counterstaining with a fuchsin solution diluted ten times. Bacteria which decolorize after Gram's stain will then be stained red.

The demonstration of *Fränkel's pneumococci* in the sputum is of considerable diagnostic importance. They are usually elongated, lance-shaped cocci, generally arranged in pairs with their bases approximated. They are surrounded by a faintly staining capsule, which in dry preparations usually does not stain at all. Fränkel's pneumococci are supposed to be the cause of croupous pneumonia. They must not be confounded with other diplococci found in the sputum, more especially with Friedländer's[1] so-called diplococci. The latter also possess a capsule, but they have nothing to do with the etiology of croupous pneumonia, although they may sometimes be present with pneumonia. Friedländer's cocci, when strongly magnified, are seen to be short bacilli. Cultural differences also exist, and Gram's stain decolorizes them, but stains Fränkel's diplococci. W. Wolf has furnished us with a very useful double stain for Fränkel's diplococci. The dry preparation is first of all stained in anilin water saturated with fuchsin, and then placed one to two minutes in a diluted aqueous solution of methylene-blue. The cocci stain blue, the capsule rose color, and the body of the preparation a bluish red.

The diagnostic significance of pneumococci is limited, because the same organism may be found in the normal secretions of the mouth and in non-pneumonic sputum. It is identical with the organism of an experimental sputum septicemia of rabbits. The cocci, however, are found in very small numbers in normal secretions from the mouth. According to modern views, any bacteria found in the body normally may, under certain conditions, acquire pathologic significance, so that we need not modify the etiologic importance of Fränkel's cocci in pneumonia.

The *influenza bacillus*[2] was described by R. Pfeiffer as the cause of the great epidemic of influenza in the early part of the nineties. It is of some diagnostic importance (Fig. 215).

It is a very small bacillus; is hard to stain; is sometimes arranged in pairs; is always found in fresh attacks of true influenza, and usually in very great numbers, free or included in the leukocytes. In parts of the sputum it may be obtained practically in pure cultures. Pfeiffer obtained the best-stained specimens by allowing dry preparations to float five or ten minutes on a very thin, pale-red solution of carbol fuchsin. Influenza bacilli are only two to three times as long as they are broad. They rarely form threads. The ends are rounded off, and if the stain is slight will be somewhat more deeply stained than the center, so that they may resemble a diplococcus.

This appearance may also be produced if two short rods lie side by

[1] [Friedländer's micro-organism is a species of bacillus belonging to the same group as the various species designated Bacillus mucosus capsulatus.—C. N.]

[2] *Zeits. f. Hyg.*, xiii., p. 357 *et seq.*, 1893.

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side. Influenza bacilli have no capsule and are not mobile. They do not stain by Gram's method. The bacillus of influenza has not been found in late years in so-called attacks of influenza, but great numbers

FIG. 215.—Influenza bacilli (× 1000) (after Pfeiffer).

of Fränkel's pneumococci, even when there were no signs of pneumonia, but only bronchitis. They seem to be of etiologic importance. If the influenza observed in the nineties be regarded as a specific disease

FIG. 216.—Saprophytic bacteria of the mouth, from the gums. The large rods are Leptothrix buccalis (× about 800) (after Fränkel).

produced by the influenza bacillus, which does not seem to the author to have been absolutely established, it might be well to discontinue using the term influenza for these pneumococcic infections and common catarrhal conditions.

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Influenza bacilli can be cultivated only on media containing hemoglobin, a fact which may serve to differentiate them, for instance, from the colon bacillus. An appropriate medium can be easily prepared by spreading a little blood with the infected material upon the surface of ordinary agar. In regard to the character of the colonies and the results of animal inoculations, we must refer to Pfeiffer's original work.

Leptothrix buccalis and *other saprophytic bacteria* are often observed in fresh sputum, for they grow in the normal mouth and there become mixed with the expectoration (Fig. 216). They may multiply in the lung and appear very abundantly in the sputum, especially in putrid diseases of the lung. They do not seem to have any special pathologic significance, except that they may play a very considerable part in the decomposition of secretions. The bacillus known as Leptothrix buccalis or pulmonalis may be easily recognized by its size and shape, and its peculiarity of frequently (not always) staining blue with Lugol's solu-

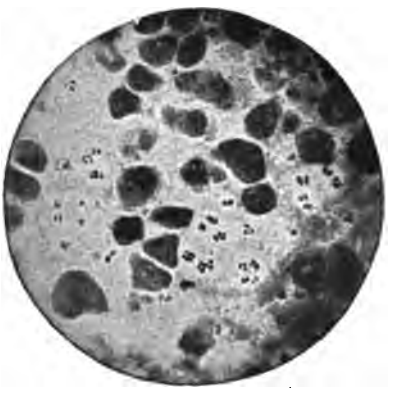

FIG. 217.—Micrococcus tetragenus (× about 800) (after Fränkel).

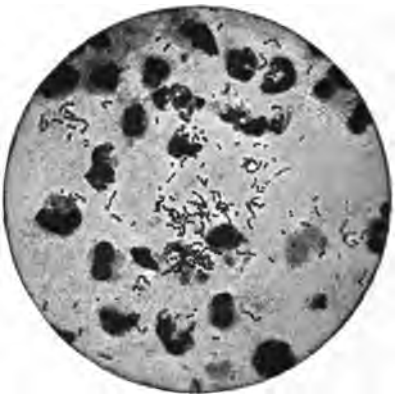

FIG. 218.—Streptococcus pyogenes (× about 800) (after Fränkel).

tion. This latter peculiarity seems to depend upon the kind of medium.

The *Micrococcus tetragenus* may be found in the sputum in various conditions: in bronchitis, in cavities of the lung especially, and sometimes in the sputum of healthy people. It is pathogenic in animals, and may also produce suppuration in man. Very possibly it aids the tubercle bacillus in destroying the lung tissue in phthisis.

The same possibility applies to the *streptococci* and *staphylococci*, which are (Fig. 218) found not infrequently in very great numbers in tubercular sputum.

The *Micrococcus catarrhalis* is almost always found in the nasal secretion in rhinitis, and is quite frequently observed in the sputum from the lungs. It differs from the ordinary staphylococci by its much larger size, by its decolorization by Gram's method, and by its position in the cells. With the exception of its large size, it closely resembles the gonococcus, and, like the ordinary staphylococcus, is frequently grouped in pairs as a diplococcus, the contiguous sides of the paired cocci being distinctly concave.

In recent years bubonic plague has occasionally occurred in regions in which the disease is not endemic, and the *pest bacillus* must consequently be described. Although bacteriologic laboratories will usually make the bacteriologic diagnosis

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of bubonic plague, it will occasionally happen that the physician will be forced to make his own diagnosis, and in such cases he should be familiar with the method of bacteriologic examination. This is particularly necessary in the cases in which the lungs are affected, since the clinical course of the disease may then almost exactly correspond to that of an ordinary catarrhal or croupous pneu-

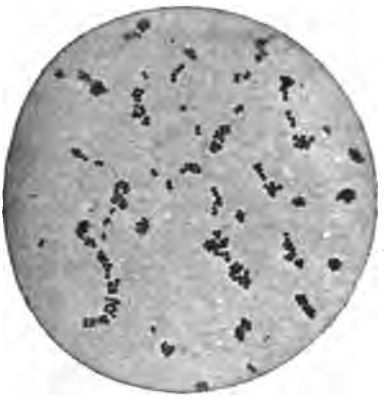

FIG. 219.—Staphylococcus pyogenes aureus (culture) (× 1000) (after Weichselbaum).

FIG. 220.—Aspergillus fumigatus (× about 350) (after Fränkel).

monia. Enormous quantities of pest bacilli may be found between the red blood-corpuscles in the hemorrhagic sputum of such cases. The pest bacilli may also be present in the expectoration in the ordinary bubonic plague. Czaplewsky describes them as follows: They are usually short rods with rounded ends, their length being two to three times greater than their breadth. They are occasionally so short, however, that they resemble cocci. They are readily stained, the poles

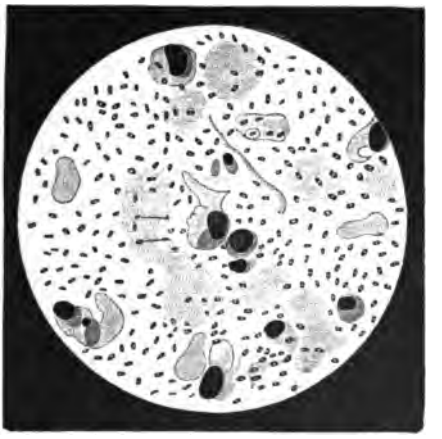

FIG. 221.—Pest bacilli in a smear made from a bubo; stained with methylene-blue (Kolle).

appearing darker than the central portion, which may be pale or even colorless (see Fig. 221). This polar stain may be most beautifully obtained by staining the bacilli for a half-minute in dilute borax-methylene-blue (stock solution with 2 per cent. methylene-blue and 5 per cent. borax). The pest bacilli are decolorized by Gram's method. They are non-motile. For methods of cultivation,

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the reader is referred to the official communications of the German government for the bacteriologic diagnosis of bubonic plague (published by J. Springer, 1902), and to the works of Kossel and Overbeck (from the Imperial Health Office, 1901, I. Heft).

In pulmonary glanders the *Bacilli mallei* may be found in the sputum. They cannot be sufficiently identified by microscopic examination, but require to be grown in cultures, and particularly to be inoculated into the lower animals. They are of about the same length as tubercle bacilli, but are thicker and have rounded ends. They are motile, and are decolorized by Gram's method. Particularly characteristic is an alkaline potato culture which, after two days at a temperature of 37° C., presents slimy drop-like colonies, varying in color from honey yellow to copper red, about which the potato is darkly stained. Experiments upon animals are performed as follows: A guinea-pig is inoculated subcutaneously with the suspicious material. From the resulting swollen glands a piece is excised and introduced into the peritoneal cavity of a second male guinea-pig. Upon the second or third day this second animal develops a characteristic orchitis produced by glanders. Since other bacteria may also produce an orchitis, the morphologic and cultural characteristics of the Bacillus mallei must also be taken into consideration.

In the so-called wool-sorters' disease, which is nothing else than pulmonary anthrax, the *Bacillus anthracis* may be found in the sputum (see Fig. 235). These bacilli are easily stained, and may be readily recognized by their size (length 5 to 10 μ, breadth 1 to 1.5 μ). The ends are frequently slightly concave, giving rise to the so-called bamboo form. They liquefy gelatin and grow out into long, waving chains, which develop endogenous, highly refracting spores. Mice quickly die after the subcutaneous inoculation of anthrax bacilli; the edematous fluid at the site of the inoculation, and the blood contain large numbers of the bacilli.

The occurrence of *typhoid bacilli* in the sputum is of slight diagnostic importance. It has been particularly observed in the pneumonia of typhoid fever. The author himself has seen a case of typhoid in which a serous pleuritis exudate containing large numbers of typhoid bacilli perforated into the respiratory passages and was expectorated.

The presence of *sarcinæ* in the sputum possesses a certain amount of interest. They resemble and are often confounded with the Micrococcus tetragenus, but are considerably larger. They are very rarely found in the sputum. True sarcinæ are best seen in unstained preparations. Fig. 141, *c* and *d*, gives some idea of their appearance. It has not as yet been definitely settled whether or not the sarcinæ of the stomach (described above) are identical with those of the lung. Sarcinæ have been found in the sputum, chiefly in gangrene of the lung, but also in tuberculosis, bronchitis, and pneumonia (pneumonomycosis sarcinica). Sarcinæ should probably be considered saprophytic, especially when we consider their occurrence in all of these conditions. The same type of sarcina may develop in the mucous membranes of the mouth and pharynx of debilitated patients, producing the grayish spots of pharyngo- or stomatomycosis, and from this source may be found in the expectoration.

It has recently been found that various kinds of molds belonging to the genus *Aspergillus* (Figs. 220 and 222), or perhaps to the genus *Mucor*, may develop in the lung; but practically only when there is some coexisting cavity formation. The cavities where these molds have been found are almost exclusively devoid of odor. The relation of the molds to putrefactive bacteria observed outside of the body would seem to make it probable that the constant antagonism between these two kinds of organism continues in the interior of the body, so that the aspergilli protect a pulmonary cavity from putrefactive bacteria; and, conversely, the saprophytic bacilli found in the majority of cases protect the lung from becoming overgrown with some mold. Molds probably do not invade an otherwise healthy lung; but if they once settle there, they aid in the general destruction, as is definitely proved by the examination of the pathologic processes. Very likely they may even displace the primary pathogenic bacilli. Cases where *pneumonomycosis*

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aspergillina or *mucorina* seems to be primary can be explained in this way; they probably never occur primarily, for the spores of aspergillus are everywhere, and yet aspergillus mycosis is extremely rare. The only diagnostic criterion differentiating pneumonomycosis aspergillina or mucorina from ordinary bronchitis, phthisis, etc., is the demonstration of the molds in a fresh specimen of sputum (Figs. 220 and 222) (mycelium, spores, conidia). The varieties, as a rule, can be separated only by culture (see special text-books).

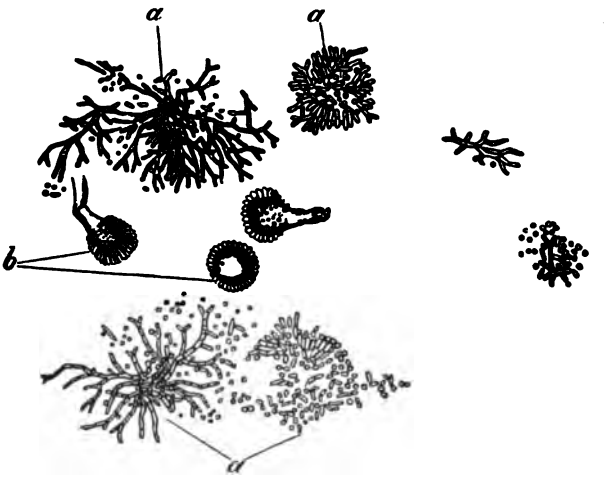

FIG. 222.—Aspergillus fumigatus of the lung (partly schematic): *a*, Mycelium of aspergillus in roset-like rays; *b*, sporangium (× 285) (after Weichselbaum).

In rare cases the *Oïdium albicans*, another mold, may develop in the lung. It is best detected in fresh unstained preparations of sputum (Fig. 223). It develops much more frequently upon the mucous membranes of the mouth, pharynx, and esophagus than in the lung. From any one of these sources it may be found in the sputum (Fig. 223).

The rare cases of *actinomycosis*, or disease caused by *ray fungi*, which occur in the lung should be mentioned. Their course clinically is similar to that of tuberculosis of the lung, except that instead of the tubercle bacilli this fungus is found.

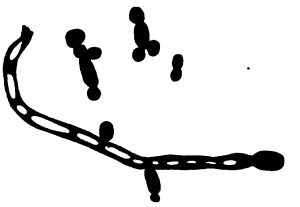

FIG. 223.—Oïdium albicans (× 400) (after Bizzozero).

The characteristic yellowish or grayish-green granules of actinomycosis can generally, but not always, be recognized with the naked eye (Fig. 224). The microscopic examination is not always decisive, either. In some cases the microscopic rosets of the fungus are to be found; in others only the branching threads staining by Gram's method (Fig. 225), with or without club-like ends. Occasionally even coccus-like structures are found, the actinomyces belonging to the pleomorphic class of streptotriches. Generally speaking, microscopic elements of this sort make the diagnosis of actinomycosis very probable; still, it is wise to

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follow Silberschmidt's[1] advice, and prepare aërobic and anaërobic bouillon and agar cultures for the purpose of differentiation.

The direct examination of fresh sputum expectorated in the presence of the observer is of much more value for differentiating micro-organisms

FIG. 224.—Young actinomyces granule (prepared section). In the middle, the mycelium; on the edge, the clubs, which become much thicker with maturity. From a preparation stained by Gram's method (× 530) (after Weichselbaum).

than the much overrated culture methods (see Examination for Diphtheria Bacilli), because most of the pathogenic micro-organisms which are found in the sputa are also present and harmless in the mouths of

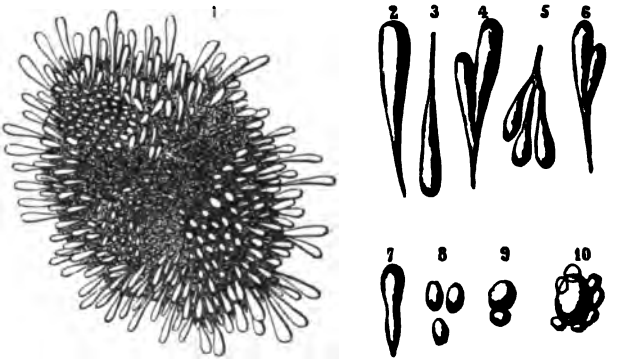

FIG. 225.—Actinomyces from a tumor of the lower jaw of a cow: 1, An entire granule (× 500); 2, 3, 4, 5, 6, 7, different forms of clubs; 8, 9, 10, round elements (× 1000) (after Macé).

healthy people, and cultures may lead to an erroneous impression concerning their preponderance over the other bacteria. Moreover, certain pathogenic micro-organisms are not readily cultivated from the sputum.

[1] *Zeits. f. Hyg. u Infectionskrankh.*, 1901, vol. xxxvii., p. 345.

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CHEMICAL EXAMINATION OF THE SPUTUM.

ALBUMIN IN THE SPUTUM.

Wagner[1] calls attention to the diagnostic importance of the presence of albumin in the sputum. The greater the quantity of albumin in the expectoration, the more marked will be the inflammatory process to which the sputum owes its origin. From a practical standpoint, Wagner directs attention to the fact that the sputum of a simple chronic bronchitis is always practically free from albumin, while that of pulmonary tuberculosis is usually characterized by a distinct quantity of albumin.

To demonstrate this albumin, a measured quantity of the expectoration is placed in a glass flask with a 3 per cent. solution of acetic acid, and violently agitated until the mucus is decomposed. The mixture is filtered, and the filtrate is washed with 3 per cent. acetic acid. The filtrate, which contains the albumoses and the albumin of the sputum without the mucin, is now treated with sodium hydroxid until it is only slightly acid in reaction. If necessary concentrated saline solution may be added, after which the albumin may be coagulated by boiling. The albumin may also be precipitated from the strong acetic acid solution by potassium ferrocyanid. In either case the resulting precipitate gives a qualitative and a quantitative test for the amount of albumin in the sputum. Esbach's method, as applied to the urine (see p. 505 *et seq.*), may also be employed for an approximate quantitative estimation of the amount of albumin.

CHIEF CHARACTERISTICS OF THE MOST IMPORTANT TYPES OF SPUTA.

SPUTUM IN CATARRH OR BRONCHITIS.

Ordinarily, catarrhal sputum is essentially mucopurulent without any other admixtures. In the beginning of an acute bronchial catarrh the mucus predominates and the sputum is scant. After a few days the expectoration becomes more abundant, less tenacious, and considerably more purulent, the general discomfort at the same time lessening. As recovery progresses, the amount of pus diminishes as the quantity of expectoration becomes less, until the sputum disappears entirely. In chronic bronchitis the nature of the expectoration may vary considerably, being at times more, at times less purulent. Patients usually feel more comfortable when the sputum is fairly abundant; whereas the discomfort is increased when the secretion ceases entirely or becomes excessive.

SPUTUM IN FIBRINOUS OR CROUPOUS BRONCHITIS.

The sputum of croupous differs from that of ordinary bronchitis. Fibrinous coagula in the shape of casts of the bronchi appear in the sputum from time to time, usually associated with more or less blood. Charcot's crystals are very frequently found in these formations. The larger coagula are apt to be expectorated with very severe paroxysms of coughing, which have usually been preceded by more or less dyspnea.

[1] *Deutsch. Arch. f. klin. Med.*, vol. lxxv., parts 3 and 4, 1902.

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SPUTUM IN ORDINARY PULMONARY TUBERCULOSIS.

Macroscopically, this cannot be absolutely distinguished from simple catarrhal sputum. Any type may be present, from a purely mucous to an almost purely purulent sputum. In well-advanced ulcerative types of phthisis the amount of pus is usually considerable. Friable opaque white particles always make one suspect tuberculosis. The sputum of tubercular patients frequently has a very bad odor, especially if there are cavities with stagnating contents. A positive diagnosis can be made only when the tubercle bacillus is found. If all other destructive processes of the lung can be eliminated, elastic fibers are very suggestive. The abundance of these morphologic elements by no means indicates the severity of the case. There are very severe cases of tuberculosis of the lungs where no bacilli and no elastic fibers can be found. These are frequently very malignant and acute cases, where the constitution is undermined before disintegration of the pulmonary infiltration begins, or cases where miliary tuberculosis is alone responsible for the grave symptoms. Again, if the catarrhal secretion is profuse, which is common especially in unfavorable cases, the number of tubercle bacilli in the sputum will seem few on account of the dilution. On the other hand, we not uncommonly find tubercle bacilli and elastic fibers in the sputum of early tuberculosis of the lung where physical examination reveals no or only slight signs. The significance of tubercle bacilli in cases of this sort is of very decided importance. Naturally, then, any variation in the number of elastic fibers or of tubercle bacilli which are present in the same individual cannot be interpreted, in and of itself, as indicating any marked change in the course of the disease. In judging the value of therapeutic measures, such false interpretations have frequently been applied.

SPUTUM IN ACUTE MILIARY TUBERCULOSIS.

This presents the characteristics of an ordinary catarrhal sputum, and if not complicated by ulcerative phthisis is without bacilli. There may be no sputum at all.

SPUTUM IN CROUPOUS PNEUMONIA.

The characteristic feature of the sputum of croupous pneumonia is the blood-content. The blood is usually uniformly mixed in a glairy menstruum, the sputum appearing almost transparent and homogeneous. Not infrequently, however, particles of sputum free from blood alternate with hemorrhagic streaks and spots or with considerable quantities of almost pure blood. In some cases the original color of the blood is almost completely preserved; in others, especially when the blood is uniformly mixed with the glairy mass, the blood-pigment is modified to a yellowish or brownish red, as previously described (*rusty sputum, sputa crocea*). Blood-corpuscles can be recognized microscopically in any pneumonic sputum, although they are almost completely laked out. The peculiar change of blood-pigment producing green and yellow sputa has been already considered (p. 579). Pneumonia not infrequently gives rise to jaundice (p. 43), in which case the sputum is apt to be yellow or greenish and show Gmelin's reaction (p. 472). Fibrin coagula are not uncommonly found in the sputum of pneumonia; we have already considered (p. 585) their peculiarities and the method of demonstrating them. If a catarrhal bronchitis of the larger bronchi is associated with the croupous pneumonia and a fibrinous bronchitis of the smaller bronchi with which it is almost always accompanied, then the purely pneumonic sputum will be mixed with catarrhal elements. Pneumonic sputum is usually very viscid, on account of the nuclein it contains, so much so that the spit cup may be completely inverted without spilling the contents. Thin liquid sputum, especially if it is abundant and dark reddish brown in color, is often an unfavorable sign in pneumonia, as it frequently indicates the beginning of pulmonary edema. Such a dark sputum has been called "prune-juice" sputum, on account of its appearance. However, we should not make a prognosis from the nature of the sputum alone. A thin liquid sputum is an unfavorable sign only when the other symp-

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toms are very urgent, for not infrequently a liquid sputum indicates the beginning of resolution. Fränkel's pneumococcus can always be demonstrated in the sputum of croupous pneumonia (Fig. 214).

SPUTUM IN BRONCHOPNEUMONIA.

We include here deglutition and hypostatic pneumonias. In these disorders the sputum sometimes resembles bronchitic sputum or, like that of croupous pneumonia, it contains blood. This is easily understood, for it is frequently very difficult, aside from the macroscopic distribution, to distinguish broncho- from croupous pneumonia histologically, and, like the latter, bronchopneumonia may also be hemorrhagic, with a more or less fibrinous exudate. The bacteriology of bronchopneumonia sputum may vary considerably. Fränkel's pneumococci are not infrequently found, as well as many other pathogenic micro-organisms.

SPUTUM IN PULMONARY GANGRENE.

This is characterized mainly by its intensely disagreeable odor. It is usually very abundant and liquid, and of a dark dirty-greenish or brown color. Small pieces of necrotic lung tissue can be found macroscopically in it, and besides them constituents characteristic of hemorrhagic, pneumonic, catarrhal, or purulent sputum. Odorless gangrene is very uncommon. The author once demonstrated an abundance of sarcinæ in the necrotic portions from a case of this sort. If allowed to stand, the sputum of pulmonary gangrene usually separates to form layers. The uppermost layer contains mucus and necrotic portions which, on account of the air contained, float. The second layer is liquid, and the sediment consists of pus corpuscles and necrotic detritus. Besides the ordinary elements of sputum, abundant bacteria of decomposition, fatty crystals, cholesterin, leucin and tyrosin crystals (Fig. 211, *c*), pigment, and bits of destroyed lung tissue are seen microscopically. Elastic fibers may be present.

SPUTUM IN PULMONARY ABSCESS.

This is essentially a purulent sputum, and often has a very foul odor. Characteristic for the pus when mixed with water is its fine, shredded, flocculent appearance (see p. 584). If catarrh exists at the same time, the pus is mixed with more or less abundant catarrhal sputum, provided the abscess has perforated slowly. If perforation takes place suddenly, large quantities of pure pus are expectorated, which contains microscopically elastic fibers, and hematoidin, cholesterin, and fatty crystals, beside lung pigment and bacteria (Fig. 211).

SPUTUM IN PERFORATING EMPYEMA.

This usually resembles very closely the sputum of a pulmonary abscess. Elastic fibers, if present, occur in very small numbers. Hematoidin and other crystals may be present. An odorless empyema may smell very badly after perforation, because the empyema cavity becomes infected with saprophytic bacteria from the lung.

SPUTUM IN PUTRID BRONCHITIS.

This is more or less purulent, with a foul odor, and abundant bacteria, but without any elastic fibers.

SPUTUM IN BRONCHIECTASIS.

The sputum of bronchiectasis with circumscribed cavities is mucopurulent, but it is often more profuse than is a simple catarrhal sputum, and it is expectorated periodically. Expectoration is easier in certain positions of the body, according to the location of the bronchiectasis. The odor is frequently foul. Its

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microscopic peculiarities are quite like those of the sputum of putrid bronchitis. In diffuse bronchiectasis the sputum resembles sometimes the above type, at other times that of simple catarrhal bronchitis. The odor may or may not be foul.

SEROUS SPUTUM IN PULMONARY EDEMA AND IN PERFORATING SEROUS PLEURISY.

The sputum in edema of the lung is usually colorless or faintly tinged with blood, foamy and somewhat opaque, and usually profuse. It separates, upon standing, into a lower liquid and an upper foamy layer. The latter is quite abundant. At the bottom there may settle a very thin layer of morphologic constituents, which consist partly of white blood-corpuscles and partly of other elements from some other affection of the lung present at the same time (bronchitis, pneumonia). Otherwise the sputum of pulmonary edema consists chiefly of pure or slightly bloody serum which contains a moderate amount of albumin (demonstrated by boiling and addition of acid). The sputum raised after a paracentesis for pleurisy may present all the characteristics of that from a case of pulmonary edema. The French call this expectoration "albumineuse." It is nothing more than the product of an acute edema of the lung following the sudden removal of pressure from the pulmonary vessels. Fortunately, however, paracentesis is not generally followed by any severe symptoms. If a serous pleural exudation perforates the lung and is then expectorated, the sputum will resemble that in edema.[1] This is, of course, very rare, but it does occur. The sputum from a pleurisy can in such cases be differentiated by means of its greater albumin content from the sputum in edema, the fluid, as a rule, becoming solid after addition of acid and boiling.

SPUTUM IN VARIOUS KINDS OF PULMONARY HEMORRHAGE AND IN HEMORRHAGIC INFARCTION OF THE LUNG.

In marked hemorrhages of the lung proper, such as occur after injury, or erosion of vessels by tuberculosis, or new growths, the sputum may consist chiefly of blood. It is usually bright red, whether it comes from the pulmonary artery or from one of the veins, because the dark venous blood of the pulmonary artery in its course along the bronchi has usually been sufficiently aërated to be rendered more or less arterial. The admixture of the blood with air in the lung causes the frothy nature of the expectoration. This frothy appearance and bright red color, and the fact that the blood is coughed up, are usually characteristic enough to establish the diagnosis that the seat of the hemorrhage is in the lung.

It is, however, sometimes difficult to determine whether the blood comes from the lung or from the digestive tract, especially from the stomach. Differentiation is usually easy, provided the physician himself sees the hemorrhage occur, but it is often very hard to judge from the patient's statements. In the first place, the patient takes no special note of his condition, on account of excitement, and is not sure whether he coughed or vomited the blood; and, in the second place, vomiting may be produced by the violent paroxysms of coughing accompanying a pulmonary hemorrhage; or, on the other hand, vomiting blood from the stomach may start a fit of coughing, on account of the aspiration of blood into the larynx.

When in doubt, reliance must be placed upon the objective examination of the blood. Frothy bright-red blood favors the diagnosis of a hemorrhage from the lung. Blood from the stomach is more apt to be dark (methemoglobin and hematin), partly coagulated and not foamy, because it has been more or less digested. Bright-red blood may come from a hemorrhage from the stomach where an ulcer has eroded an artery of the stomach and the blood is vomited in such large quantities that it is still bright red without having undergone any

[1] Sahli, "Ueber die perforation seröser Exsudate," etc., *Mittheilungen aus klin. u. med. Instituten der Schweiz*, 1894, vol. i., part 9.

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change. On the other hand, in exceptional cases dark blood is observed in a hemorrhage from the lung when the eroded vessel is a large branch of the pulmonary artery, from which the dark venous blood has been expectorated so quickly that it has undergone no change.

The reaction is frequently mentioned as a differential point between blood from the lungs and that from the stomach. The former is said to be alkaline; the latter acid, on account of the admixture of gastric juice. But this is true only when the stomach contains a considerable quantity of acid secretion at the time that the blood is vomited.

No one point can be considered absolute in differentiating between a hemorrhage from the lung and one from the stomach. Nevertheless, it is usually not very difficult to decide in any given case. Examination of the patient is more important than is the consistence of the blood, and especially a careful attention to the symptoms preceding or following the hemorrhage. A patient with hemorrhage from the stomach usually gives a history of previous gastric difficulty, or such a disturbance can be determined after the hemorrhage has taken place. Patients who have had a hemorrhage from the stomach usually pass blood in their movements. On the other hand, a patient with hemorrhage from the lung has usually been subject to a cough before or after the bleeding. The expectoration is usually blood-tinged for days after the hemorrhage, either blood red or brownish. If all of these points are taken into consideration, the diagnosis will not, as a rule, be difficult.

Slight hemorrhages from the lung, as contrasted with a true *hemoptysis*, will cause more or less tinging of a catarrhal sputum. The blood is not as intimately mixed with the expectoration as it is in the sputum of pneumonia, but occurs in streaks. A sputum of this sort does not always come from the lung, and so often causes patients quite unnecessary worry. It is very uncommon to have hemoptysis proper from the larynx or trachea (for the simple reason that there are no large vessels there). But slight streak-like hemorrhages may arise from the small vessels in the mucous membrane of the larger bronchi, the trachea, the larynx, or of the pharynx itself. Bronchitis may sometimes be hemorrhagic. During the last influenza epidemic some patients expectorated for weeks a blood-tinged catarrhal sputum without any especial disturbance of the general condition. It is not always possible to distinguish between these various conditions. Besides this, it must be mentioned that the clots coming from a nose-bleed which has occurred during sleep may become mixed with the sputum in the pharynx as the blood is swallowed. Such sputum may be erroneously attributed to a pulmonary hemorrhage. A careful examination of the anterior and posterior nasal passages will generally settle the diagnosis.

Most cases of hemorrhagic infarction of the lung present a typical sputum, dark and bloody and resembling pure blood. In its tenacious consistence, however, it resembles the sputum of pneumonia. In fact, the sputum in these cases is an intimate mixture of blood with a tenacious exudation. Besides this typical form, the sputum of infarction shows many varieties, in some cases resembling the sputum of tubercular hemoptysis, in other cases that of pneumonia.

EXAMINATION OF THE BLOOD.

THE examination of the blood furnishes a number of important diagnostic data. Several of the methods are so simple that they can be employed at the bedside in daily practice, whereas others are too complicated for such a purpose.

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METHOD OF OBTAINING BLOOD FOR EXAMINATION.

A few drops are sufficient for microscopic examination for the determination of the coagulability and the estimation of the alkalinity of the blood. This may be obtained by puncturing the tip of a patient's finger or the lobe of his ear with a needle or sharp lancet.

FIG. 226.—Francke's needle for removing blood for clinical purposes (about one-half its natural size).

Francke's instrument[1] (Fig. 226) pierces the skin very rapidly with a narrow needle-like lancet to a depth which can be readily regulated. It does not cause serious pain. Pressing on the lever suddenly releases a spiral spring in the interior of the instrument, which drives the point of the lancet into the skin to a depth regulated by the guard (*d*). The blade may be easily removed and cleansed. The instrument may be adjusted so as to obtain sufficient blood where a larger quantity is needed (alkalinity). The skin should first carefully be cleansed (with alcohol) and then thoroughly dried.

The writer has constructed a simpler, cheaper, and more compact instrument, in which the puncture is also made by a lancet. It is, however, made by manual instead of spring pressure; its depth is regulated by an adjustable cover. Since the depth of the puncture cannot exceed the length of the lancet exposed, the stab may be very quickly made, and is no more disagreeable to the patient than when Francke's needle is employed.

If several cubic centimeters of blood are required, wet-cupping may be employed; but since the blood coagulates so rapidly that certain methods of investigation are difficult of execution, it is better to use a cannula inserted into a vein. An ordinary hypodermic syringe with a

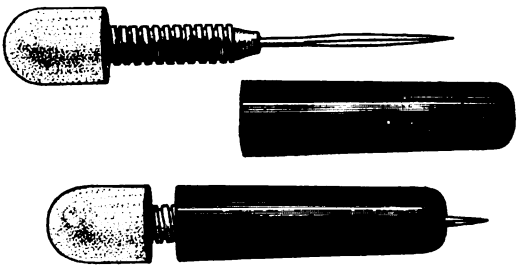

FIG. 227.—Blood-needle.

large needle may be all that is required; but when quite large amounts are necessary they are best obtained by a larger cannula, through which the blood flows directly from the vein. A piece of tubing is attached to the end of the cannula. The cannula should have a lumen of at least

[1] See *Deutsch. med. Woch.*, 1889, No. 2, p. 27. [A similar one is made by Baker & Co., of London. It is smaller and very satisfactory (see Fig. 227).—ED.].

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1 mm. Its length should not exceed 5 cm., otherwise the flow of blood will soon be interrupted by coagulation; moreover, a longer cannula is difficult of manipulation. Whether a needle or a larger cannula is used, it should be very sharp, as the vein may easily elude the puncture. In introducing the cannula the median vein is selected, a fillet is placed about the arm, and the instrument is thrust toward the periphery, and as nearly parallel as possible to the cutaneous surface. The writer has convinced himself that this method is not adapted to the quantitative examination of the blood, since the stasis produced by the fillet quickly changes the composition of the blood withdrawn, particularly in respect to the proportion between the solid constituents and the water. This condition of affairs may be avoided, however, by loosening the fillet before the blood is withdrawn.

QUANTITY OF THE BLOOD; DIAGNOSIS OF HYDREMIC PLETHORA.

As yet we know no reliable method of determining the exact quantity of blood even in animals. In man it is estimated to be about one-thirteenth of the body weight, but it is very probable that this ratio may be changed under pathologic conditions. There are as yet no positive data, although it would be of the greatest clinical interest to have accurate information upon this point. After acute losses of blood the quantity doubtless remains diminished; perhaps, however, only for a short time. Furthermore, when the body is deprived of water, the liquid portion of the blood is utilized, the blood becomes somewhat thicker, and is diminished in quantity. This probably occurs in cholera and in the acute infantile diarrheas, judging from the great amount of water lost, the slight distention of the vessels, and the increased hemoglobin. Quantitative spectroscopic examinations and hemoglobin estimations have recently shown that the concentration of the blood increases after marked perspiration and after the use of saline cathartics and diuretics, due to the loss of water.

The demonstration of hydremic plethora—*i. e.*, an increased quantity of blood due to the retention of water—is of particular clinical interest. This demonstration is sometimes possible from a determination of the hemoglobin percentage (see p. 616, *et seq.*), which falls when the water is retained. It is clear, however, that it will be possible to base a diagnosis of hydremic plethora upon the oligochromemic character of the blood only when the latter develops acutely under the observation of the physician, and when hemorrhage or other common causes of anemia can be excluded. With these limitations, the fall of the hemoglobin percentage is a useful indication of hydremic plethora, since hemoglobin cannot quickly enter or leave the vessels, as can the soluble constituents of the blood. It consequently follows that hydremic plethora is frequently to be recognized only by the lowering of the hemoglobin percentage, while the specific gravity, the dry residue, and the osmotic pressure remain normal, since the dissolved constituents, particularly the salts, may remain in the blood. In cases of nephritis, for example, the hydremo-plethoric blood very usually exhibits even an increased osmotic pressure (see p. 669 *et seq*).

The conditions of so-called "bloodlessness"—the various types of *anemia*—are by no means due to a deficient quantity of blood, as has been previously supposed, and as is indicated by the name. But the constant sign of anemia (oligochromemia)—pallor of the blood—is due to a diminution in the percentage of blood-pigment. Traumatic anemia, due to loss of blood, is, of course, at the first associated with a diminution in the quantity; but even this condition is rapidly changed to a simple oligochromemia, the blood lost being rapidly replaced by absorption of lymph from the tissues.

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SPECIFIC GRAVITY OF THE BLOOD.

The specific weight of the blood may be determined in two ways—one the "areometric" method, the other the "pycnometric." In the *areometric method* (Roy, v. Jaksch, Devoto, *et al.*),[1] a drop of blood is placed in fluids of different but known specific gravity—*i. e.*, in different mixtures of water and glycerin. The fluid is then so mixed that the drop remains suspended, when the specific gravity will be the same as that of the fluid. The influence of coagulation and diffusion militates against this method, and, besides, it is difficult to obtain a great many drops of blood successively from one and the same patient; it has, however, the advantage that it may be performed at the bedside, without any analytic balance.

Hammerschlag's Method.—Hammerschlag[2] modified this method by placing a mixture of benzol chloroform, of an average specific gravity of 1050 to 1060, in a test tube or in a urinometer. A drop of blood is then allowed to fall gently into this mixture, and, according to whether the drop floats or sinks, benzol or chloroform is added until the drop is exactly suspended. The drop of blood can then be easily removed by filtering through a piece of linen, and the specific gravity of the mixture determined with the areometer. The fluid may be saved for further use.

In the *pycnometric method*, Schmalz[3] employs a capillary tube (capillary pycnometer) 1½ mm. in internal diameter and 12 cm. long, slightly constricted at the ends so as to easily retain its contents. This tube is dried, filled with distilled water, and then weighed. It is then carefully dried with alcohol and ether, filled with the blood to be examined, and weighed again. If c equals the weight of the empty capillary tube, c' the weight of the capillary tube plus the water, and c'' the weight of the capillary tube plus the blood, then $c'-c$ will be equal to the weight of the water, and $c''-c$ will be the weight of an equal volume of blood. Therefore the specific weight of the blood will be $\frac{c''-c}{c'-c}$.

Experiments at the Berne Clinic have proved that this method is easy and accurate if scales weighing to $\frac{1}{10}$ mg. are used.

The results of determining the specific gravity of the blood thus far have shown that it is diminished in all anemic conditions (oligochromemia), and in some other cachectic conditions (nephritis, digestive disturbances), although the percentage of hemoglobin need not be diminished. The normal figures vary between 10,455 and 10,665. In men it averages about 10,550; in women, 10,535; and in children, 10,512 (Peiper).

Hammerschlag[4] advocates a method of estimating the specific gravity of the *blood-plasma*, based upon the above-described areometric principle. The blood is drawn into a capillary tube ¾ cm. (probably 3 to 4 cm. is meant, instead of ¾) long, and 1 to 2 mm. in diameter. The tube should be previously washed out with a 3 per cent. solution of oxalate of sodium, to prevent coagulation, and then blown out. Both ends of the tube are closed with wax, the tube is set in a vertical position, and the blood is allowed to settle. After the blood-corpuscles have separated from the plasma, the capillary tube is filed in two at the point where the two layers meet, and the plasma is examined according to Hammerschlag's areometric method. The admixture of the sodium oxalate solution causes a slight error in the result, but so slight, according to Hammerschlag, that it need not be considered (?). The specific gravity of blood-serum may be determined in a similar way. The blood is allowed to coagulate in the capillary tube without adding the oxalate, and to stand until the clot has pressed out a sufficient quantity of serum. According to Hammerschlag, the specific gravity of blood-serum varies but very little from that of the plasma. The specific gravity of plasma

[1] Roy, *Proc. Physiol. Soc.*, 1884; Devoto, *Zeits. f. Heilk.*, No. 11, p. 175, 1889; v. Jaksch, *Klin. Diagnostik*, 1892.
[2] *Zeits. f. klin. Med.*, vol. xx., p. 444, 1892.
[3] *Deutsch. Arch. f. klin. Med.* vol. xlvii., p. 145, 1890, and *Deutsch. med. Woch.*, 1891, No. 17, p. 555.
[4] *Zeits. f. klin. Med.*, vol. xxi., p. 475, 1892.

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in a healthy individual ranges from 1029 to 1032. It is diminished in conditions associated with hydrops, especially in nephritis. In these conditions, however, the specific gravity of the plasma may be increased, since in addition to water proportionately larger quantities of the solid constituents may be retained in the blood.

REACTION OF THE BLOOD.

The normal reaction of the blood is alkaline, the degree of alkalinity varying under pathologic conditions. According to Cantani, blood may become acid in cholera.

The peculiar color of blood renders titration very difficult, so that an attempt has recently been made to estimate the degree of alkalinity by determining the amount of carbonic acid contained in the blood, on the ground that this amount depends essentially upon the alkalinity. Theoretically this deduction is questionable, and, moreover, the method of estimating carbonic acid is too complicated for clinical purposes and requires altogether too much blood.

TITRATION OF OPAQUE BLOOD (after Landois-v. Jaksch.)[1]

This method consists in a modified titration of minute quantities of blood. A series of tartaric acid solutions of known acidity is kept in stock. A measured small quantity of blood—for instance, 0.1 c.c.—is added consecutively to 1 c.c. of each of these acid solutions. They are stirred quickly, and the reaction is tested with some very sensitive litmus paper. The degree of acidity of the tartaric acid solution which is exactly neutralized by the blood indicates the alkalinity of the blood.

v. Jaksch employs the following eighteen test solutions to carry out this method:

Solution	1	contains in	1	c.c.	0.9	c.c.	$\frac{1}{100}$	Normal tartaric acid and	0.1	concent. solution of	Glauber's salt.	
"	2	"	"	1	"	0.8	"	"	0.2	"	"	"
"	3	"	"	1	"	0.7	"	"	0.3	"	"	"
	etc.					etc.			etc.	etc.	etc.	etc.
"	9	"	"	1	"	0.1	"	"	0.9	"	"	"
"	10	"	"	1	"	0.9	"	$\frac{1}{1000}$	0.1	"	"	"
"	11	"	"	1	"	0.8	"	"	0.2	"	"	"
	etc.					etc.			etc.	etc.	etc.	etc.
"	18	"	"	1	"	0.1	"	"	0.9	"	"	"

The Glauber's salt solution is added instead of distilled water to preserve the red blood-corpuscles and make the solution permanent.

v. Jaksch obtains the specimen of blood by a wet-cup. Miss Freundberg undertook to determine the alkalinity of the blood in cases at the Bern Clinic. She employed Francke's needle (p. 610), but used only 0.05 c.c. of blood, because it was difficult to get 0.1 c.c. from so small a puncture. The blood is sucked into a capillary pipet. Immediately after removal, it is blown into a watch glass containing 0.5 c.c. of tartaric acid solution of an average degree of acidity. The mixture in the watch glass is stirred rapidly with a little glass rod, and the reaction tested with litmus paper. If acid, the experiment is repeated with a weaker solution until the point is reached when the amount of blood employed neutralizes the acid solution exactly. It is best not to proceed from one solution to the next, but to skip several, so that the outside limits between which the degree of acidity lies may be quickly obtained. Care must be taken to remove the blood as rapidly as possible, as a considerable amount of alkalinity is lost by chemical changes outside of the vascular system. Especial care must also be devoted to preparing a very sensitive litmus paper. (See text-books on Chemistry).[2] The method of using litmus paper is as follows: A drop of the mixture

[1] Landois, *Eulenburg's Realencyklopädie*, vol. iii., p. 161, 2d ed., 1895; v. Jaksch, *Zeits. f. klin. Med.*, vol. xiii., p. 350, 1887.

[2] *Cf.* Fresenius, *Qualitative Analyse*, 1895, 16th ed., p. 100, note.

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of blood and acid is dropped upon the paper with a glass rod, and the fluid immediately removed with a piece of white filter paper which is known to be neutral. The blood-pigment, which is the disturbing factor, is taken up by the filter paper, leaving an unmistakable spot on the litmus paper, provided neutralization is not complete. Only natural litmus paper can be used for testing, because the blood-pigment, even after it has been taken up with filter paper, renders it impossible or very difficult to recognize the reaction upon red acid litmus paper. For this reason blue litmus paper should be used. Successively weaker solutions of tartaric acid should be tried until no red spot remains.

It has not yet been definitely determined whether this method of blood-titration with litmus paper gives reliable results. The uncertainty depends upon the well-known peculiarity of litmus pigment to react amphoterically to mixtures of both alkaline phosphates contained in the blood (primary and secondary).

The principal fact obtained by v. Jaksch with this method, and corroborated by Miss Freudberg, is that the alkalinity of the blood diminishes in all anemias. v. Jaksch found the same to be true for diabetes mellitus, for uremia, and for fever.

The normal alkalinity of the blood determined in this way corresponds, according to v. Jaksch, to 0.26 to 0.30 sodium hydroxid, for 100 c.c. of blood.

TITRATION OF LAKED BLOOD (after Löwy and Engel).

In the method of titration described above it is doubtful how much the red blood-corpuscles, which are preserved by the addition of Glauber's salt, influence the reaction. For this reason Löwy employed lake blood. A test tube provided with a long, narrow, partially graduated neck, capable of containing 50 c.c., is filled with 45 c.c. of ¼ per cent. solution of oxalate of ammonia and 5 c.c. of blood. This solution of oxalate of ammonia lakes the blood, and at the same time prevents coagulation. Titration is performed with a $\frac{N}{25}$ tartaric acid solution (see above) and lackmoid[1] paper, which has been saturated with a concentrated solution of magnesium sulphate. The 5 c.c. of blood may be taken from a vein (see p. 610). With fresh human blood Löwy's values varied between 400 and 600 mg. NaOH pro 100 c.c. of blood. These are higher values than v. Jaksch obtained with unlaked blood. H. Strauss'[2] values were medium, 300 to 350 mg. pro 100 c.c. of blood.

S. Engel[3] modified this method by using a melangeur, diluting 0.05 of blood 100 times with neutral distilled water. With this mixture in a small beaker glass he then titrates from a buret, graded into $\frac{1}{20}$ c.c., dropping with great care a $\frac{N}{75}$ tartaric acid solution (1 gr. of tartaric acid to 1 liter of water). The end reaction is tested after the addition of each drop from the buret by wetting a piece of lackmoid paper with one drop of the solution from a glass rod, and then determining the moment when the yellowish drop (hemoglobin) shows a sharp red line around its margin. Engel's and Löwy's results correspond. The relation of lackmoid pigment to mixtures of primary and secondary alkaline phosphates should be more carefully examined before any judgment is formed as to the reliability of Löwy's or Engel's method. The close correspondence of their values with those of Salkowski is in favor of their accuracy.

From a practical standpoint, it is interesting to note that Magnus-Levy,[4] by the use of Löwy's method, in diabetic coma, has observed extraordinarily marked diminutions of the alkalinity of the blood—diminutions corresponding to 220 to 260 mg. NaOH to 100 c.c. of blood. He attributes this diminished alkalinity partly to neutralized carbonates and partly to the combination of the proteids with acids, which consequently lessens the quantity of acid to be neutralized by titration.

[1] *Cf.* Böckmann, *Chem.-techn. Untersuchungsmethoden*, Berlin, 1893.
[2] *Zeits. f. klin. Med.*, 1896, vol. xxx. [3] *Berlin klin. Woch.*, 1898, vol. xiv., p. 308.
[4] *Arch. f. exp. Pathol.*, vol. xlii, 1899, p. 197.

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SALKOWSKI'S METHOD OF DETERMINING THE ALKALINITY OF THE BLOOD.

Salkowski[1] has recently announced a method of determining the alkalinity of the blood which has the advantage of avoiding direct titration with all its associated difficulties (color of the blood, uncertainty of the indications in titration in phosphate mixtures). Schlösing's apparatus for determining the amount of ammonia in urine is employed. A known quantity of sulphate of ammonia is added to the blood the alkalinity of which is to be determined, and the amount of ammonia liberated from the alkalies of the blood is then estimated by Schlösing's method. The technic is as follows: Twenty grams of finely pulverized sulphate of ammonia are placed in the large lower dish of Schlösing's apparatus, and then dissolved by adding 20 c.c. of water. Ten cubic centimeters of a normal solution of sulphuric acid are placed in the upper dish containing the ammonia sulphate. It is well to wash the graduate in which the blood is measured with a 1 per cent. solution of sodium oxalate, so as to prevent coagulation. The blood is mixed with the solution of ammonia sulphate, and the globe placed over it as soon as possible.

After five or six days all the ammonia that has been freed has been taken up by the sulphuric acid, and can then be estimated by titration. The entire quantity of sulphuric acid must be taken into account in the titration because, as has been pointed out by Waldvogel, the volume of the acid may change on account of water being given off and combined with the sulphate of ammonia solution.

Waldvogel gives as normal values for men 350 to 400 mgr. NaHO pro 100 c.c. of blood; for women, 300 to 350. In febrile and also in anemic conditions the values were lower. The blood-corpuscles doubtless enter into the reaction in this method. The correspondence of the values found by Waldvogel with those obtained by Löwy would seem to support the value of the method.

Dare's Spectroscopic Method for the Determination of the Alkalinity of the Blood.

Dare[2] has recently suggested a method of determining the alkalinity of the blood by titrating laked blood with a $\frac{N}{1000}$ tartaric acid solution. The end reaction in this method is the disappearance of the characteristic spectrum of hemoglobin and its replacement by that of methemoglobin, the author assuming that the disappearance of the hemoglobin spectrum is coincident with neutralization. Further investigations are necessary, however, to determine the utility of this method, since it has not yet been conclusively shown that the hemoglobin is not destroyed before complete neutralization, and, furthermore, the spectroscopic changes dependent upon variations in the reaction are quite slow in their appearance.

COAGULATION TIME OF THE BLOOD.

The time elapsing before coagulation of blood occurs varies decidedly under pathologic conditions. It is difficult to give any constant normal figures, because coagulation depends so much upon external conditions, such as temperature, the shape of the vessel in which coagulation takes place, the amount of blood used, and the nature of the wound from which the blood is taken. This is the reason why so little is as yet known regarding pathologic variations.

H. Vierordt[3] recommends a method for determining the coagulation time of human blood which can be performed with very small amounts of blood. The technic is as follows: A capillary glass tube, about 5 cm. long and 1 mm. in internal diameter, is filled with blood for about one-half cm. From the other end there is introduced through the blood a white horsehair (about 10 cm. long), which has pre-

[1] *Centralbl. f. die med. Wissenschaften*, 1898. Ref. in *Maly's Jahresbericht*, 1898, and Waldvogel, *Deutsch. med. Woch.*, 1900, No. 43.

[2] *Proc. Pathol. Soc. of Philadelphia*, April, 1903.

[3] *Arch. der Heilk.*, 1878, vol. xix., p. 193.

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viously been carefully boiled with alcohol and ether, care being taken not to touch the end which comes in contact with the blood. At first no blood sticks to it, but the moment coagulation begins the horsehair will show a reddish discoloration. As soon as coagulation is complete, the horsehair will appear white when pushed further into the tube, or the entire quantity of blood will adhere to it as a compact clot. In order to determine more accurately the length of time necessary for complete coagulation, it has been the writer's custom to knot the posterior portion of the horsehair. At the moment of complete coagulation the entire coagulated blood-column may be drawn out of the tube as a compact mass when this knot is brought in contact with its posterior portion. Vierordt found the average time of coagulation with this method was nine minutes. The writer has found, however, that this is subject to extreme variations dependent upon the external temperature and the diameter of the capillary tube, so that the time of coagulation can be assumed to be pathologic only when it is compared with a control test in which normal blood is employed under exactly similar conditions. The time of coagulation is shortened by stasis, after transfusion, after hemorrhage, by hunger, and by the majority of diseases. The statement that the coagulation time of the blood is shortened when it is taken from an area of passive congestion is in marked contradiction to the fact that the blood in the corpse after suffocation is usually liquid. In the intervals between the hemorrhages of hemophilia the writer has found the coagulation time to be markedly prolonged. During the hemorrhage, however, this coagulation time may be even shorter than that observed in normal blood. For further details in this connection the reader is referred to the writer's article in the *Zeitschrift für klinische Medicin*, 1904–05—"Ueber das Wesen der Hämophilie."

DETERMINATION OF THE HEMOGLOBIN IN THE BLOOD.

A large number of methods have been recommended for use in this important part of blood-diagnosis. The methods available for clinical purposes indicate only the relative amount of hemoglobin contained in the blood examined compared with the normal. Clinically these relative values, expressed in percentages of the normal, are of primary interest. It is very easy to obtain from them absolute figures if the normal amount of hemoglobin contained in the blood is known. This is in a normal adult 13 to 14 gm. in 100 c.c. of blood. As hemoglobin contains about 0.4 per cent. of iron, this would correspond to about 0.05 per cent. of iron content. Leichtenstern found the following values for the hemoglobin content at the various ages, expressed in grams per 100 c.c. of blood:

36 hours.	19.329	3 years.	10.971
2 days.	21.160	4 "	11.341
8 "	20.451	5 "	11.151
4 "	19.488	6–10 years.	11.796
8 "	17.869	11–15 "	11.701
10 "	17.129	16–20 "	13.034
14 "	16.124	21–25 "	13.870
3 weeks.	15.023	26–30 "	14.727
4 "	15.362	31–35 "	15.013
10 "	14.293	36–40 "	14.685
12 "	13.828	41–65 "	14.420
14 "	14.388	46–50 "	12.484
20 "	12.928	51–55 "	12.696
½–1 year.	11.373	56–60 "	13.150
2 "	11.151	Over 60 years.	14.790

In this table a large number of observations were made on middle-aged and old people, and average figures were taken; but for the earlier periods of life only single observations were made. Nevertheless the amount of hemoglobin contained in the blood in the first week of life is without doubt one-third larger than the

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average during the third and fourth decades. During the following weeks the quantity gradually diminishes, and remains one-fourth to one-fifth below the average from the second half of the first year to about puberty. From puberty to the forty-fifth year of life, it remains about at the average, and then gradually diminishes. These figures correspond very closely to the variations in the number of blood-corpuscles, except that they are somewhat more striking.

The percentages upon clinical hemoglobinometers corresponding to these absolute values can be readily calculated from the above table if 13 to 14 gm. of hemoglobin in 100 c.c. of blood is taken as 100 per cent. Stierlin[1] calculated these percentages, and found them to be as follows:

Newborn (1st to 3d day)	138.88	
½–5 years	76.58	78.44.
5–15 "	80.50	
15–25 "	88.88	
25–45 "	100.00	
45–60 "	87.50	

Hemoglobin determinations are of great value (any practitioner can employ the Gowers' instrument or Tallquist's book at the bedside). Only since we have been able to determine the amount of hemoglobin have we recognized that an individual who is very pale need not necessarily be anemic, and that the pallor of the skin of the face may be due to lack of cutaneous transparency, or to the fact that the skin contains only a very slight amount of blood. And only since we have been able to limit iron therapy to diseases where there is really a deficient amount of hemoglobin has it become rational therapy.

Clinically the anemias include those conditions in which there is deficient percentage of hemoglobin (oligochromemia). Increased percentages of hemoglobin up to from 110 to 120 per cent. are by no means uncommon in so-called full-blooded healthy individuals.

Numerous observations have established the fact that the hemoglobin percentage increases with increased altitude, just as does the number of blood-corpuscles (compare p. 628).

GOWERS' HEMOGLOBINOMETER.

This instrument (Fig. 228) consists of two glass tubes (*a* and *b*) about 11 cm. long and 0.8 cm. in diameter. One of them (*a*) contains 2 c.c. of standard picrocarmin solution, the shade of which corresponds as closely as possible to a 1 per cent. solution of normal blood. The other tube is closed at one end, and graduated so that the level to which 2 c.c. of fluid will reach corresponds to 100. If the calibers of the two are exactly alike, this will be exactly the level of the standard fluid in the other tube. Below the 100 mark the tube is subdivided into 100 equal parts, every ten of which are numbered. Each of these parts contain 20 c.mm. Both tubes can be held in a vertical position by setting them into a perforated rubber block (*e*). A capillary pipet (*c*) containing 20 mm., with a small rubber tube attached, is used for convenience in sucking up the blood. A non-graduated pipet (*d*) is used for making the appropriate dilution. It contains about 3 c.c., with an opening so small that water which has been sucked into it can only escape drop by drop.

The technic of estimating the amount of hemoglobin is as follows:

[1] Blutkörperchenzählungen und Haemoglobinbestimmungen bei Kindern, *Arch. f. klin Med.*, vol. xlv., 1889.

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Twenty c.mm. of blood are sucked up in the capillary pipet as quickly as possible, so as to avoid coagulation. The point of the pipet is wiped off, care being taken not to allow blood to escape from the lumen. Its contents are then quickly blown out into the graduated tube (*b*) partly filled with water. The blood and water are intimately mixed by stirring, and the pipet washed by repeated sucking up and blowing out of the fluid. Tube *b* with the blood-mixture and tube *a* with the colored solution are now set up side by side in the rubber block. A thin piece of white tissue paper is held behind the tubes, and their colors are compared by transmitted light. Water is added from the larger pipet, drop by drop, with repeated stirring, until the color in

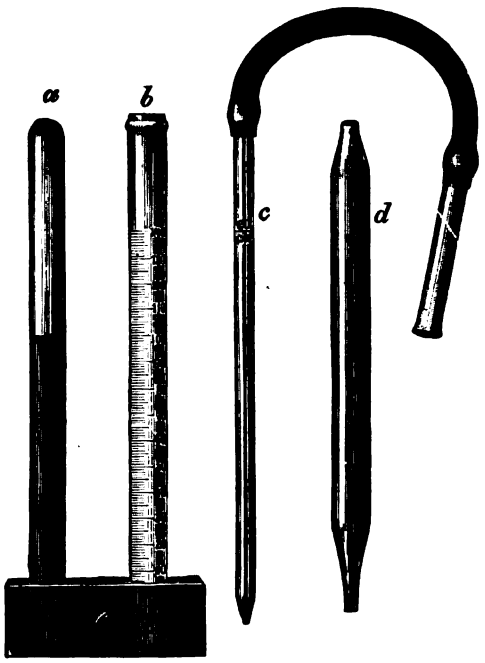

FIG. 228.—Gowers' hemoglobinometer (about two-thirds the natural size).

both tubes is as nearly alike as possible by transmitted light. The line to which the mixture reaches at this moment indicates the percentage of hemoglobin in the specimen of blood examined. This instrument is accurate within 5 or 10 per cent., provided that the shade of the standard solution exactly corresponds to the color of blood. The disadvantages of the instrument are, first, that it is quite difficult to make the standard solution so that it will exactly match the color of normal blood, and, secondly, that its color will change after a certain length of time. A special standard solution must be used if the examination is made by artificial light. In view of these disadvantages, the writer has constructed a new hemometer, in which some of the principles of Gower's instrument are retained, and which will meet with the requirements of daily prac-

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tice. The standard solution, however, is made up with blood-pigment instead of picrocarmin (see p. 621 *et seq.*).

FLEISCHL'S HEMOMETER WITH MIESCHER'S IMPROVEMENTS.

Fig. 229 represents Fleischl's instrument for the clinical determination of hemoglobin with Miescher's improvements. The principle of the instrument is as follows: The stand is like that of a microscope. In the central opening of the objective stage is placed a cylindric chamber, divided into two parts by a vertical partition. This chamber (*m*) has a glass floor. One-half (*a*) is filled with a diluted solution of the blood to be examined. The other half is filled with water. A glass wedge stained purple slides beneath the chamber by means of the screw T R. The glass wedge and the chamber are illuminated from below by a plaster-of-Paris reflector P S. The colors in the two halves of the chamber

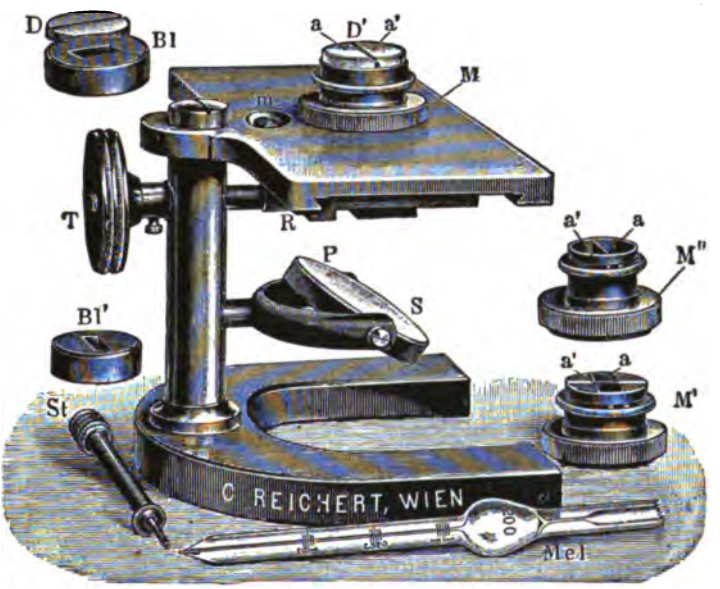

FIG. 229.—The new Fleischl-Miescher hemometer.

are compared from above by transmitted light. The glass wedge is then adjusted until both sides of the chamber show the same shade—*i. e.*, until the portion of the glass wedge underneath the chamber filled with water has the color of the blood-solution to be examined. The position of the wedge is read off through the window (*m*) in percentage of hemoglobin. Artificial light must be employed, best a candle light.

The modifications of Fleischl's old apparatus introduced by Miescher are as follows: 1. The amount of blood employed can be much more accurately measured with the specially constructed pipet (melangeur) than with Fleischl's former capillary tubes. 2. Blood of varying degrees of concentration can be compared with the aid of this melangeur so that a control is kept of the individual examinations and of the evenness of the coloring of the glass wedge. 3. The scale of the new hemometer is supplied with a caliber table for each instrument of absolute hemoglobin values (in milligrams), whereas the scale of the old instrument showed the percentage of hemoglobin in relation to a more or less arbitrarily selected average. In this way many of the errors which with the old apparatus were due to the irregular coloring of the glass slide are avoided. 4. The

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chamber is covered with a glass (D) before attempting to read the percentage, and also with a diaphragm (Bl), so that the fields examined are sharply defined on all sides without any meniscus. 5. The coloring of the glass wedge has been improved upon technically, and is much more even than in Fleischl's original instrument.

The mixing pipet above mentioned (mélangeur) (Mel. Fig. 229) is made like a blood-counter. It consists of a capillary portion and a chamber 200 times the capacity of the former. This contains a glass pearl in the interior. A rubber tube is attached to the melangeur. The drop of blood is obtained in the same way and under the same precautions as for counting the blood-corpuscles. To dilute 200 times, blood is sucked up into the capillary portion to the mark 1, and then the diluting fluid (1 per cent. soda solution), until the chamber is filled. It is then well shaken. There are two other marks on the mixing pipet ($\frac{3}{4}$ and $\frac{1}{2}$), one of which corresponds to a dilution of the blood of 1 : 300, and the other of 1 : 400. If the blood does not suck up readily enough to the desired mark, and there is danger of coagulation, the deficiency or excess in the quantity of blood may be determined by the small cross-lines above and below the main marks. Each of these accessory marks corresponds to the $\frac{1}{100}$ of the entire blood-column (up to 1), so that the excess or deficiency of aspirated blood may easily be deducted or added later.

For accurate comparison of the color shades, the solution of blood must be perfectly clear; otherwise the blood always seems somewhat darker than the color of the glass wedge. This is the reason that we employ a solution of soda, which dissolves the stroma of the blood-corpuscles. This soda solution must be reasonably fresh, as otherwise the blood will not appear perfectly clear, on account of the formation of bicarbonate of soda.

The details of technic in using the Fleischl-Miescher instrument for determining hemoglobin are clearly described in the directions accompanying the apparatus. They are copied from an article by Vaillon.[1] Jaquet[2] claims from numerous experiments that the margin of error in the absolute quantity of hemoglobin determined by this instrument does not exceed 0.15 to 0.22 per cent. by weight of the blood, provided all precautions are taken. This instrument is therefore almost as accurate as the spectrophotometer, and is much simpler.

SAHLI'S NEW HEMOMETER.

In the previously described clinical methods for the estimation of hemoglobin, which are those most commonly employed, the solution of blood is compared with an artificially colored substance—*i. e.*, either with a colored wedge of glass or with a solution of picrocarmin.

To meet the demands of accurate colorimetry, however, the colored fluid to be examined should not be compared with a different substance similar in color, but with a solution of known strength of the *same* coloring matter. This is the only method by which the ability of the human eye to differentiate shades can be completely utilized. In addition to the great difficulty in obtaining a standard color exactly like the color of the blood, whether it be by means of a glass wedge or a picrocarmin solution, the shades of the two colors will not coincide for all dilutions unless the standard color be made with the same substance as that contained in the fluid to be examined. This difficulty is quite apparent in any colorimetric appliance, such as the Fleischl-Miescher hemometer, in which different shades of the standard color must be

[1] *Arch. f. exp. Pathol. u. Pharmakol.*, vol. xxxix., 1897, p. 385.
[2] *Correspondenzbl. f. Schweizer Aerzte*, 1897, pp. 129 and 164.

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compared with different concentrations of the blood to be examined. For analogous reasons, an artificial standard color can never be produced the shade of which will coincide with that of the blood both with natural and with artificial light.

The difficulty in applying this colorimetric principle lies in the fact that solutions of hemoglobin are not permanent. It consequently follows that if, in the interest of accuracy, we avoid employing artificial colors, we must utilize some permanent hemoglobin derivative in making the standard solution. The blood-solution to be examined is then converted into a solution of the same derivative, when a comparison may be accurately made. The application of this principle is limited practically by the necessity of employing only those derivatives of hemoglobin which may be produced in solutions of blood by simple chemical reactions.

After numerous attempts the author has succeeded in devising a method by which the hemoglobin of a solution of blood may be transformed into a derivative by quite a simple chemical reaction. With this derivative permanent standard solutions and colorimetric estimations may be made in accordance with accurate colorimetric principles.

The procedure consists simply in diluting the blood with 10 times its volume of $\frac{N}{10}$ hydrochloric acid. After a few seconds the fluid becomes dark brown from the formation of minute particles of hematin hydrochlorate.[1] Although this substance is not held in true solution, it has the appearance of being, and when diluted with water forms a clear brownish-yellow fluid, the pigment percentage of which may be colorimetrically determined by comparison with a permanent standard solution of the same substance.

The color comparison may be made by means of any of the colorimetric appliances, such as the colorimetric double pipet (see below). In the author's hemometer he has employed the same principle of comparison that is found in Gowers' instrument (see p. 618), and which has proved to be so well adapted to the requirements of the practitioner. The standard hydrochloric acid solution, in a concentration corresponding to a 1 per cent. solution of normal blood, is sealed in a graduated glass tube. A similar accurately graduated tube, each division of which corresponds to 20 c.mm., is filled to the mark 10 with $\frac{N}{10}$ hydrochloric acid which has been saturated with chloroform. With a capillary pipet like that employed in Gowers' instrument, 20 c.mm. of blood are introduced into the tube and well shaken. As soon as the mixture approaches a clear dark-brown color, ordinary water is added until the shade of the mixture exactly corresponds to that of the standard solu-

[1] The substance produced is not methemoglobin, since the shade differs from that of methemoglobin formed in hemoglobin solutions by the addition of other acids. Its spectrum also differs from that of methemoglobin. The author has recently succeeded in producing Teichmann's hemin crystals from this fluid by extraction with acetic alcohol and subsequent evaporation.

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tion, when the hemoglobin percentage may be directly read off, as in Gowers' instrument. The author has kept this standard solution for three years without being able to detect the slightest change in color, and consequently is sure that it is permanent. He must, however, call attention to the fact that after long disuse the suspended particles (which are *not* in solution, see above) may be deposited upon the walls of the tube, by reason of which the fluid appears lighter in color. After such a period of disuse the mixture may be easily made homogeneous again by gently shaking and repeatedly inverting the tube. When this is done a cloud of pigment may be seen to diffuse itself gradually throughout the fluid. The pigment never adheres firmly to the glass. Violent agitation must be avoided, since it fills the fluid with air bubbles, which do not disappear for several hours, and make an accurate comparison impossible.

Physicians have repeatedly sent the author standard tubes which appeared to be too light in color, and which consequently gave rise to the suspicion that the fluid became lighter with age. The author does not believe that this is the case, but has been able to show that the error was due to the fact that the glass blower, when filling the tubes, neglected to agitate the stock mixture sufficiently, and, since this contained minute particles in suspension instead of in solution, the individual tubes did not receive the proper quantity of the pigment. In such cases the faulty tubes were replaced free of cost by the manufacturing firm.

The standard solution was purposely made somewhat dark—*i. e.*, corresponding to the highest hemoglobin percentage of normal blood as found in the author's strongest assistants. The author emphasizes this point, since an absolute standard does not exist, and variations amounting to 20 per cent. (by his hemometer) occur in healthy individuals. The occurrence of such marked variations was first brought to his attention by the use of this instrument, which is based upon reliable colorimetric principles. He was led to employ the bloods richest in hemoglobin for the purpose of making a standard color by observing that a comparison can be more accurately made with the darker than with the lighter shades. The standard fluid now furnished in his hemometer corresponds to a blood which, in a dilution of 1 : 200 in the Fleischl-Miescher instrument, furnishes a reading of 109, or an absolute quantity of hemoglobin of 17.2 per cent.

The author has further improved this instrument, as compared with Gowers' (see Fig. 230), by placing the two tubes in a perforated black stand of hard rubber, which serves as a colorimetric shield. When the tubes are compared by transmitted light, the rays passing through the sides of the glass blend with the side light, and the colored surfaces are seen upon a black background. The stand is also provided with a ground-glass plate which diffuses the light before it reaches the tubes, and obviates all disturbing reflections. By means of these appliances the optical impression obtained is that the fluid is contained in a small compartment with plane parallel sides. Such com-

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partments are really necessary for accurate colorimetric estimations, in order to compare completely homogeneous colored surfaces. They are very dear, however, and cannot be obtained in the small size necessary for the minute quantity of blood which is procured for examination. The appliance above described renders the employment of plane parallel glass vessels entirely superfluous, as may be demonstrated by the effect upon the eye. In addition to these improvements, the scale of the graduated tube may be turned completely behind the margin of the stand, so that it is not seen until the moment when the percentage is to be read. This has a twofold advantage in that the homogeneity of the colored surface is not disturbed by the scale, and that the investigator, in adding the diluting fluid, is not influenced by a preconceived opinion in reference to the expected percentage. The method may be made still more objective by covering all but the lowermost portion of the tube with black paper, so that the height of the fluid may not be seen at all.

FIG. 230.—Sahli's new clinical hemometer.

The author has reached the conclusion that by means of these various contrivances, but chiefly on account of the absolute chemical congruity of the compared fluids, the simple hemometer devised by him, and made by the optician Büchi in Bern, gives as accurate results as the most exact methods of colorimetric examination of the blood, not excepting the more complicated ones. Furthermore, it is clear that the instrument must give the same result with both artificial and natural light, which is of the greatest advantage to the practitioner, who must work under the most varied conditions. The most accurate result will possibly be obtained in a dark room with artificial light, since under these conditions the eye is the most sensitive to light and color; but even with daylight the results are quite exact. The procedure is almost as simple as Gowers'.

The author would finally call attention to the fact which he has directly proved experimentally, and upon which the entire procedure is based, that the brownish-yellow color produced by the addition of hydrochloric acid and subsequent dilution with water is directly proportional to the hemoglobin percentage of the blood examined. In other words, the accuracy of the colorimetric dilution method is not impaired by a dissociation of the hematin hydrochlorid combination which is formed.

HEMATOSPECTROPHOTOMETER.

The most accurate method of determining the amount of hemoglobin is by means of the spectrophotometer, first used by Vierordt and perfected by Hüfner.

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This instrument, however, is much too complicated and too expensive for practical purposes, so that the author will refer for a description to Hüfner's essay in the third volume of the *Zeitschrift für Physiologische Chemie*, p. 560.

THE HOPPE-SEYLER COLORIMETRIC DOUBLE PIPET.

This very excellent apparatus is also too complicated for clinical purposes.[1]

THE COUNTING OF BLOOD-CORPUSCLES.

THE COUNTING OF RED BLOOD-CORPUSCLES (Erythrocytes.)

It is almost impossible to form any idea of the number of red blood-corpuscles contained in the blood by simply inspecting a microscopic preparation, unless the number is so greatly diminished that the preparation shows few or no rouleaux. Actual counting is the only proper procedure. Numerous methods of counting have been advocated. In all of them a known quantity of blood is diluted to a definite proportion with a solution which preserves the red blood-corpuscles, and then those contained in a definitely measured space are counted. The number of red blood-corpuscles in 1 c.c. of undiluted blood can then easily be calculated by simple multiplication.

The simplest and most practical blood-counting apparatus is that constructed by Thoma and Zeiss (Fig. 231). It is based upon the older principles of Malassez, Hayem, and Gowers. It consists of two parts, a counting-chamber upon a slide, and a mélangeur pipet. The latter consists of a capillary pipet with a bulb containing a freely movable glass pearl. As is shown in the figure, the mélangeur is so constructed that the bulb contains 100 times the cubic contents of the capillary tube. A rubber tube is attached to the upper end for convenience in sucking up the blood.

The counting-chamber itself is represented in Fig. 231, *B* from above and *C* in cross-section. Two plane glass plates are cemented to a slide. The smaller (*f*) is circular; the larger (*e*, *e*, *e*, *e*) is square, somewhat thicker than *f*, and with a circular opening in the center, in which (*f*) is centered, leaving between the two a circular moat (*g*). The floor of the moat is formed by the slide itself. The upper surface of *f* is exactly 0.1 mm. below that of *e*. In the center of *f*, which forms the floor of the counting-chamber, 1 sq. mm. of the glass is ruled with a microscopic scale into 400 (20 × 20) squares (Fig. 231, *D*). Each square has a surface of $\frac{1}{400}$ sq. mm., each side being $\frac{1}{20}$ mm. This chamber is covered with a carefully ground cover-glass, made thick enough not to bend. The chamber is therefore divided into little square prisms, each one of which has a cubic content of $\frac{1}{4000}$ c.mm. This fraction forms the basis of subsequent calculations. The chamber is very carefully ground, and to avoid error the cover-slip should rest very accurately upon the slide. It is carefully pressed down upon the square glass *e*, *e*, *e*, *e* until the "Newton's rings" which are produced remain permanently—*i. e.*, after the pressure has been removed—merely from the adhesion between the cover-slip and the margin of the chamber. The moat (*g*, *g*) around the glass (*f*) prevents the blood-mixture upon *f* from running in between the cover-slip and the square glass (*e*, *e*, *e*, *e*), and so altering the depth of the chamber.

The method of procedure is as follows: In extracting the blood we must avoid any pressure in the vicinity of the puncture, otherwise congestion or lymph exudation may make the results inaccurate. The blood is sucked up into the

[1] *Zeits. f. Physiol. Chem.*, vol. xvi., 1892, p. 505, and *Lehrb. der physiol.-chem. Analyse*, 1893, p. 414.

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capillary tube to the mark 1; the bulb is then filled up to the mark 101 with a 3 per cent. solution of sodium chlorid or a 5 per cent. solution of Glauber's salts, or better still, Hayem's fluid.[1] While sucking up the diluting fluid, the mélangeur should be held vertically and slightly rotated to whirl the glass pearl a little, and so free it from any adherent air bubbles which would make the degree of dilution inaccurate. A 1 : 100 dilution of the blood is thus prepared for counting. After thorough shaking, 2 or 3 drops of the fluid are first blown out, the point of

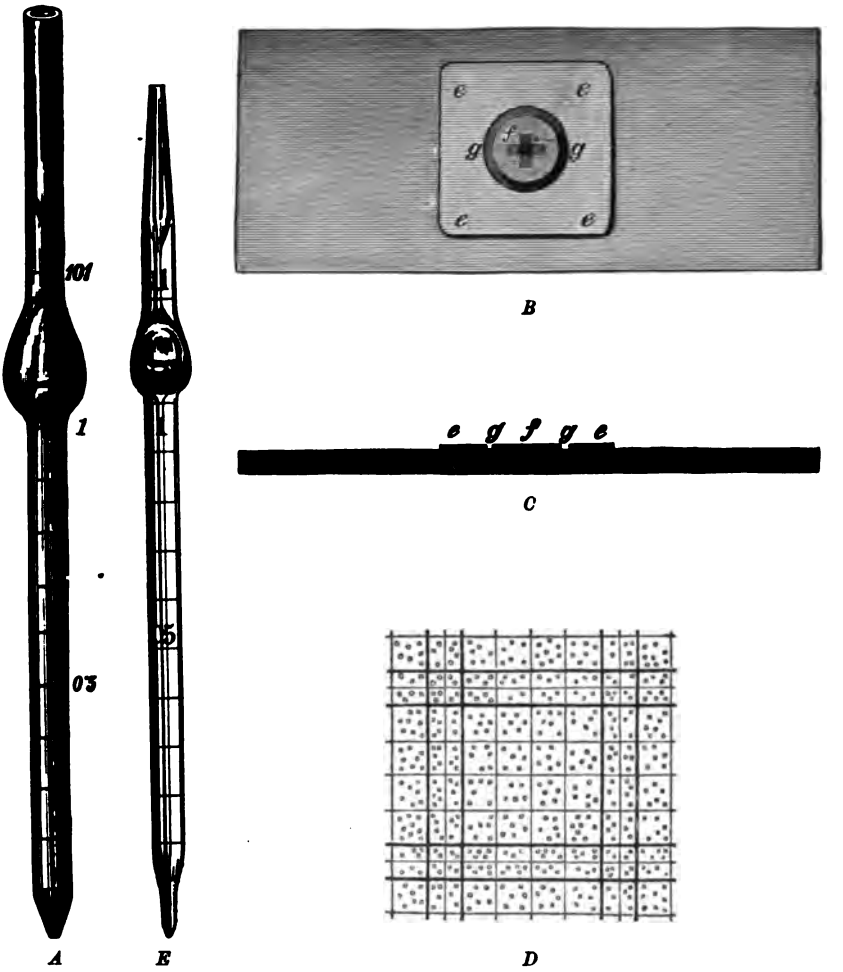

FIG. 231.—Thoma-Zeiss blood-counting apparatus: *A*, Mélangeur; *B*, counting-chamber, seen from above; *C*, profile of counting-chamber; *D*, microscopic picture of a portion of ruled field with blood-cells; *E*, white counter.

the pipet is wiped off, and a small drop of the solution is carefully placed upon the center of the counting-chamber—*i. e.*, over the ruled scale. The cover-glass is now adjusted, and pressed down at the margin until Newton's rings appear.

[1] Hayem's fluid: Sublimate, 0.5; sodium sulphate, 5; sodium chlorid, 2; aquæ destillatæ, 200. This also preserves the red color of the cells, and facilitates a distinction between the red and the white blood-corpuscles.

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If these do not remain after removal of the pressure, the preparation must be made over again. The chamber and cover-glass must then be still more carefully cleansed, for the trouble is usually due to dust getting between the margin of the chamber and the cover-glass. The slide is allowed to remain horizontal for about a minute, until the blood-corpuscles have settled to the bottom of the chamber, and then the corpuscles are counted. For greater accuracy two counting-chambers may be prepared, and compared to be sure that the blood-corpuscles are about evenly distributed in each. If this is not the case, fresh preparations should be made after repeatedly shaking the mélangeur. A medium power is the best for counting; for instance, a Leitz No. 5 or a Zeiss C or D objective.

In the modern apparatus each fourth and fifth division line of the chamber is more heavily ruled, and an extra line is ruled halfway between them (Fig. 231, *D*). This device is helpful in pointing out the squares more plainly, and series of 16 (4 × 4) or 36 (6 × 6) squares can be counted more rapidly.

To determine the number of cells contained in 1 c.mm. of blood, we divide the number of cells counted by the number of squares; the resulting quotient is the number of cells in one square; this we multiply by the dilution, and divide by the size of the tiny prism. Suppose, for example, that in counting 100 squares we find 980 cells.

$$\text{Number of cells, } \frac{980}{\text{Number of squares, } 100} \times 100 \text{ (the dilution)} \times 4000 = 3,820,000$$

The size of each tiny square prism is marked upon the counting-slide, and so need not be memorized.

Unless there is a decided diminution of red cells (anemia), it is easier to count a preparation diluted 200 times. For such a dilution we suck up blood to the first mark 0.5, and then suck the diluting fluid to the 101 mark (just above the bulb). Some examiners find that a sliding stage makes counting easier and more accurate. The more cells we count, the more accurate our result.

THE COUNTING OF THE WHITE BLOOD CORPUSCLES (Leukocytes).

It is very difficult to judge the number of leukocytes in a specimen of undiluted blood, because they are usually distributed very irregularly, and often seem to be hidden or jammed in between the rouleaux of the red blood-corpuscles. For this reason we dilute the blood, and count them as explained in the following:

Two conditions must be fulfilled: first, the leukocytes must be made easily recognizable with the rather low power used in counting; and second, considerably larger amounts of blood must be used than in counting red blood-corpuscles, because the number of leukocytes is so small (unless, of course, they are very considerably increased, leukemia). The blood is diluted with a $\frac{1}{3}$ per cent. aqueous solution of acetic acid. This solution dissolves the red blood-corpuscles, leaving the remaining cells leukocytes. A special mélangeur (Fig. 231, *E*) is used; its bulb contains only 10 times as much fluid as the capillary tube. After preparing the mixture, the technic is very similar to that for counting red cells. It is advantageous, however, to employ a larger counting-chamber, such as those devised by Elzholz, Türk, and B. Breuer. On account of its clearness, Breuer's chamber seems best. Fig. 232 shows how it is divided. The depth of this chamber is 0.1 mm., as in that of Thoma, but the sides are 3 mm. in length, so that the square has a surface of 9 sq. mm. The units for calculation are the elongated rectangles seen in the illustration. The triple line is merely for the purpose of orientation. Upon request, the Zeiss firm will furnish this counting-chamber with the central square millimeter subdivided into smaller squares of $\frac{1}{400}$ sq. mm., as in the counting-chamber of Thoma, so that this portion of the field may be utilized for counting red blood-corpuscles, making a second counting-chamber unnecessary.

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The division of Türk's counting-chamber is also very practical.[1] With this large chamber a great number of leukocytes may be rapidly counted, and this is something which is necessary in the interest of accuracy (see below). After the drop of diluted blood has been placed upon the floor of the counting-chamber, it should be examined with a low power, so that the examiner may convince himself that the leukocytes have been homogeneously distributed. Just as in counting erythrocytes, it is best to employ two counting-chambers simultaneously, and to be sure that the leukocytes are present in about the same proportions in both chambers.

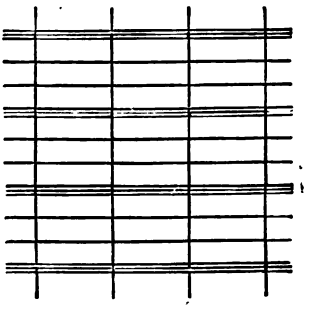

FIG. 232.—The divisions of Breuer's counting-chamber for leukocytes.

Instead of the $\frac{1}{3}$ per cent. acetic acid solution, we may employ the same 3 per cent. sodium chlorid solution which was mentioned for counting the red cells, adding gentian violet in the proportion of 1 : 10,000. This will stain the leukocytes, but does not dissolve the red cells, so that it is not as convenient nor as accurate as the acetic acid solution.

Türk recommends counting 300 to 1000 leukocytes. This is very easy if Elzholz's chamber is used. Reinert found that the probable error was 3.6 per cent. one way or the other even when 4000 cells were counted.

In pathologic cases the count should, if possible, be taken just before a meal—*i. e.*, at a time to avoid the leukocytosis of digestion. The leukocytosis of digestion is almost always absent if very small and frequent meals of fatty and starchy food are taken (Rieder).

NUMBER OF RED AND WHITE BLOOD-CORPUSCLES UNDER PHYSIOLOGIC CONDITIONS.

The number of red blood-cells (Vierordt) in 1 c.mm. of blood is 5,000,000 in man and 4,500,000 in women. In Switzerland the counts show that healthy women have 5,100,000 or over, and men about 6,000,000. These correspond very well with the following figures which Sörensen published to show the variation in the number of blood-corpuscles at different ages:

Table.

	Males.			Females.		
	Age.	Number of red cells in 1 c.mm. of blood.	Number of individuals examined.	Age.	Number of red cells in 1 c.mm. of blood.	Number of individuals examined.
Newborn	5-8 days	5,769,500 (5,284,500–6,105,000)	3	1-14 days	5,560,800 (5,262,500–5,960,000)	6
Children	5 years	4,950,000 (4,750,000–5,145,000)	2	2-10 yrs.	5,120,000 (4,980,000–5,260,000)	2
Adults (Students)	19½–22 "	5,606,000 (5,422,000–5,784,000)	7	15–28 "	4,820,000 (4,417,000–5,350,000)	14
Adults (Young physicians)	25–30 "	5,340,000 (4,900,000–5,800,000)	6	41–61 " (Nurses)	5,010,000 (4,800,000–5,470,000)	7
Adults	50–52 "	5,137,000 (4,910,000–5,359,000)	2			
Adults	82 "	4,174,700	1			

[1] Türk, *Vorlesungen über klinische Hämatologie*, W. Braumüller, Wien and Leipzig, 1904, p. 95.

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A number of physiologic conditions have some influence upon the number of red blood-cells. As to the influence of food, the authorities differ. According to the observations of Vierordt and Limbeck, there is usually a slight diminution in the number of red cells after eating or drinking, probably to be explained by the dilution of the blood due to absorption of fluid. On the other hand, fasting produces a relative increase. Obese people average a somewhat lower red blood-cell count than thin people (Leichtenstern). Menstruation and pregnancy seem to have no especial influence upon the number of blood-cells. Cathartics and diuretics increase the number of red cells if their action is very marked, possibly on account of the removal of water. A cold bath temporarily increases the number of red cells. The cause of this phenomenon is not known. The influence of climates and high altitudes upon the number of red blood-corpuscles is interesting. According to Viault, Miescher, Egger, Jaquet, and others, the number of red blood-cells is considerably increased in altitudes of 1000 to 2000 meters above the level of the sea. This increase occurs within a very short space of time. In Arosa, Egger found that inside of two weeks the number of red blood-corpuscles increased in newcomers from 5,000,000 to 6,000,000. Egger has proved that this increase is due neither to diminution of the plasma, owing to the dry air of the high altitude, nor to a mere difference in the distribution. (See Miescher's Histochemic and Histologic Works, Leipzig, 1897.) Gottstein claimed that this state of affairs was an illusion, due to the fact that the depth of the counting-chamber was greater with a low than with a high air pressure; but physics contradicts such an hypothesis. Besides, the hemoglobin percentage increases correspondingly.

The ratio of the white to the red blood-corpuscles varies between 1 : 300 and 1 : 700. This wide range is probably due to the difference of the ratio in individual cases, to the leukocytosis of digestion, and to the varying number of red blood-corpuscles. Rieder examined fasting people, and found that the ratio of the white to the red blood-cells in healthy adults with a normal red blood-count is 1 : 651, and in children (nine to fifteen years) 1 : 518. Considering the varying number of red cells, these comparative figures are of less value than the absolute number per cubic millimeter. Rieder found in fasting adults an average number of 7680, and in children 9660. (Compare p. 646 with reference to the increased number of leukocytes in the newborn.)

NUMBER OF RED BLOOD-CELLS AND PERCENTAGE OF HEMOGLOBIN IN BLOOD UNDER PATHOLOGIC CONDITIONS.

(Compare p. 659 *et seq.*)

All conditions included under the title "anemia" produce a more or less marked diminution in the number of red blood-cells and in the hemoglobin. Any acute or chronic illness may diminish both the number of blood-cells and the amount of hemoglobin, and so produce an anemia. This is, however, by no means always the case. In acute febrile infectious diseases the red blood-corpuscles and the hemoglobin are as often increased as diminished, the increase being due to the diminution in the amount of blood-plasma, the decrease, to the retention of the water and the destruction of the red blood-cells. No accurate diagnostic or prognostic conclusions can as yet be drawn from these conditions.

Chronic cachexia generally tends to diminish both the amount of

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hemoglobin and the number of red cells. In the cachexia of cancer the anemia may be very marked and diagnostically important. Whenever a marked anemia accompanies cancer, the growth has almost always caused a loss of blood. This loss may be very gradual and not noticed at the time (carcinoma of the stomach and intestines). The blood of tuberculous patients shows at times a diminished quantity of hemoglobin and a smaller number of red blood-corpuscles. This is, however, by no means the rule. Many tuberculous patients with marked pallor and cachexia frequently show a normal blood. Neurasthenic patients rarely show a diminution in the coloring matter of the blood. Determining the amount of hemoglobin in these cases is of great value with reference to the treatment, as we have repeatedly emphasized, for a pale appearance is often deceptive. (See section on the Color of the Skin, and p. 662, on Secondary Anemia.)

In venous congestion both the hemoglobin and the number of blood-cells are increased. This is a purely mechanical result, due to the slowing of the blood-stream, which leaves the blood-cells in the capillaries. This condition does not exclude an increase in the amount of water contained in the blood-plasma, nor hydremic plethora.[1]

The Hemoglobin Quotient or Hemoglobin Value of the Red Blood-corpuscles (the So-called Blood-corpuscle Quotient or Value).—If the quantity of hemoglobin and the number of blood-cells in any given disease are expressed in percentages of the normal, and the percentage of hemoglobin is divided by the percentage of the number of red cells, a fraction is obtained which, as will readily be seen, indicates how much hemoglobin any one blood-cell contains, as compared with the normal amount. This fraction is called the color index, the blood-corpuscle quotient or value. In order to avoid confusion with the volume quotient described on p. 639, the author would suggest using "hemoglobin quotient" or "hemoglobin value." Normally this equals 1, but in certain anemias it sometimes exceeds 1 (pernicious anemia), or is less than 1 (chlorosis)—*e. g.*,

Hgb. = 45 per cent.
Red cells = 2,000,000 (40 per cent.).
C. I. = $\frac{45}{40}$ = 1.1 +.
or, Hgb. = 40 per cent.
Red cells = 4,000,000 (80 per cent.).
C. I. = $\frac{40}{80}$ = 0.5.

VOLUME OF BLOOD-CORPUSCLES CONTAINED IN A GIVEN QUANTITY OF BLOOD; HEMATOCRIT.

An attempt has recently been made to supplant the counting of blood-corpuscles by estimating the volume which they occupy as compared with that of the plasma. The method recommended by Blix-Hedin, and modified by Gärtner, consists in first adding a definite amount of a solution to a certain amount of blood in order to preserve the corpuscles and prevent coagulation (Müller's fluid), and then centrifuging in a graduated capillary tube closed at one end. This is known as a hematocrit. The specimen is centrifuged about five minutes, until the blood-corpuscles are packed into the end of the capillary tube, so that their volume can no longer be diminished by further centrifuging. The red blood-corpuscles form a red layer at the bottom of the capillary tube, and above them comes a white layer of leukocytes. The method is easy, but cannot take the place of

[1] See K. Baranoff, *Beiträge zur Theorie der Flüssigkeitsentziehung und der Behandlung der Circulationsstörungen*, J. A. D., Bern, 1895.

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counting the blood-corpuscles, as was first thought, because there are several sources of error. For a detailed description of the hematocrit, we refer to the original communication of Blix-Hedin[1] and Gärtner.[2]

According to Capps,[3] more accurate results are obtained with the hematocrit if pure blood is used without any diluting fluid. In this case, however, the work must be done very rapidly to avoid coagulation, and the centrifuge must be a powerful one, propelled preferably by electricity. The investigations in reference to the volume quotient of the erythrocytes, described upon p. 639, were carried out by this method, and demonstrated that, in view of the variations in size of the erythrocytes, the hematocrit can never replace the actual count of the latter, but that it may give valuable information when they are of the average size.

POWER OF RESISTANCE OF THE ERYTHROCYTES TO HYPOSMOTIC INJURY.

It is well known that when a small amount of blood is placed in an excessive quantity of a hypotonic saline solution, the blood-pigment passes out of the erythrocytes, so that a so-called laked blood results. In such experiments the stromata of the erythrocytes are not dissolved. In saline solutions which are isotonic or hypertonic to the blood-plasma, the blood-pigment does not pass out, at least not within a limited period of time. For this reason it was formerly believed that the osmotic pressure of the blood-plasma could be determined by finding the concentration of the saline solution which would preserve the erythrocytes intact. It has nevertheless been shown that the escape of the hemoglobin from the erythrocytes under these conditions is by no means a pure osmotic phenomenon, but that it is also dependent upon the specific sensibility of the cortical layer of the erythrocyte, which holds the hemoglobin within the corpuscle. This has been absolutely proved by the observation that all of the erythrocytes of an individual are not equally sensitive to the same hypotonic solution. In a slightly hypotonic saline solution, the osmotic concentration of which closely approximates that of the blood, a number of erythrocytes are destroyed by the escape of their hemoglobin, while others do not lose their pigment until the saline solution is much more markedly diluted. This could not be the case were the phenomenon purely osmotic in character. For this reason we are no longer justified in assuming that the osmotic concentration of the blood-plasma may be indirectly obtained by determining the resistance of the erythrocytes to saline solutions of varying concentration. This false idea is also responsible for the incorrect supposition that a saline solution of 0.6 or even 0.7 per cent. is physiologic—*i. e.*, isotonic with the blood-plasma—when as a matter of fact the direct determination of the osmotic pressure of the blood by means of cryoscopy (see p. 667) has shown that a 0.9 per cent. saline solution is isotonic with human blood.

The above facts should be borne in mind in utilizing Limbeck's[4] modification of Hamburger's[5] method for determining the resistance of the erythrocytes to hypotonic saline solutions. Limbeck's modification is as follows: One cubic centimeter of a sodium chlorid solution of varying concentration is placed in each of a series of 16 small glasses. The weakest is 0.4 per cent., and the concentration increases from one glass to the next by 0.03 per cent., so that the series varies from 0.4 to 0.85 per cent. A small amount of blood is taken from a punctured wound of the finger by means of a glass rod, and placed in each glass of the series. The blood is well mixed with the saline solution and allowed to stand for six hours. After the lapse of this period of time the mixtures are inspected, to determine which solution has preserved the blood without extracting its pigment. The lower the concentration of the saline solution fulfilling this requirement, the greater will be the resistance of the erythrocytes.

[1] *Skandin. Arch. f. Physiol.*, 1890, p. 134. [2] *Allg. Wien. med. Zeit.*, 1892.
[3] *Jour. Med. Research*, vol. x., No. 3, 1903.
[4] *Grundriss der klin. Pathologie des Blutes*, Jena, Fischer, 1896.
[5] *Arch. der Physiol.*, von Du Bois Reymond, 1886, p. 476; 1887, p. 31, *Zeits. f. Biol.*, 26, neue Folge, viii., p. 414.

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This method is open to a number of objections, however, in reference to which the reader is referred to the monograph of G. Lang.[1] The chief difficulty depends upon the different degrees of resistance exhibited by the individual corpuscles of the same patient. Lang's monograph gives a number of methods proposed by Janowski for the purpose of avoiding these difficulties.

In carcinoma and icterus it has been observed that the erythrocytes exhibit a most marked degree of resistance to hyposmotic saline solutions.

The determination of the power of resistance of the erythrocytes to hyposmotic injury is not sufficient to settle all of the questions in reference to their resisting power, since Chvostek[2] found that in paroxysmal hemoglobinuria the power of resistance of the erythrocytes to saline solutions was normal, while they showed but slight resistance to mechanical injury (shaking) and to stasis within the body.

OTHER MORPHOLOGIC RELATIONS OF THE BLOOD.

TECHNIC OF THE MICROSCOPIC EXAMINATION OF THE BLOOD.

For this purpose either fresh or stained dried preparations may be employed.

MICROSCOPICAL EXAMINATION OF FRESH BLOOD.

The most important alterations in the blood which are peculiar to diseases of the blood proper may be recognized in fresh specimens. Certain precautions should be taken in preparing the latter. Too much pressure applied near the puncture to increase the flow may deform the red blood-corpuscles. A scrupulously clean cover-slip should be very gently touched to the drop of blood, so that only a very tiny amount of blood adheres. Too large a drop makes too thick a preparation and too pronounced a rouleaux formation, so that the finer details cannot be detected. The cover-slip is immediately dropped very carefully upon a clean slide. Pressure, pulling, or sliding the cover-slip should be avoided. Unless these precautions are observed, a most beautiful micro- or poikilocytosis (compare below) may be artificially produced. The blood should be distributed under the cover-slip in a uniformly thin layer, but this effect must be produced entirely by capillary action. The preparation should be examined immediately, because the blood-corpuscles soon begin to shrink from loss of water, and assume all sorts of bizarre shapes.

PREPARATION AND STAINING OF DRIED SPECIMENS.

The staining of the histologic elements of the blood is almost exclusively confined to dry preparations. These stained specimens give information in regard to the granulation of the leukocyte protoplasm discovered by Ehrlich, as well as to the coarser morphologic conditions and staining propensities of the nuclei and of the protoplasm of all the blood-cells. Ehrlich, by a careful study of the staining peculiarities of the granulations with various anilin colors, has found that with given mixtures of anilin dyes they possess elective affinities and stain differently. He classifies the available anilin dyes, which are chemical salts, into an

[1] *Zeits. f. klin. Med.*, vol. xlvii., parts 1 and 2. 1902.
[2] *Ueber das Wesen der paroxysmalen Hämoglobinurie*, Wien, F. Deuticke, 1894.

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acid group, in which the acid radicle, and a *basic group*, in which the basic radicle, determines the staining property of the dye, and further into a *neutral group*, in which both radicles possess staining properties. *Eosin* and *acid fuchsin* are types of acid stains, *methylene-blue and -green* of basic stains, and *rosanilin* and *picric acid*, of neutral stains.

He found that certain granulations in the white blood-corpuscles stained only with acid, others only with basic stains; a third group could be stained with both dyes, and a fourth by neutral pigments only. On the strength of this he distinguishes (α) oxyphilic, (β) amphophilic, (γ and δ) basophilic, (ε) neutrophilic granulations. The difference between γ and δ is chiefly in the size of the granules. The (γ) granules are the so-called *mast-cell granules;* they are basophilic and larger than all the others. According to Ehrlich, each leukocyte contains only one kind of granule. The β and δ varieties are not found in human blood.

The following rules should be followed in preparing dry specimens: Very thin cover-slips (less than 0.1 mm.) should be cleansed with the utmost care. Fat can be removed with a mixture of ether and alcohol and a soft linen cloth without shreds.[1] In subsequent manipulations the cover-slips should be touched only with dry finger-tips or with forceps, and best at the corner. A very minute drop of freshly removed blood from the tip of a finger or the lobe of the ear is touched to a cover-slip, and a second slide laid on top of this without pressure. Within a few minutes, by capillary action, the blood will spread out between the cover-slips in a uniform layer; then, without exerting any pressure, the slips can be slid rapidly apart and dried in the air.

Fixing or hardening of the dry preparation can be accomplished by drying in the air, or, according to Erhlich's original method, by heating in an incubator at 110° to 120° C. for several minutes up to an hour, according to the stain used. A simpler method later recommended by Ehrlich is to place a copper plate horizontally upon a stand, and to heat one end with a flame. After a short time each individual portion of the plate will remain at a uniform temperature, the part nearest the flame hottest, the more distant portions less so. The spot upon the plate is determined where a drop of oil of toluol no longer boils, but presents *Leidenfrost's phenomenon*.[2] The cover-glass, after being dried in the air, is placed at this spot. The temperature is about 111° C. (with barometer 760 mm.). It should remain here from one-half to one minute if ordinary stains (hematoxylin, eosin, triacid) are to be used. With many other stains the cover-slip must be left there longer or a higher temperature employed.

Rubinstein[3] recommends more intense heat even for the triple stain. For this purpose he places the cover-slip face down at a spot on the copper plate where a drop of water still shows Leidenfrost's phenomenon,

[1 Soap and hot water and careful rubbing with a soft linen cloth will cleanse them thoroughly. Occasionally it may be necessary to wash new cover-slips in strong acid to remove the glaze.—ED.]

[2] The drop runs around without wetting the copper.

[3] *Zeits. f. wissensch. Mikroskopie*, vol. xiv., 1897, p. 456.

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and in such a way that a corner of the glass reaches over toward the cooler end of the plate.

Preparations may also be fixed by immersing them for five minutes in absolute alcohol, or by boiling them in it for one minute, or by immersing them in a 1 per cent. solution of formalin for about one minute. After fixation the specimen may be stained immediately or later.

A simple method which usually suffices for recognizing the coarser relations consists in immersing the specimen in a saturated solution of eosin in 5 per cent. carbolglycerin for several hours (Rieder). The color is then washed off with water, and it is counterstained for a minute with 50 per cent. Delafield's hematoxylin.[1] After repeated washings, the preparation is dried in the air and mounted in Canada balsam. The red blood-corpuscles and eosinophilic granules are stained red, nuclei and cell membranes dark blue, and the protoplasm of the white cells violet or reddish (see Plate 8, Figs. 4 and 5).

To recognize the finer details of the nuclear structure, and especially of the mitoses, Rieder recommends that blood-films be first fixed, and then immersed in a saturated aqueous solution of picric acid. They should then be washed for one or two days in running water, and stained several hours with a very dilute Delafield hematoxylin; then washed in water again, and finally in water containing hydrochloric acid, dried in the air, and mounted in Canada balsam.

Ehrlich's triple stain is usually employed when examining for Ehrlich's granules. This is prepared in the following way:

Saturated aqueous solution of orange-G., 120 to 125 c.c.; saturated aqueous solution of acid fuchsin, 80 to 165 c.c.; saturated aqueous solution of methylene-green, 125 c.c.; water, 300 c.c.; absolute alcohol, 200 c.c.; glycerin, 100 c.c.

Fixed dry preparations are immersed in this solution for two minutes, washed in water, and then dried and mounted. The neutrophilic granules are most distinctly stained. The hemoglobin is orange-colored, the nuclei green, the neutrophilic granules violet, the eosinophilic granules copper-colored, and the basophilic granules indistinctly stained.

Excellent results are also obtained by the use of **Jenner's stain,**[2] which contains eosin and methylene-blue, probably as a chemical combination, in solution in methyl alcohol. This stain is prepared with difficulty, but may be obtained from Dr. Grübler, Bayrische Strasse, Leipzig, or of still better quality from Baird and Tatlock,[3] Cross St., Hatton Gardens 14, London. The great advantage of this method is that the specimen does not require a previous fixation, since the methyl alcohol in the solution fixes the blood while it is being stained. The specimens are almost as good as those obtained by the use of the tri-acid stain, and successful results are much more readily attained. The illustrations upon Plate 6, are from specimens stained by Jenner's method. According to May and Grünwald, it is important to wash specimens stained in this manner with distilled and not with hydrant water.

Chenzinsky's eosin-methylene-blue solution is to be recommended for certain purposes. Forty cubic centimeters of concentrated aqueous methylene-blue

[1] Two solutions: (*a*) 1 gm. of a crystalline hematoxylin dissolved in 6 c.c. of absolute alcohol; (*b*) 15 gm. of ammoniate of alum dissolved in 100 c.c. of distilled water, and filtered after cooling. These solutions are mixed in an open dish, and exposed to light three days. The mixture is then filtered, and mixed with 25 c.c. of pure glycerin and 25 c.c. of methyl alcohol. After three days this mixture is filtered and kept in stock.

[2] *Lancet*, 1899, p. 370.

[3] [A very good preparation can be obtained from Dougherty, West Fifty-ninth Street, or from Eimer & Amend, Third Avenue and Eighteenth Street, New York City.—ED.]

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solution, 20 c.c. of ½ per. cent. eosin solution in 70 per cent. alcohol. and 40 c.c. of distilled water are mixed. This mixture should always be filtered before using. Six to twenty-four hours in an incubator are necessary for staining. The nuclei, the mast-cell granules, and the basophilic granules of red blood-corpuscles appear intensely blue, malaria plasmodia sky blue, the red blood-corpuscles and eosinophilic granules red.

Willebrandt's Method.[1]—Willebrandt claims that all the granules of human blood may be very well seen after staining with a solution of methylene-blue and eosin. A solution is prepared in the following way: One-half per cent. of eosin in 70 per cent. alcohol is mixed with an equal quantity of concentrated aqueous solution of methylene-blue. Willebrandt describes the peculiarities of this solution as follows:

The fluid usually stains the preparation diffusely blue, on account of the preponderance of methylene-blue. If, however, dilute acetic acid (1 per cent.) is added drop by drop—usually 10 or 15 drops to 50 c.c. of solution—the eosin will gradually acquire an increased staining power until it becomes quite efficient. After experimenting a little, it is always possible to prepare a solution that stains well and gives a characteristic picture. The solution should always be filtered before using. The blood-preparation should always be carefully fixed, otherwise the stain will not be successful. The staining requires five to ten minutes with repeated heating until steam is given off. The specimen is then washed in water without any other decoloration. The stain from this method is very instructive. The red blood-corpuscles appear red, the nuclei dark blue and sharply outlined, the neutrophilic granules of the mast cells intensely violet. This method has the advantage of staining all the granules and the nuclei at the same time.

In staining for special granules, the eosin hematoxylin stains eosinophilic granules very well, while a saturated aqueous solution of methylene-blue is best adapted for basic granules (stain for several minutes), and the neutrophilic granules are best stained with Ehrlich's triple stain.

Ehrlich's directions are the best for demonstrating *microscopic iodin reaction of the blood.* The dried blood-film (which must, however, not be heated) is immersed in a solution of iodin and gum arabic (iodin 1, potassic iodin 3, water 50, and enough gum arabic to render the fluid thick). Ehrlich[2] has since announced that it is an advantage to substitute iodin itself for the solution of iodin. This is done by placing the preparations dried in the air under a closed glass containing crystals of iodin, for several minutes, until the color is dark brown. The preparation should then be examined in a saturated syrupy solution of levulose; and to preserve it, should be surrounded with varnish. The color of the leukocytes varies in individual cases. The red blood-cells are stained diffusely brown, although this, at present at least, has no special interest. Zollikofer has established the following facts with regard to the leukocytes.[3] Under physiologic conditions, brown-stained protoplasmic granules are found most frequently in lymphocytes, oftentimes in mast cells, and exceptionally in the large mononuclear cells; the polynuclear neutrophilic cells are faintly stained brown. If the last-mentioned cells show distinct deposits of brown-stained granules and flakes, it is considered pathologic. This condition is the one which authors have paid most attention to up to the present time. It is ordinarily spoken of as the *iodin reaction* of the leukocytes or the *intracellular iodin reaction of the blood. Zollikofer's brown granules* are very rarely found in eosinophiles, and then under conditions which are not understood or defined. They are never found in the neutrophilic mononuclear cells (myelocytes). Ehrlich[4] has also described a so-called extracellular reaction, in which stained brown granular masses are found outside of the leukocytes, Gabritschewski[5] and Zollikofer have shown, however, that these formations are blood-plates, which under conditions not

[1] *Deutsch. med. Woch.*, 1901, No. 4, p. 57.
[2] Ehrlich and Lazarus, "Die Anämie," *Nothnagel's Sp. Path. and Therap.*, 1898.
[3] *Zur Iodreaction der Leukocyten*, J. A. D., Bern, 1899.
[4] *Zeits. f. klin. Med.*, 1883, vol. vi. [5] *Arch. f. exp. Path. u. Pharm.*, 1891, vol. xxviii.

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definitely known give the iodin reaction, at times in normal blood, at times under pathologic conditions, especially in diabetes mellitus. This reaction does not necessarily accompany the intracellular reaction.

This brown-staining substance is generally supposed (and by Ehrlich, too) to be glycogen. But Czerny,[1] Kamminer,[2] and especially Zollikofer, discredit this theory. Very likely it is related to amyloid. Under certain conditions it is stained violet and not brown by iodin, which favors this latter view.

The pathologic iodin reaction, at present at least, applies only to the appearance of brown-stained granules in the polynuclear neutrophiles. Very little can be said as yet in regard to the clinical importance of this "iodin reaction." It is almost always present with *leukocytosis*, especially in purulent conditions, but not, for instance, in erysipelas.

According to the investigations of Zollikofer in the Bern Clinic, the presence of this reaction—*e. g.*, in appendicitis—does not always prove the presence of pus, neither does its absence exclude the presence of pus. About the same results were observed in cases which recovered spontaneously and in those which came to operation. No definite conclusions can be drawn from the absence or presence of the iodin reaction in diabetes mellitus, although several observations of Zollikofer's would seem to indicate that a diminution of diabetic acidosis is associated with diminution of the reaction. It was thought that the reaction might possibly be used in the diagnosis of amyloid degeneration, an opinion based upon the chemical nature of the brown-staining substance, and upon the observations of Czerny relative to the presence of the iodin reaction in artificially produced amyloid degeneration; but observations in the author's clinic have disproved this. Zollikofer found that if iodin vapor acted upon moist preparations, the reaction was considerably increased, so that even in normal blood fine brown granules appeared in the neutrophilic leukocytes, instead of the diffused light-brown stain. The absence of these normal granules in the dry specimens treated with iodin is due, according to Zollikofer, to the disintegration of the granules in the interior of the cells during the process of drying.

Examination of the Blood in Reference to the Formation of Rouleaux.

Under normal conditions, in fresh microscopic blood-specimens which are not too thin, we find the red blood-corpuscles, as a result of their peculiar disk-like shape and the viscosity of the blood, standing upon their edges and arranged in cylindric conglomerations which have been compared to a roll of coins. This formation occurs in the circulation only when the blood stagnates, but it normally occurs quite rapidly (in a few seconds) outside of the body. This process is known as the formation of rouleaux. It will easily be understood that the formation of rouleaux must be influenced by the number of the red blood-corpuscles. We consequently find a diminished formation of rouleaux in all conditions which are associated with a diminution in the number of the red blood-corpuscles, and with a properly made specimen the diminished tendency to form rouleaux may be made the basis of a probable diagnosis of a decreased number of red blood-cells. By a properly prepared specimen we mean one having a sufficient thickness to allow the erythrocytes to stand upon their edges. If the preparation be too thin, the erythrocytes are flattened out by the cover-glass and cannot adhere to each other by their flat surfaces. When a specimen of proper thickness is immediately examined, individual erythrocytes should be

[1] *Arch. f. exp. Path. u. Pharm.*, vol. xxxi.
[2] *Deutsch. med. Woch.*, 1899, vol. xv.

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visible standing upon their edges. In order to obtain a layer of blood of the requisite depth, we should vary the size of the drop of blood upon the slide and avoid exerting any pressure upon the cover-glass. The specimen should not be too thick, since this interferes not only with the formation of rouleaux, but also with their recognition. Poikilocytosis, as well as a diminished number of erythrocytes, naturally tends to prevent the formation of rouleaux. From our investigations up to this time we cannot say whether the formation of rouleaux is disturbed by a diminished viscosity of the blood.

Microscopic Determination of the Amount of Fibrin in the Blood.

In addition to the quantitative estimation of the separated and purified fibrin from a given quantity of blood,[1] the microscopic examination of a fresh blood-specimen may sometimes give a fair idea of the amount of contained fibrin. If a fresh blood-specimen be protected against drying by smearing the edges of the cover-glass with paraffin, and allowed to stand for a quarter or a half-hour, the separated fibrin may be recognized by strong magnification in the shape of a fine network more or less distinctly spread out between the red blood-corpuscles. In order to form a conception of the quantity of fibrin present, the thickness of the specimen must be considered, since, other things being equal, the thicker the specimen, the more marked will be the fibrin network. If the specimen be very thin, the fibrin network may entirely escape observation. A place in the specimen should consequently be selected which fulfils the requirements laid down for the examination of the blood for rouleaux (p. 635)—*i. e.*, immediate observation should reveal isolated red blood-cells standing upon their edges, and, subsequently, distinct spaces should be seen between well-formed rouleaux. When the blood is rich in fibrin, as in inflammatory affections and particulary in pneumonia, the spaces between the rouleaux are entirely filled by a thick fibrin network; while if the blood contain but little fibrin, it is concentrated about the collections of blood-platelets, in the shape of poorly formed stars. In general the quantity of fibrin in the blood goes hand in hand with the degree of leukocytosis; and in leukopenic diseases, such as typhoid fever, a pronounced diminution of the amount of fibrin is consequently not without diagnostic importance.

MICROSCOPIC APPEARANCE OF ERYTHROCYTES.

(Poikilocytosis; Staining Capacity of the Red Blood-cells; Polychromatophilic Changes; Granular Basophilic Degeneration.)

The red cells or erythrocytes are normally biconcave disks. Under pathologic conditions, however, they may present very abnormal shapes, a condition which has been called by Quincke poikilocytosis. Poikilocytes may assume any shape (compare Fig. 233). Their size, too,

[1] After washing the clot with chloroform water, alcohol, and ether, the quantitative estimation may be made either by weighing or by Kjeldahl's method.

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may vary considerably. Poikilocytosis is observed in all cases of *grave anemia*, especially in *pernicious types*, in *leukemia*, in the *cachexia* of *carcinoma of the stomach*, and rarely in the innocent types of anemia, such as *chlorosis*.

Maragliano considers these degenerative types of red blood-cells. Thus, if normal blood is examined under a cover-glass at the temperature of about 26° to 27° C., a number of progressive changes occur in the corpuscles, and these same changes are found in the fresh blood under pathologic conditions. After thirty to seventy minutes the so-called *endoglobular changes* occur. A colorless region irregularly outlined develops from the center and shows an ameboid motion. Considerably later, three to four hours, so-called total alteration of the blood-cells begins, and the external shape is changed. This change commences with the formation of a mulberry-like appearance, which, according to Maragliano, has incorrectly been considered to be the result of drying. The blood-corpuscles gradually become more and more deformed (ten to twelve hours); pseudopodia develop very much with an ameboid-like motility like that of the decolorized spots above described. Poikilocytes are formed in this way. Maragliano has called attention to the fact that not only poikilocytes, but all the other types of degeneration described, especially the crenated types of blood-cells, may be found in the fresh preparation of severe blood-diseases. Less serious pathologic changes of the blood manifest themselves by the fact that these changes take place outside of the body sooner than in normal blood-specimens.

FIG. 233.—Poikilocytes in pernicious anemia.

These changes have diagnostic value only when they are seen in absolutely fresh specimens. Pressure on the tissues when removing the blood and on the cover-glass must be avoided, because this may produce artificial deformity of the blood-cells. On the other hand, the abnormally rapid appearance of deformity in blood apparently normal on removal indicates a certain lack of resistance of the red cells, and is not entirely without diagnostic interest. The crenated types are of especial interest in this respect, although they have not yet been studied from a clinical standpoint.

So far as the staining capacity of the blood-corpuscles is concerned, the dry preparations take acid stains very readily (eosin, orange-G). The intensity of this stain depends upon the amount of hemoglobin contained in the cells. This is of practical importance, because after a little practice the degree of anemia and the oligochromatic quality of the blood can be made out from dry specimens. The difference between normal red cells and those deficient in hemoglobin is much more striking in the stained dry preparation than in unstained cells.

Degeneration of the red cells described by Maragliano may be demonstrated by staining. Living red cells are achromatophilic—*i. e.*, they do not take any stain. After they have been fixed by drying and heating, they stain with eosin and the chemically related acid pigments. Endoglobular degenerated blood-cells devoid of color in the center do not act in this way. The decolorized portion in dry preparations stains with hematoxylin, whereas the peripheral region containing hemoglobin stains with eosin. To demonstrate this, the normal staining methods described upon p. 631 may be employed. The fact that the endoglobular region stains, proves that it is not a vacuole, as was formerly believed, a view which is still held by some authorities.

The change in tinctorial properties of the red cells, termed "anemic degeneration" by Ehrlich, and "*polychromatophilic degeneration*" by Gabritschewski, is as yet little understood. In this condition the normal red blood-cells and the poikilocytes, which stain with the acid component (eosin) in the usual staining mixtures, are tinged various shades, from eosin-red with a bluish tinge to pure violet, when they are treated with eosin methylene-blue (Chenzinski's solution)

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or eosin hematoxylin. This change has been noticed in nucleated red cells, and, like poikilocytosis, is found in all severe cases of anemia. Occurring under such conditions, it is suggestive of degeneration, and was, therefore, described as such by Ehrlich and Gabritschewski. This view, however, has become untenable, since we know that polychromatophilic staining is present in the early developmental stages of erythrocytes. It could be imagined quite as easily that the polychromatophilic changes were indicative of processes of regeneration; at any rate, it is well for the present time to avoid the term degeneration.

Another peculiar change is the so-called *granular (granular basophilic) degeneration of the erythrocytes.* This has been closely studied by Lazarus, Askanazy, Plehn, Grawitz,[1] and others. In specimens stained with Jenner's solution, Chenzinski's solution, or by brief immersion in Löffler's methylene-blue,[2] some of the erythrocytes are seen to contain a number of bluish-black (basophilic) granules (see Plate 6, Fig. 4, *a*). This change has been found chiefly in conditions associated with destruction of the erythrocytes, such as pernicious anemia, leukemia, certain forms of tropic anemia, carcinoma, chronic lead-poisoning, sepsis, and malaria. Basophilic granulation is of the greatest diagnostic importance in the recognition of chronic lead-poisoning (Nägeli). The statement of Grawitz, that granular basophilic degeneration has never been found in chlorosis, has not been confirmed. Rosin and Biber state that they have observed basophilic granules in the erythrocytes of healthy individuals. Grawitz succeeded in producing this change in mice by overheating the animals. Lazarus and Askanazy look upon the basophilic granules as the remains of disintegrated nuclei ("karyolytic fragments"), but Grawitz attributes them to degenerative changes in the stromata of the erythrocytes. Others, on the contrary, regard the basophilic granulation as a phenomenon holding some relation to the regeneration of the blood. Jawein,[3] for instance, observed this change in a patient recovering from a Bothriocephalus anemia. It is probable that basophilic granulation is intimately related to erythrocytic polychromatophilia, with which it is frequently associated. Ehrlich and Lazarus designate these red blood-corpuscles by the non-committal term "punctate erythrocytes." They are also spoken of as erythrocytes with basophilic granulation.

ERYTHROCYTIC SHADOWS.

These forms appear in the blood and have the shape of red blood-corpuscles, but are absolutely devoid of color, so that the central depression can no longer be distinctly recognized. They are the stromata of red blood-corpuscles which have lost their color. They are produced artificially when sufficient water to lake the red blood-corpuscles is added to normal blood. They are found pathologically where red blood-corpuscles are being rapidly destroyed, as in hemoglobinuria occurring independently or as the result of poisoning with chlorate of potash or other blood-poisons. They may also be found, although in smaller numbers, in pernicious anemia.

VARIATION IN THE SIZE OF RED BLOOD-CORPUSCLES.

Even in a healthy individual all the red blood-corpuscles are not of the same size. The normal size varies, according to different authors, between 6 and 9 μ (averaging about 7). The very large cells are called giant corpuscles, or macrocytes; the small ones, microcytes. According

[1] *Deutsch. med. Woch.*, 1899, No. 36, and 1900, No. 9.

[2] Concentrated alcoholic solution of methylene-blue 30.0, 0.01 per cent. potassium hydroxid 100.

[3] *Berlin. klin. Woch.*, 1901, p. 35.

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to Gram, the red blood-corpuscles are larger in northern than in southern countries. There may be very great differences in size in the same blood under pathologic conditions, especially in so-called pernicious anemia, where both macrocytes and microcytes are numerous. The latter are assumed to represent broken-down poikilocytes. The presence of macrocytes seems to bear some relation to the megaloblasts; they are therefore more or less peculiar to pernicious anemia.

The Volume Quotient or the Volume Value of the Erythrocytes.—We are indebted to J. A. Capps[1] for an interesting study of the size of the erythrocytes. By means of the hematocrit and without the addition of fluid (see p. 629) this author has determined the volume of the erythrocytes as compared to the volume of the whole blood. In normal cases he obtained a volume of 50 per cent. If this value be regarded as 1, the volume in pathologic cases may be calculated in per cent. of the normal volume, just as is the amount of hemoglobin. Capps next counts the erythrocytes in the blood under observation, and expresses this number in per cent. by comparing it with the normal number of erythrocytes. By dividing the volume of the erythrocytes by the number of the erythrocytes (both expressed in percentages) he obtains the so-called volume index of the erythrocytes, for which expression the author would suggest substituting "volume quotient" or "volume value" of the erythrocytes, analogous to the hemoglobin quotient or hemoglobin value of the erythrocytes. This quotient is the measure of the average volume of the individual erythrocyte. Under normal conditions, it is evidently equal to 1. Capps found that an increase in the volume quotient of the erythrocytes is one of the most constant and accurate characteristics of pernicious anemia, which agrees with the well-known fact that many macrocytes are present in this disease, and that the hemoglobin quotient is likewise greater than 1. In contrast to this, the so-called secondary anemias usually show a diminished volume quotient of the erythrocytes. The same is true of chlorosis, in which affection a normal or slightly diminished volume quotient gives a good prognosis, while a markedly diminished volume quotient is less favorable. When utilized in this manner, the volume quotient is much more reliable for prognosis in chlorosis than is the hemoglobin percentage or the hemoglobin quotient.

Capps also found that in normal erythrocytes with a volume quotient of 1, the discoplasm is saturated with hemoglobin, so that if the hemoglobin quotient becomes greater than 1, it indicates an enlargement of the erythrocytes. Upon the other hand, the hemoglobin quotient may fall irrespective of a corresponding diminution of the volume quotient. It consequently follows that if the hemoglobin quotient of the erythrocytes is above normal, the volume quotient must also be increased; while if the hemoglobin quotient is below normal, the volume quotient is not necessarily diminished. The osmotic pressure of the blood-plasma seems to have an influence upon the volume quotient; this is difficult to understand, and requires further investigation.

NUCLEATED RED CELLS (ERYTHROBLASTS) AND FREE NUCLEI.

Nucleated red blood-corpuscles do not occur in normal blood. They may be considered as evidence of some abnormal condition of development. They are found especially in anemic conditions. Ehrlich subdivides the nucleated red blood-cells into normoblasts and megaloblasts. The normoblasts are red blood-corpuscles about the size of normal cells, with one or sometimes several nuclei. These nuclei stain intensely with nuclear stains, such as hematoxylin, usually much more intensely than the nuclei of leukocytes or than any other nuclei. This staining quality

[1] *Jour. Med. Research*, vol. x., 3, Boston, 1903.

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is very characteristic, and enables us to recognize the free nuclei of normoblasts when found in the blood. Unstained, the nuclei of normoblasts appear as bright centers in the red blood-corpuscles, free from hemoglobin. They differ from the normal depression of red cells by their sharp outline, and from endoglobular degeneration in being somewhat granular (a normoblast is represented in Plate 6, Fig. 4). Megaloblasts, which are also nucleated red cells, are considerably larger than normoblasts—two to four times as large. The nucleus is somewhat larger than that of a normoblast, but occupies a comparatively smaller portion of the cell. It is less distinctly outlined, and does not have the same affinity for nuclear stains, so that it is usually but very faintly stained. Megaloblasts not infrequently show polychromatophilia. It is usually easy to differentiate well-marked normoblasts from megaloblasts, but many nucleated red blood corpuscles are difficult to classify. Most hematologists agree with Ehrlich that there is a distinct difference between normoblasts and megaloblasts, that the former represent the type of blood-formation in the adult, and the latter the embryologic stage of blood formation. The fate of the nuclei in the two types varies. The normoblasts become fully developed red blood-cells by extrusion of the nuclei; the megaloblasts, by disintegration of the nuclei inside of the corpuscle. Of clinical significance is the fact that normoblasts are observed in those anemic conditions in which the erythrocytes develop after the type of the fully grown organism. To this group belong cases of acute and chronic hemorrhages, and anemia following inanition, cachexia, blood-poisoning, hemoglobinemia, etc.—*i. e.*, in so-called secondary anemias and also in chlorosis. Megaloblasts, on the contrary, seem to be the clinical indication of some severe degenerative change of the bone marrow, which it is presumed is subjected to abnormal chemical (toxic) influences. They are frequently found in pernicious anemia. This occurrence makes the prognosis grave and unfavorable, except in the type of anemia which is due to the presence of the Bothriocephalus. In leukemia both normoblasts and megaloblasts are observed, the latter predominating.

In secondary anemia large numbers of normoblasts may at times be found in the blood, the so-called "blood-crises" of v. Noorden. Megaloblastic blood-crises have not been observed.

In poikilocytosis, deformed blood-corpuscles with nuclei are sometimes found. They are termed poikiloblasts, and according to their morphology may be normoblasts or megaloblasts.

VARIETIES OF LEUKOCYTES.

The following varieties of white blood-corpuscles may be differentiated (Ehrlich and Lazarus):[1]

I. LEUKOCYTES IN NORMAL BLOOD.

(*a*) **Lymphocytes.**—These are derived from the lymph glands. They are small cells about the size of red blood-corpuscles, with a large centrally placed nucleus and a small margin of protoplasm. The nucleus

[1] Die Anämie, *Nothnagel's Sp. Path. and Therap.*, 1898.

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DESCRIPTION OF PLATE 6.

FIG. 1.—**Pernicious Anemia**: *a*, Normal erythrocytes; *b*, erythrocytes poor in hemoglobin; *c*, poikilocytes; *c'*, microcytes; *d*, macrocytes; *e*, polychromatophil erythrocytes; *f*, basophil erythrocytes; *g*, polychromatophil megaloblast; *h*, polynuclear neutrophil leukocyte. (Magnified 570; oil-immersion Leitz $\frac{1}{12}$, oc. 1; tube-length 170 mm. Jenner's stain).

FIG. 2.—**Myelemia**: *a*, Erythrocytes; *b*, polynuclear neutrophil leukocytes; *c*, neutrophil myelocytes with large nuclei; *d*, neutrophil myelocytes with small nuclei; *e*, polynuclear eosinophil leukocyte; *f*, eosinophil myelocytes; *g*, mast-cell. (Magnification and stain as in Fig. 1.)

FIG. 3.—**Simple Elements of the Blood**: *a*, Basophilic erythrocyte; *b–g*, various forms of erythrocytes; *b*, normoblast; *c*, polychromatophil megaloblast; *d*, poikilomegaloblast; *e*, polychromatophil erythroblast the size of which corresponds with the megaloblasts, but the nucleus of which is of the normoblast type; *f*, punctate megaloblast; *g*, megaloblast with nucleus split into four parts; *h–l*, various forms of small normal lymphocytes from the same preparation; *k*, the most common form; *m* and *n* (see also Fig. 4 x), large normal lymphocytes (in the common form): these all stain more deeply than the pathologic lymphocytes shown in Fig. 4; *o*, large mononuclear leukocyte from normal blood; *p*, form intermediary between the mononuclear and polynuclear leukocyte containing sparse granules. (Magnification and staining as in Fig. 1.)

FIG. 4.—**Acute Lymphoid Leukemia**: *a*, Erythrocytes; *b*, polynuclear neutrophil leukocytes; *c*, large lymphocytes; *d*, neutrophil myelocyte; *e*, polychromatophil erythroblast; *f*, free nuclei of normoblast. (Magnification and staining as in Fig. 1.)

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PLATE 6.

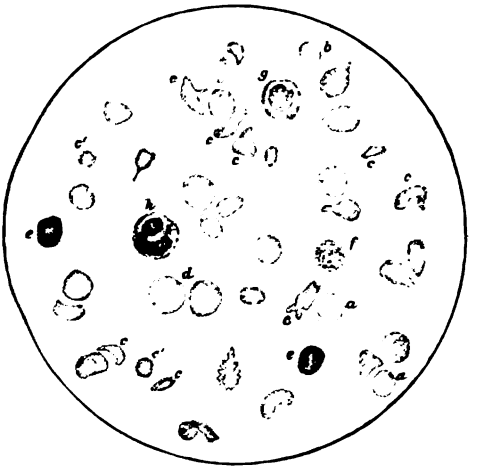

FIG. 1.

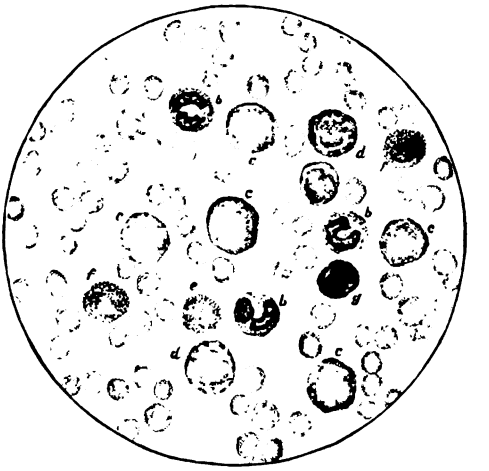

FIG. 2.

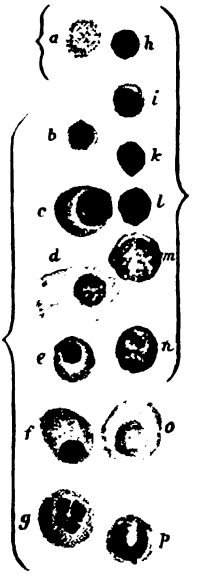

FIG. 3.

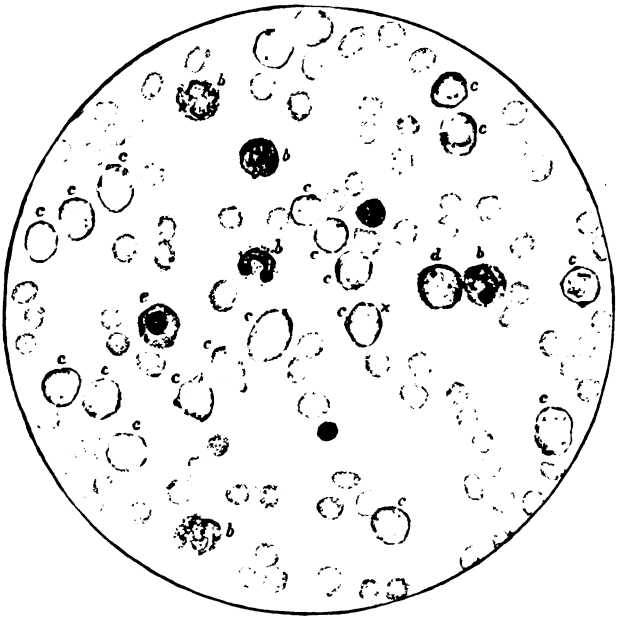

FIG. 4.

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stains rather intensely with nuclear stains, especially hematoxylin, and somewhat less intensely with methlyene-blue and the triple stain. The protoplasm is free from granules. In children and in lymphatic leukemia so-called "large lymphocytes" are found as well as the small ones. These may be double the size of red blood-corpuscles, but are otherwise very much like the smaller ones in appearance. The nucleus may be irregular or may be composed of several parts. The normal number of lymphocytes is 22 to 25 per cent. of the total number of leukocytes—*i. e.*, 1500 to 1700 per cubic millimeter.

According to Pappenheim,[1] lymphocytes are also found in the bone marrow; in fact, he believes that these lymphocytes of the bone marrow are the progenitors of all the myeloid elements. This conception, if it prove correct, has a far-reaching significance, since the principal difference between lymphatic and myelogenic leukemia, in reference to the localization of the changes, would then disappear, and this is what Pappenheim actually claims. A fact in favor of this supposition is that a myeloid metamorphosis of the lymphatic glands has occasionally been observed in myeloid leukemia.

(*b*) **Large Mononuclear Leukocytes.**—These are cells two or three times as large as red blood-corpuscles, with large oval nuclei, usually situated eccentrically and staining faintly. There is a relatively large amount of protoplasm, which is also free from granules. They are frequently mistaken for large lymphocytes, but have more protoplasm, and a nucleus which stains very faintly. They are derived in all probability from the bone marrow, and should be considered the parent types of the following cells (*c* and *d*). The normal percentage of this group is about 1 per cent. of the leukocytes—*i. e.*, 70 for each cubic millimeter.

(*c*) **Transitional Cells.**—These are very much like (*b*), except that the nucleus is quite irregular in shape and stains more readily, and the protoplasm contains scattered neutrophilic granules. The number of transitional cells is normally from 2 to 4 per cent. of the leukocytes —*i. e.*, 140 to 380 per cubic millimeter. In counting they are usually included in one group with the related type (*b*). The normal number for the combined group (*b* and *c*) is about 3 to 5 per cent—*i. e.*, 210 to 350 per cubic millimeter.

(*d*) **Polynuclear** or, better, **polymorphonuclear neutrophilic leukocytes,** are characterized by a polymorphous, irregularly shaped or bent nucleus, which may be readily confounded with multiple nuclei. Indeed, acted upon by acetic acid, it may be separated into several nuclei, a change which also occurs probably when the leucocytes leave the vascular system and become pus corpuscles (hence the incorrect name "polynuclear cells," which is almost impossible to eradicate). The nuclei stain very intensely, and the protoplasm is very densely packed with neutrophilic granules. The iodin reaction affects chiefly these cells (see p. 634). The normal number of polymorphonuclear cells is about 70 to 72 per cent. of the leukocytes—*i. e.*, 4900 to 5040 per cubic millimeter.

[1] "Nenere Streitfragen aus dem Gebiete der Hämatologie," *Zeits. f. klin. Med.*, vol. xlvii., p. 3 and 4, 1902.

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(*e*) **Eosinophilic Cells.**—These resemble the polymorphonuclear neutrophilic cells, except that the small neutrophilic granules are replaced by coarse oxyphilic granules. The coarse granules refract the light so strongly that these cells are readily recognized without staining. When unstained, however, they might be mistaken for mast cells (see below), although the granules of the latter are usually much coarser. The eosinophiles normally furnish between 2 to 4 per cent. of the leukocytes —*i. e.*, 140 to 280 per cubic millimeter.

(*f*) **Mast cells** are cells of the polymorphonuclear or transitional type with marked basophilic granules, which are quite large, uneven, and irregularly distributed and are not distinctly stained by the triple stain. The number of these cells in normal blood is about 0.5 per cent. of the leukocytes—*i. e.*, 35 per cubic millimeter.

These figures for the proportions of the various kinds of leukocytes in the blood should be modified in the case of children below five years of age, where the mononuclear cells predominate. From the fifth year on the polynuclear cells become 50 per cent.[1] There is as yet no more accurate information regarding the occurrence of any individual type of leukocyte in children of different ages.

II. PATHOLOGIC LEUKOCYTES.

(*a*) **Mononuclear Neutrophilic Cells** (*Myelocytes, "Ehrlich's Markzellen."*)—These are large cells with a large, faintly staining nucleus, differing from the large mononuclear cells of normal blood by the presence of neutrophilic granules contained in the protoplasm. They are not found in normal blood, or if so they are so scattered that they are not included among normal leukocytes. They should be considered a preliminary or transitional stage of abnormal polymorphonuclear cells, and are characteristic of the blood in splenomyelogenous leukemia. They may be found under other pathologic conditions; for instance, in malignant tumors of the bone marrow, in infantile anemia, in pseudoleukemia, in leukocytosis, and especially in diphtheria.

The presence of a considerable number of myelocytes in the blood always indicates a serious change in the bone marrow. When associated with leukocytosis, their occurrence evidently signifies that immature elements have gained access to the circulation from the bone marrow through chemotactic influences, and in a certain sense is indicative of an insufficiency of the bone marrow. Their occurrence in great numbers in diphtheria makes the prognosis unfavorable. In the medullary metastases of malignant tumors, the appearance of myelocytes in the blood is of great diagnostic importance.

(*b*) **Mononuclear Eosinophilic Cells** (*Ehrlich's and Lazarus' Eosinophilic Myelocytes.*)—These authors consider them to be early transitional forms of abnormal eosinophilic cells, bearing the same relation to these cells as do the neutrophilic myelocytes to the neutrophilic polymorphonucleated cells. They occur especially in myelogenic leukemia. Very small cells of this sort have been termed eosinophilic microcytes.

(*c*) **Small Neutrophilic Pseudolymphocytes.**—These are

[1] Besredka, *Annales Pasteur*, 1898, No. 5, p. 327, *et seq.*

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small mononuclear cells with a deeply staining nucleus and neutrophilic granules. They are very rare. Ehrlich and Lazarus believe that they result from the division of ordinary polynuclear cells.

(*d*) **Irritation Forms** (Türk).—These are mononuclear cells without granulation, resembling the lymphocytes in size. Their protoplasm stains dark brown with the triacid mixture. Türk found them under conditions similar to those associated with myelocytes.

With the exception of the lymphocytes, which are derived from the lymph glands, probably all normal and pathologic leukocytes are derived from the bone marrow. Recent investigations show that the spleen does not appear to play an important, but quite a secondary, part in their formation. This source may nevertheless give origin to some of the lymphocytes. The pathologic forms originate chiefly in the bone marrow, although there is considerable variation under different pathologic conditions. Sometimes the bone marrow is actively employed in the production of lymphocytes (lymphoid leukemia), while in other instances there is a myeloid metamorphosis of the substance of the lymphatic glands which furnishes elements such as those which normally originate only in the bone marrow. In some leukemias the spleen may also take part in the production of lymphocytes and liberate them into the blood, or throw elements into the circulation which are usually of myeloid origin.

From his investigations of the bone marrow Nägeli[1] has recently described, under the name of *myeloblast*, a new type of cell. This is a collective term for bone-marrow cells without granules, and is especially applicable to the smaller type, which up to the present time have been included in the bone marrow and in the blood as lymphocytes, because of their correspondence in size. According to Nägeli, they have a reticular nucleus and exhibit different staining characteristics from the lymphocytes, the chief of which is that in specimens stained with the triacid mixture the nucleus of the lymphocytes is dark, and that of the myeloblasts considerably lighter. If, on the other hand, the slide is kept in an incubator at 131° C., and stained with pure alcoholic methylene-blue, the nuclei of the lymphocytes will appear pale, and the nuclei of the myeloblasts dark. The reverse is true for the protoplasm, that of the lymphocytes staining dark, and that of the myeloblasts staining lighter. Myeloblasts, as contrasted with the fully developed types, are found in increased numbers in the bone marrow in pernicious anemia, myelogenic leukemia, and typhoid.

It remains for future investigation to decide how much of Nägeli's observations will be corroborated, and to determine the relation of the myeloblasts to the above-mentioned varieties of cells. According to Nägeli, the large mononuclear cells in normal blood correspond to the large myeloblasts. Nevertheless the small myeloblasts (myeloblast in the strictest sense of the word) may appear in the blood under pathologic conditions (leukemia, pernicious anemia, and typhoid), and should then be distinguished from the lymphocytes, with which they have been confounded up to the present time. This may be done by the above-named characteristics, although it is not always easy.

DIFFERENTIAL COUNTING OF LEUKOCYTES AND THEIR PROPORTIONS UNDER NORMAL CONDITIONS.

If it be desired to differentiate simply the mononuclear and polynuclear elements, the differential count may be made from blood treated with dilute acetic acid, which renders the nuclei quite distinct. For

[1] *Deutsch. med. Woch.*, 1900, No. 18.

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the further differentiation of the several varieties of leukocytes (see p. 640), dry specimens stained with eosin hematoxylin have usually been employed, and carefully prepared give a sufficient idea of the numeric ratios of the individual varieties. Advantage may also be taken of dry preparations stained by other methods; for example, by Ehrlich's triacid mixture or Jenner's solution. If these preparations have been carefully prepared—*i. e.*, if the layer is thin and uniform—we can form some opinion of the proportion of the different varieties. If a movable stage is used, the entire preparation may be counted. The total number of leukocytes is determined in the ordinary way.

For differential counting, Ehrlich uses an ocular made by Leitz, by means of which the field of vision is made rectangular and of known size. A larger or smaller field is employed, according to the abundance of the corpuscles in the dry preparation. The total number of the white, and the number of each individual kind, can be conveniently counted by moving the preparation so that different parts enter the field of vision. The figures thus obtained are sufficiently accurate. Ehrlich also employs this method to determine the proportion of white and red blood-cells.

Our knowledge of leukocytosis was at first considerably restricted by the fact that attention was drawn exclusively to the determination of the quantitative relation between the white and the red corpuscles. Similarly, the limitation of the investigation to the determination of the quantitative relations of the various leukocytes, without due regard to the absolute number of these cells, has also considerably restricted our knowledge of this topic. The recent literature, however, indicates that the importance of determining the absolute numbers has been recognized.

Zollikofer's[1] *method* of staining fresh blood-preparations in the counting-chamber, and then determining the absolute number of the different kinds of leukocytes present, is a welcome simplification.

The following two solutions are required:

I.

Eosin, w. g. (Grübler)	0.05
Formalin conc. (40 per cent.)	1.00
Aquæ destillatæ	100.00

Filter.

II.

Methylene-blue, B. X. (Grübler)	0.05
Formalin conc.	1.00
Aquæ destillatæ	100.00

Filter.

Both solutions are kept in dark bottles, and before using are mixed in about equal quantities with a medicine dropper.

The blood is drawn into a Thoma-Zeiss leukocyte counter, and diluted twenty times with the freshly mixed stain. The stain after being mixed must be used immediately, because a precipitate is formed if it stands for any length of time. The dilution of the blood with the stain must be done very rapidly, for if the

[1] *Zeits. f. mik. Technik.*, 1901.

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blood remains in the capillary tube for any length of time, it is hard to render the red blood-corpuscles invisible.

The pipet is shaken for about five minutes, and a drop then placed on the counting-slide. The red blood-corpuscles, unless nucleated, are invisible. The leukocytes are preserved, and the individual kinds can be distinguished. The α granules are stained yellowish to carmin, and are characterized by their size. The ε granules are grayish violet, often filling up the entire body of the leukocyte, but in rare cases scattered only in the protoplasm. The γ granules remain unstained. The leukocytes without granulations may be distinguished by the amount of protoplasm and the size of the nucleus. They are lymphocytes and large mononuclear cells. The nucleus is less distinctly stained in the granular types, and for this reason the mononuclear granulated leukocytes (myelocytes) cannot be accurately separated from the polynuclear. The blood-plates are of a bluish-violet tinge, and may be recognized by their typical grape-like arrangement.

The proportion of the eosin to the methylene-blue can be so regulated that neither the nuclei nor the granules predominate.

The same mixture can be used in dry preparations which have been well fixed at 120° C. They should be stained from one-half to one minute. Besides differentiating the various kinds of leukocytes, it stains malarial parasites, bacteria, and the basophilic granules of the erythrocytes, and shows the polychromatophilic changes of the red-cell plasma.

LEUKOCYTOSIS AND LEUKOPENIA.

By the term *leukocytosis* is understoood the state of the blood, occurring under various conditions, in which there is an increase of the white corpuscles, except in those cases where such an increase is due to the specific or idiopathic disease of the blood, leukemia. Most leukocytoses are characterized, in contradistinction to leukemia, by an increase in the polynuclear neutrophiles, which are derived from the bone marrow, so that the term leukocytosis, used without qualification, usually means the increase of this kind of cell.

An unusual form of leukocytosis, except when it is associated with leukemia, is characterized by an increase in the number of lymphocytes, the so-called lymphocytosis.

Leukopenia is the reverse of leukocytosis—*i. e.*, a diminution in the number of leukocytes.

Leukocytoses are either physiologic or pathologic. The polynuclear elements of pathologic leukocytoses are usually neutrophilic, so that a polynuclear neutrophilic leukocytosis is usually pathologic. There is, however, a pathologic eosinophilic polynuclear leukocytosis. These pathologic leukocytoses are explained by the presence of chemotactic substances in the blood-plasma, most of which exert a positive influence on the neutrophilic, and but little on the eosinophilic, cells. A leukocytosis in which the third kind of granular cells, the mast cells, are increased is not as yet known. But in leukemia these cells are usually very much increased.

PHYSIOLOGIC LEUKOCYTOSES.

To this group belong the leukocytosis of digestion, the leukocytosis after exertion and after a cold bath, the leukocytosis of pregnancy and that of the newborn.

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The **leukocytosis of digestion** begins about one hour after a meal, and reaches its maximum (a 30 to 40 per cent. increase of the white cells) in about three to four hours (Rieder). It is most marked after food rich in proteids. Considering the comparatively slight digestion leukocytosis, any great degree of pathologic leukocytosis can be recognized even during digestion. The lymphocytes are increased in this type of leukocytosis.

Very little definite information is as yet at hand regarding the *leukocytosis after exertion and cold baths.* It is not yet known whether there is an actual increase in the total number of leukocytes, or whether this is only a pseudo-leukocytosis—*i. e.*, one produced by an accumulation of leukocytes in the skin vessels. The *leukocytosis of pregnancy* may amount to 50 or 80 per cent., is present only in the later months of pregnancy, and disappears rapidly after delivery.

Leukocytosis of the Newborn.—The white count is two to three times the normal during the first day of life. It then diminishes to normal, and increases again after the first week, remaining for several weeks at about 50 per cent. above the normal.

In these physiologic leukocytoses the proportions of the individual types of leukocytes are about normal, and consequently there is chiefly an increase of polynuclear neutrophilic leukocytes; but in the leukocytosis of the newborn the mononuclear cells predominate.

LEUKOCYTES IN THE INFECTIOUS DISEASES; INFECTIOUS LEUKOCYTOSIS AND LEUKOPENIA.[1]

Pneumonia.

Pneumonia is usually accompanied by a pronounced leukocytosis. The degree does not correspond to the severity of the infection, nor does it permit of an absolute prognostic conclusion. The leukocytes usually run from 15,000 to 30,000 per cubic millimeter. A sudden drop in the number usually comes on the day of the crisis, or sometimes on the day before. It is not, however, usual to have the number drop below the normal after the crisis. The leukocytosis of pneumonia is composed of neutrophiles, the eosinophiles disappearing during the course of the disease. The reappearance of eosinophiles indicates that the height of infection has been passed, and is, therefore, more or less indicative of a favorable prognosis. It usually takes place on the day of the crisis, but sometimes one or two days before. Later the eosinophiles may be increased beyond the normal limits. A normal leukocyte count with a relative increase of the polynuclear neutrophiles indicates a severe infection and reduced resistance. Leukopenia in pneumonia suggests a dubious prognosis, although it need not necessarily indicate a fatal termination.

Typhoid.

Typhoid differs sharply from pneumonia in being usually characterized by leukopenia. Nägeli continued Rieder's and Türk's investigations, and studied the hematology of typhoid more minutely. He has arrived at the following conclusions:

In the first stage (ascending fever curve) there is usually a neutrophilic leukocytosis. This may be inferred from the condition found in recurrences. It soon diminishes, and is then followed by a diminution in the neutrophiles. The eosinophiles disappear entirely or very nearly. The diminution of lymphocytes is moderate.

[1] Chiefly compiled from Rieder, *Beiträge zur Kenntnis der Leukocytose*, Leipzig, Vogel, 1882; Türk, *Klinische Untersuchungen über das Verhalten des Blutes bei acuten Infectionskrankheiten*, Wien and Leipzig, 1898; O. Nägeli, "Ueber die Typhusepidemie in Oberbipp, ein Beitrag zur Aetiologie and Haematologie des Typhus abdominalis," *Correspondenzbl. f. Schweizer Aerzte*, 1899, No. 18; and the same, "Die Leukocyten beim Typhus abdominalis," *Deutsch. Arch. f. klin. Med.*, 1900, vol. lxvii., p. 279.

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In the second stage (fastigium, continued fever) the number of neutrophiles and lymphocytes diminishes still further, although the latter may be somewhat increased toward the end of this period.

In the third stage (period of remissions) the lymphocytes are frequently increased, and sometimes decidedly so. The neutrophiles are still more diminished, and the eosinophiles begin to reappear at the end of this period. In adults the lymphocytes may remain few in number.

The fourth stage (descending fever curve) is characterized by a still further diminution of the neutrophiles, which reaches its maximum during this period. The lymphocytes are considerably increased, and may be much more numerous than the neutrophiles (crossing of the two curves). The eosinophiles begin to increase slowly and regularly.

Soon after the fever disappears the neutrophiles begin to increase. The lymphocytes are very abundant and the eosinophiles continue to increase. A few days after the disease has run its course, there may be a considerable lymphocytosis, a marked increase of eosinophiles, and normal or slightly increased numbers of neutrophiles. This condition is most pronounced in young individuals, especially two or three months after defervesence. In adults it is less marked, and usually disappears within two months; but in children it persists considerably longer.

During the active stage of the disease the variations in the leukocytes are more pronounced in children than in adults, especially while the lymphocytes are increasing. On the other hand, even if children are extremely ill, the number of leukocytes is rarely as low as in adults (apparently due to a less severe involvement of the bone marrow and the lymphatic apparatus).

Complications, of course, influence the number of neutrophiles. They are usually increased, but generally not excessively, by suppuration, cystitis, parotitis, pleuritis, bronchopneumonia, nephritis, etc. Absence of leukocytosis in spite of complications indicates an insufficiency in the function of the bone marrow. This is a very dangerous condition (impossibility of forming new neutrophiles).

The persistence or early reappearance of eosinophiles, the slight diminution of neutrophiles, and the marked and early increase in lymphocytes all favor the prognosis. Marked diminution of all kinds of leukocytes, or an absence of leukocytosis despite complications, is unfavorable. In the recurrences the blood-picture is similar in all respects to that of the primary attack.

The blood-findings may persist long after the disease has disppeared; they are very characteristic, and, as Nägeli has shown, may prove that a patient has typhoid, although he is apparently well, often with even greater accuracy than is possible with Widal's reaction, a fact which may be of considerable importance in determining the beginning of an epidemic of typhoid.

Acute Articular Rheumatism.

In uncomplicated cases of this disease there is usually a slight polynuclear neutrophilic leukocytosis (about 15,000), which persists so long as there is fever and exudation (Türk). The leukocytosis increases if complications occur (pleura, pericardium). Eosinophiles are absent only in the very early cases, before there has been any amelioration of the symptoms. Later they usually reappear. The presence of eosinophiles is of favorable prognostic importance in this disease also.

Meningitis.

In epidemic cerebrospinal meningitis there is always a pronounced polynuclear neutrophylic leukocytosis of varying degree. In tubercular meningitis the leukocyte count may be normal or distinctly increased (up to 20,000 and more). Absence of leukocytosis, then, points toward tuberculous disease, but its presence does not exclude this disease. The blood-examination in cerebrospinal meningitis furnishes no prognostic data.

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Septicemia.

In this disease there is usually a polynuclear neutrophilic leukocytosis. Individual cases, usually the very severe, may have no leukocytosis.

Erysipelas.

In the majority of the cases there is a moderate degree of polynuclear neutrophilic leukocytosis; in others the leukocyte-count may be normal or only slightly increased. The degree of leukocytosis is of no prognostic value, except that when marked it suggests the formation of pus. In this disease, too, the number of eosinophiles is diminished at the height of the disease.

Scarlet Fever.

Here, too, there is an increase of polynuclear neutrophiles. The characteristic feature of the leukocytosis of this disease is its persistence, for it does not disappear for a considerable period after the rash and the fever have vanished, a fact which can be made use of for diagnosis after the disease has run its course. The eosinophiles are diminished during the early stages of the disease; but after the rash has reached its climax and during the period of desquamation they may be normal, or at times considerably increased. The persistence of leukocytosis after this disease has run its course may possibly have some prognostic value in regard to the danger of late nephritis.

Measles.

Uncomplicated cases in adults are accompanied by a diminution of leukocytes (leukopenia) similar to that in typhoid. This is most decided during the period of eruption, and especially when the latter is at its height. The ratio between the polynuclear cells and the lymphocytes remains undisturbed. After the rash has disappeared the total number of leukocytes increases to the normal, and the number of large mononuclear cells may be above the normal. The eosinophiles up to the height of the disease are either normal or diminished, but subsequently they become slightly increased. A subnormal or later a normal number of leukocytes may be useful in the differential diagnosis between measles and scarlet fever. A marked eosinophilia and leukocytosis would be in favor of scarlet fever rather than measles.

Varicella.

In this disease Erben found an increase of the mononuclear leukocytes, although the total number of leukocytes was normal.

Pertussis.

Pertussis leads to an increase of the lymphocytes and, to a less marked degree, also to an increase of the polynuclear leukocytes.

Mumps.

In mumps, even with high fever, F. Pick[1] found no leukocytosis. He regards this of importance in the differential diagnosis of orchitis due to mumps from that due to gonorrhea, and concludes from it that the former is purely a serous inflammation.

Malaria.

The ordinary benign cases of malaria, according to Türk, regularly show a diminution in the total number of leukocytes both during the attacks and in the interval. During the attack the percentage of neutrophiles is relatively increased

[1] *Wien. klin. Rundschau*, 1902, No. 16.

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at the expense of the lymphocytes and eosinophiles. Schindler has reached different conclusions. He observed a diminution of the leukocytes and an increase of the mononuclear cells between the attacks, with a reappearance of the normal figures at the height of the attack. Türk has counted the plasmodia in connection with the leukocytes in this disease (see p. 656).

Tuberculosis.

This disease gives rise to a polynuclear leukocytosis if it is associated with suppuration. In acute miliary tuberculosis the number of leukocytes is usually normal.

Infectious Inflammations, Tetanus, Suppurations, Perityphlitis, Empyema, etc.

These usually produce a moderate leukocytosis.

In infectious inflammations we are not justified in diagnosing suppuration from the degree of leukocytosis obtained by a single examination; but if we carefully follow the blood-picture during the course of a perityphlitis, as recommended by Curschmann, we may obtain valuable diagnostic aid. The majority of observers have confirmed Curschmann's statements that marked leukocytosis (above 15,000 to 20,000 leukocytes per cubic millimeter) occurs early in perityphlitis, even in the benign cases which recover without operation; but that when this marked leukocytosis continues or assumes a progressive character, reaching 30,000 to 40,000 leukocytes, we have evidence in favor of progressive suppuration and an indication for surgical interference. The leukocytic count is of no value in old encapsulated abscesses, since it may be perfectly normal under these conditions. It is probable that it may be of value for the diagnosis and prognosis of pleural empyemata. The same is true of suppurative affections of the biliary passages.

OTHER POLYNUCLEAR NEUTROPHILIC LEUKOCYTOSES.

Toxic and Medicinal Leukocytosis.—This is observed chiefly in poisoning with the blood-poisons, potassium chlorate, phenacetin, and arsenic, after chloroform, and in hemoglobinuria. These conditions have not been especially studied. The drugs which produce a polynuclear neutrophilic leukocytosis when used internally are antipyrin, antifebrin, and others. The pilocarpin leukocytosis seems to be mainly a lymphocytosis.

Anemic Leukocytosis.—This is observed chiefly after acute loss of blood (posthemorrhagic leukocytosis) and in those types of anemia where the bone marrow is in a state of increased regenerative activity (secondary anemia). The polynuclear neutrophilic cells are chiefly affected. The leukocytosis may be very marked. It disappears when the loss of blood has been replaced by regeneration.

Cachectic Leukocytosis.—This is polynuclear and neutrophilic, and is observed chiefly in malignant tumors, carcinoma and sarcoma.

Leukocytosis of Agony.—This has been observed shortly before death from various diseases, even in those which in themselves, as a rule, produce no leukocytosis. Ehrlich and Lazarus do not regard this as a true leukocytosis, but claim that the white corpuscles are deposited in the peripheral vessels on account of the general depression of the circulation. This accumulation affects chiefly the polynuclear neutrophilic cells.

EOSINOPHILIC LEUKOCYTOSIS, OR EOSINOPHILIA.

Is not only a relative, but an absolute, increase of polynuclear eosinophilic leukocytes. The number of eosinophiles is normally 140 to 280 per cubic millimeter—*i. e.*, 2 to 4 per cent. of the total number of leukocytes. They may increase up to 5000. It must be borne in mind in estimating the degree of eosinophilia that the eosinophiles are more numerous in children than in adults. A pathologic eosinophilia is observed:

1. In *bronchial asthma*, where the eosinophiles may be increased to about 20 per cent. of the total number of leukocytes.

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2. In *pemphigus*, Zappert found 4800 eosinophiles in a cubic millimeter.

3. In various other *cutaneous diseases*, Lazarus found 60 per cent. of the leukocytes to be eosinophiles in a case of urticaria.

4. In *helminthiasis* or *uncinaria* (ankylostoma, oxyuris, bothriocephalus, tenia, trichinosis, and perhaps in other intestinal parasites). Eosinophilia has been observed especially when Charcot's crystals are present in the stools. The diagnostic significance of this condition is manifest. In cases of grave anemia following bothriocephalus infection the eosinophiles may be entirely absent.

5. In *trichinosis*, since eosinophilia is present in the overwhelming majority of these cases, it is of the greatest diagnostic importance,[1] especially in differentiating this affection from typhoid fever, in which the eosinophilic cells entirely disappear at the height of the disease. In trichinosis the number of eosinophilic leukocytes after the fourteenth day may be increased to 40 to 50 per cent. of the total number. The absolute number of total leukocytes is not always increased.

6. After the disappearance of the fever in some infectious diseases, especially if accompanied by the ordinary neutrophilic leukocytosis (pneumonia, acute rheumatism, malaria). In scarlet fever the eosinophiles are increased not only after the fever has vanished but during the fever also. After injection of tuberculin, postfebrile eosinophilia has been observed.

7. In malignant tumors, leading to cachexia.

8. After removal of the spleen and in chronic tumors of the spleen.

LYMPHOCYTOSIS.

This name is applied to those conditions of the blood where there is an increase of lymphocytes, but it does not include the lymphocytosis of lymphatic leukemia, which is sufficiently characterized as an independent disease. We know very little as yet about the occurrence of a pure lymphocytosis. It should be mentioned, however, that in some conditions of ordinary polynuclear leukocytosis—for instance, in the digestion leukocytosis—the lymphocytes may also be increased, and that in the newborn a lymphocytosis is physiologic. Pathologically, a lymphocytosis with a slight polynuclear leukocytosis has been observed in whooping-cough, and after injection of tuberculin and pilocarpin. A lymphocytosis occurs in certain stages of typhoid. A lymphocytosis is of an entirely different clinical significance from a polynuclear leukocytosis, not only because the former cells are derived from the lymphatic glands, but also because, on account of their slightly developed ameboid contractility, their origin must be attributed, according to Ehrlich and his school, not to chemotactic irritants in the blood, but rather to a mechanical washing out of the lymphocytes from the lymphatic glands, and consequently to anatomic changes in the latter structures. In contradistinction to the earlier supposition, it should be noted that recent observations indicate that the lymphocytes are by no means immobile.

BLOOD-PLATES.

Bizzozero and Hayem have recently demonstrated that the granules and clumps of granules visible in any fresh blood-preparation are postmortem formations which owe their existence to rapid disintegration of the elements called blood-plates or hematoblasts.

[1] See Schleip, 75 Naturforscherversammlung, 1903, ref. in *Berlin. klin. Woch.*, 1903, No. 41, p. 946, and *Arch. f. klin. Med.*, vol. lxxx.

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These blood-plates are small, circular or oval, colorless formations, about 3 μ in diameter. They disintegrate very easily, and adhere very readily to each other and to the other elements of the blood. They seem to play an important part in the etiology of white thrombi. Their number is variously estimated at from 200,000 to 500,000 per cubic millimeter.

For the purpose of examining blood-plates before they are destroyed, it is necessary to add some preservative fluid to the blood the moment it is drawn. Hayem recommends the following:

(1) A solution of 1 part of methylene-violet and 5000 parts of 75 per cent. (physiologic) solution of sodium chlorid.

(2) A mixture of 1 part of a 1 per cent. aqueous solution of osmic acid and 2 parts of 0.75 per cent. solution of sodium chlorid. The latter fluid fixes the blood-plates permanently, whereas the former stains them. Bizzozero puts a drop of one of these solutions on the finger-tip, the skin of which has been carefully cleansed. The skin is then punctured through the fluid, so that the elements of the blood come in immediate contact with the fluid. This blood-mixture is placed under the microscope; the characteristic blood-plates are then seen, but as granules.

Blood-plates are counted in a very similar way to the red blood-corpuscles. Bizzozero, however, believes that if this method of counting blood-plates is used, a considerable number adhere to the side of the pipet. He therefore advises that the blood be received directly in a 14 per cent. solution of magnesium sulphate by the process just described. The blood-plates are a little deformed, to be sure, but this solution has the advantage over the other fluids of keeping them isolated. The ratio between red blood-corpuscles and blood-plates can then be estimated by the Thoma-Zeiss counter, the number of red blood-corpuscles determined in the usual way, and the absolute number of blood-plates then reckoned. Affanasiew has recommended the use of a solution consisting of 0.6 per cent. sodium chlorid with 0.6 per cent. peptone and a little methylene-violet. The number of blood-plates may also be found by a determination of the ratio between it and the number of the leukocytes in a dry preparation; the disadvantage of this method is that the blood-plates are found continually clumped.

Little is as yet known regarding the relation of blood-plates to physiologic and pathologic conditions. According to Bizzozero, they are increased in pregnancy, after the loss of blood, in various anemias (chlorosis), in tuberculosis, cholera, etc. They are diminished in fever and acute diseases, but, according to Hayem, increase toward the end of a fever. Denys observed a diminution of blood-plates in purpura. (Compare p. 634 with reference to the Iodin Reaction of Blood-plates.)

THE SO-CALLED CASTS IN THE BLOOD.

Litten[1] first called attention to the fact that peculiar large cylindric formations, moderately refractile to light, were found in fresh unstained blood-preparations both from healthy and from diseased individuals. They are sometimes granular, sometimes homogeneous and flaky. Litten considers them artificial formations due to the preparation of the slide. He maintains that if the cover-glass is dragged a little, they can easily be formed from blood-plates or from red corpuscles, and are of no diagnostic interest. Buttersack[2] considers that they are formed within the vessels, and represent capillary blood-plate thrombi.

MELANEMIA.

By *melanemia* is meant the presence of granular brownish or black pigment in the blood. It is usually found in the interior of white blood-corpuscles, which are often irregular in shape, and less frequently as free plates between the cellular elements of the blood. Melanemia has as yet been found only following long-

[1] *Deutsch. med. Woch.*, 1896, No. 15, p. 230; *ibid.*, 1898, No. 18, p. 188.
[2] *Zeits. f. klin. Med.*, vol. xxxiii.

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continued malarial cachexia and in recurrent fever. In malaria the pigment derived from hemoglobin may show all shades of color. Sometimes it may even be black. The presence of white blood-corpuscles with pigment or of free pigment is of great diagnostic importance in those cases in which the malarial parasites are not readily discoverable.

LIPEMIA.

Blood always contains some fat under physiologic conditions. More marked lipemia is observed physiologically during digestion, and pathologically in chronic alcoholism, in acute phosphorus-poisoning, in severe diabetes, and in fracture of bones, which may lead to fat-emboli. If the blood contains a considerable amount of fat, its pallor and cloudiness is apparent to the naked eye. Under the microscope the fat is usually seen in the form of very fine granules, just as in chyle; in embolic lipemia distinct light-refracting drops may be seen. They are stained black by osmic acid, and red by Sudan III., and dissolved in a dry preparation by the addition of ether.[1]

BACTERIA IN THE BLOOD.

To demonstrate bacteria in the blood, dry preparations are spread between two cover-slips in the usual way. The slides are passed through the flame two or three times, and then stained like specimens of sputum (compare p. 593).

Since the staining of the erythrocytes and of the dried albumin of the blood interferes with the beauty and transparency of the preparations, the procedure suggested by Günther may be followed[2] with advantage, as by it the hemoglobin and part of the albumin are removed from the dried specimen. Günther fixes

FIG. 234.—Recurrence spirillæ in blood. Photograph by Weichselbaum (× 1000).

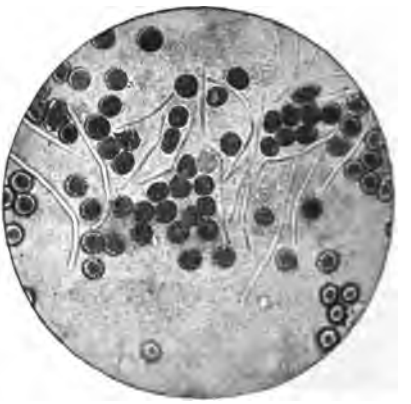

FIG. 235.—Anthrax bacilli in blood, unstained (× 430) (Fränkel).

the dried blood by heat, and then washes it for ten seconds in a 5 per cent. solution of acetic acid. The cover-glass is then dried, and for several seconds the smeared side is held directly over an open bottle of ammonia, which has been previously well shaken, so that the last remains of the acid may be neutralized. The cover-glass is now immersed in the staining solution for a short time and then washed in water. Since the tinctorial characteristics of the erythrocytes depend upon their contained hemoglobin, these structures will appear colorless, and any bacteria which may be present will be easy of recognition.

The *spirillæ of recurrent fever* and the *bacilli of anthrax* (Figs. 234 and 235)

[1] Rieder, *Arch. f. klin. Med.*, vol. lix., p. 444.
[2] *Fortschritte der Med.*, 1885, vol. iii., p. 756.

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are pathognomonic when found in the blood. Both of these micro-organisms may be recognized in unstained specimens.

Bacteria are more easily demonstrated in the blood by cultures than by a microscopic examination—*e. g.*, the streptococci and staphylococci (compare Figs. 218 and 219) found in septicopyemia. The number of micro-organisms found in the blood is always comparatively small, so that considerable blood should be employed to inoculate the culture medium. For this purpose a sufficient quantity of blood is removed from some vein of the arm with the aid of a small syringe fitted with an asbestos piston and thoroughly sterilized. This syringe should contain about 5 c.c. The skin should be first carefully disinfected with alcohol and corrosive sublimate. About 1 c.c. of this blood is added to a tube of agar (liquefiable at 40° C. at the most), to one with 10 per cent. gelatin, and to two bouillon tubes. The fluids are carefully shaken, and the agar and the gelatin tubes are plated. The agar plates and the bouillon tubes are placed in the incubator at 37° C., the gelatin plate at 22° C. Sittmann[1] found pus cocci or staphylococci in the blood in every case of septic pyemia which he examined in this way. The bacteriologic examination of the blood is the most accurate aid for determining pyemia.

In severe cases of pneumonia we have not infrequently been able to demonstrate pneumococci in the blood either by direct microscopic examination or by culture methods. This shows the relationship existing between pneumonia of the human subject, which is usually regarded as a purely local affection, and the pneumococcic sepsis of laboratory animals. The procedure given upon p. 652 for the removal of the hemoglobin with acetic acid greatly aids the demonstration of the capsules of the pneumococci.

Typhoid bacilli may frequently be demonstrated in the blood of typhoid fever patients, either in fresh specimens or in cultures, and, in the writer's experience, may even be found in cases running a mild course. They are most frequently found in the first week of the disease.

The demonstration of *pest bacilli* (Fig. 228) plays an important rôle in the diagnosis of bubonic plague. (For their recognition the reader is referred to the section upon Examination of the Sputum.)

The occurrence of the *bacillus mallei* (p. 602) in the blood is of less diagnostic importance.

Tubercle bacilli (p. 593) have as yet been found in the blood only in acute miliary tuberculosis, and then in very small numbers.

THE BLOOD IN MALARIA; MALARIAL PLASMODIA.

We owe our knowledge of the malarial parasite, first of all, to the pioneer investigations of the French military surgeon Laveran. Further development in the study of the etiology of malaria has been accomplished chiefly by Italian investigators, especially Golgi, Marchiafava, and Celli, and recently by R. Koch and many others. Of the comprehensive articles upon this topic may be cited the monograph of Mannaberg,[2] and more recently those of Marchiafava, Celli, Thayer, and others.

Malarial parasites, ordinarily called plasmodia, are unicellular organisms belonging to the class of sporozoa, subclass hemosporidia, which are on the border line of the animal and the vegetable kingdoms. They consist of small masses of protoplasm, the diameters of which vary between 1 and 10 mm., according to the age and species of the individuals. The younger specimens show active ameboid motion. They develop in the interior of the red blood-cells, destroying their host in their growth. Most varieties alter the hemoglobin of the blood-corpuscles which they inhabit to a brownish-black pigment. This pigment is

[1] *Deutsch. Arch. f. klin. Med.*, vol. liii., p. 327, 1894; see Petruschki, *Zeits. f. Hyg. u. Infectionskrankheiten*, vol. xvii., p. 59.

[2] Jul. Mannaberg, *Die Malariaparasiten*, Wien. 1893, and "Die Malariakrankheiten," in *Nothnagel's Spec. Pathologie u. Therapie*, 1898.

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generally visible in the interior of the parasite, undergoing active dancing motion, which may partly depend upon the intrinsic motion of the protoplasm, and should not be confounded with the much slower ameboid motion of the organism. After the parasite has reached a certain stage of development within the red blood-cell, and has more or less completely consumed the latter (Plate 7, Figs. 1 to 4 and 11 to 13), it multiplies by sporulation (Plate 7, Figs. 5 to 8 and 16). This takes place by a process of division, varying in the different types, but always in such a manner that nothing remains of the mother organism but the pigment. The free pigment is taken up by the white blood-corpuscles, and, when these are subsequently disintegrated, is deposited in the organs (melanemia). The young parasites produced by subdivision are known as spores. They differ, however, from the fully developed parasites only in their size. They penetrate fresh blood-corpuscles, and then repeat the stages of development. The type of development of the quartan malarial parasite is represented in Plate 7, Figs. 11 to 17; of the tertian organism, in Figs. 1 to 10. The attacks of fever so characteristic of malaria are, generally speaking, associated with sporulation of the parasite. These attacks follow at regular intervals in the ordinary types of malaria, because the time for the cycle of development of one generation of parasites is a definite quantity which can be usually expressed in terms of days—*e. g.*, tertian and quartan. Types with longer periods (quintan) are more uncommon and not so thoroughly understood. Most quotidian types, at least in regions where tertian and quartan fever prevail, represent composite forms which arise by different generations of tertian or quartan parasites developing in the organism on succeeding days, the one generation being a day older than the other. A quotidian type of fever will be observed if two generations of tertian or three generations of quartan parasites complete their cycle of development on days succeeding each other. This view of quotidian types was prevalent even before the parasite of malaria was discovered, because the attacks were peculiarly grouped together in pairs with reference to their severity and other symptoms (tertiana duplex, quartiana triplex). Besides these combined quotidian types, a true quotidian type whose cycle of development lasted only one day was supposed to exist, until in recent times the investigation of R. Koch (more in detail later) disproved this idea.

Our knowledge of the morphologic conditions and the development of the malarial parasites is at the present time so complete, thanks to the work of the above-mentioned Italian authors, that a physician familiar with the morphology of the parasites is able, by examining the blood, not only to recognize the presence of malaria, but also to determine the form, the type, and the clinical course of the attack. We shall refer to this later.

Two other kinds of parasites have been observed in the blood in malaria, the so-called crescents of the æstivo-autumnal type of the disease, and flagellate bodies.

Figs. 25 to 27, Plate 7, represent types of the crescent series, which are peculiar to the malignant (tropic) form of malaria. They differ in shape from the ordinary plasmodia, and possess a double capsule. The crescents (Figs. 25 to 27, Plate 7) develop in the interior of the red blood-corpuscles, as indicated by the remnant of the red corpuscles frequently seen attached to them. Elongated cigar-shaped and spheric formations (ovals and spheres of the æstivo-autumnal crescents) develop from the crescents by changes in shape slow enough to be observed under the microscope. These forms do not possess any ameboid motion proper. Mannaberg believes that the crescents are formed by approximation and fusion of two plasmodia in the interior of the red blood-cells, and that they are therefore a kind of copulation type, a so-called syzygy formation. The crescents may divide into their component parts by subsequent segmentation. Clinical conditions seem to make it probable that the crescents are more or less permanent forms, which produce symptoms—*i. e.*, fever—only after transformation into the vegetative type. How this process takes place is still uncertain, whether the crescents segment into their component parts, or whether, as Canalis claims, they sporulate also directly after they have changed to the spheric form. At any rate, the crescents seem to have a slow stage of development. During the intermissions of eight to fourteen days free from fever in

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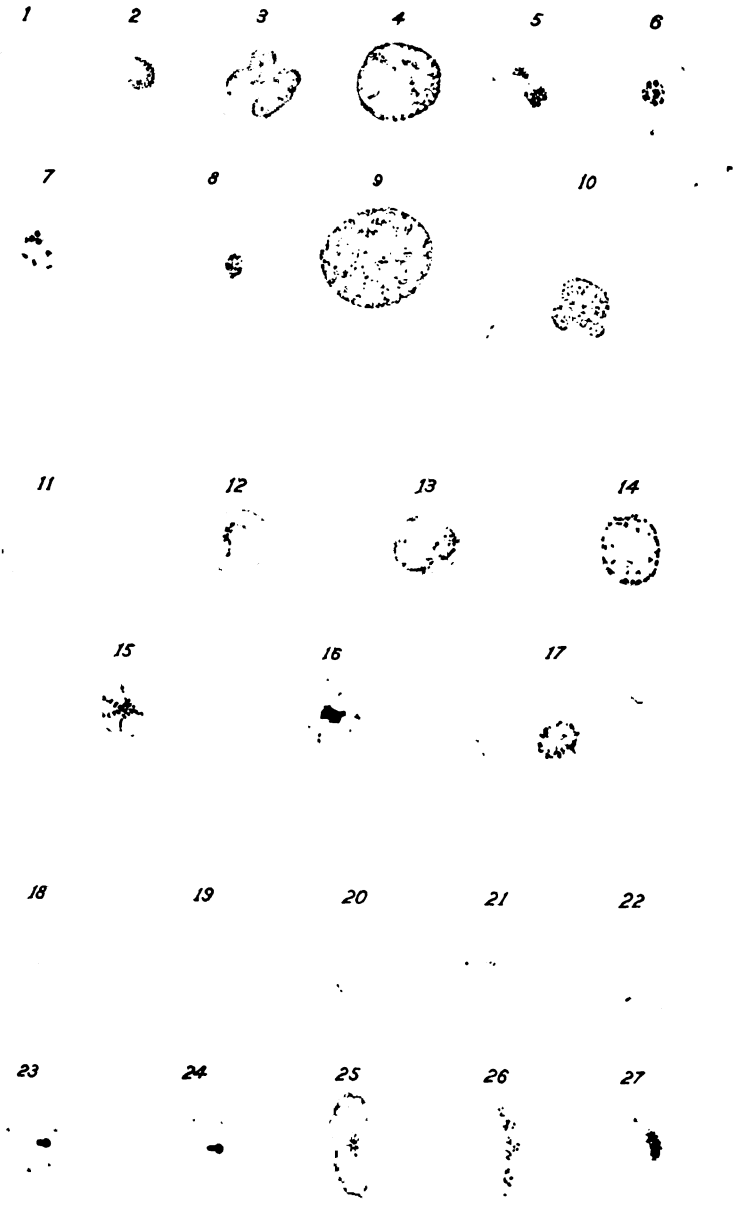
PLATE 7.



VARIOUS FORMS OF MALARIAL PARASITES (Thayer and Hewetson).

FIGS. 1 to 10 inclusive, tertian organisms; FIGS. 11 to 17 inclusive, quartan organisms; FIGS. 18 to 27 inclusive, estivo-autumnal organisms.

FIG. 1.—Young hyaline form; 2, hyaline form with beginning pigmentation; 3, pigmented form; 4, full-grown pigmented form; 5, 6, 7, 8, segmenting forms; 9, extracellular pigmented form; 10, flagellate form.

FIG. 11.—Young hyaline form; 12, 13, pigmented forms; 14, fully-developed pigmented form; 15, 16, segmenting forms; 17, flagellate form.

FIGS. 18, 19, 20.—Ring-like and cross-like hyaline forms; 21, 22, pigmented forms; 23, 24, segmenting forms; 25, 26, 27, crescents.

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the severe tropic malaria, which is, except for such intermission, quotidian, only crescents can be found in the blood. It is therefore probable that these free intervals correspond to the stage of development of the crescents. At any rate, both the crescents themselves and the secondary types developed from them (ovals and spheres) are not independent formations, but stages in the development of the plasmodia, and are entirely confined to the malignant types of malaria. In the tertian, quartan, and composite quotidian types of the temperate zones crescents have never been observed.

The flagellate bodies are found in all kinds of malaria (Plate 7, Figs. 10 and 17). Under the microscope they may be seen to develop from adult plasmodia which have destroyed their blood-corpuscle and have not sporulated and again from the spheres of the crescent series. These flagella suddenly appear at the margin of the parasite; they move very quickly and lash the surrounding blood-corpuscles without, as a rule, causing any particular motion to the parasite itself. Every once in a while individual flagella will tear themselves off and move about with great rapidity in the field. These isolated flagella are the only formations in the whole cycle of development of malarial parasites which have the power of changing their position to any great extent. Probably the flagellate forms develop only in microscopic preparations; Mannaberg therefore considers that they correspond to a saprophytic existence. Recently, however, their biologic significance has been explained in a very interesting and probable way, which will be discussed later.

Unstained blood-preparations may be employed to detect the malarial parasites. They should be especially thin (p. 631), so that the individual blood-corpuscles may not overlap nor stick together and form rouleaux. The preparations are best examined with an oil immersion, Abbé's condenser, and a half-open iris. The pigmented forms, the crescents, and the flagellate types can be very easily detected, but the hyaline forms are not so easily differentiated from the endoglobular types of degeneration or the so-called vacuoles of the red blood-corpuscles. Differences of light refraction, however, cause a more sharply outlined contour in the latter than in the plasmodia. Ameboid motion is common to both. Doubtful cases can be decided by examining stained preparations. Where so few plasmodia occur as to be difficult to demonstrate, Türk recommends diluting the blood ten times with a ½ per cent. solution of acetic acid, just as for counting plasmodia and leukocytes. The leukocytes and plasmodia will be preserved and the red blood-corpuscles destroyed.

Malarial blood should be stained as indicated on p. 631. Fixation should be accomplished not by heating, but by allowing the slides to remain for five minutes in absolute alcohol. They are then placed for half an hour in a half-concentrated and freshly filtered aqueous solution of methylene-blue, or else stained for five or six minutes in the following (Plehn's contrast stain):

Concentrated aqueous methylene-blue	40
Two per cent. solution of eosin, 60 per cent. alcohol	80
Water	40
Twenty per cent. potassic hydroxid	12 drops.

The endoglobular degeneration spots remain colorless, but the plasmodia stain well with methylene-blue and show a characteristic structure—namely, an unstained nucleus, and sometimes a stained nucleolus. This structure, especially the stained nucleolus, enables us to distinguish blood-plates and granular débris of the blood from the spores of malarial parasites. The blood-plates have no nucleus and are devoid of structure.

Mannaberg recommends staining the plasmodia, to show the finer structures, with the following:

Concentrated solution of aqueous methylene-blue	24 parts.
Five per cent. borax solution	16 "
Water	28 "

Filtered after twenty-four hours.

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The preparations remain twenty-four hours in this solution and are then washed with water.

R. Koch has recently recommended a similar solution of borax-methylene-blue for rapid staining. An aqueous solution of 2 per cent. methylene-blue and 5 per cent. borax is diluted with water before being used until a layer 1 cm. thick begins to become transparent. The dry specimen is once passed through the flame, placed in alcohol for twenty minutes, dried, and then dipped into this solution several times, and washed with water until it has become of a slightly greenish tinge. It is then dried with filtered paper and mounted in cedar oil. The malarial parasites will be stained intensely blue, the blood-corpuscles pale green, and the nuclei of the leukocytes darker than the plasmodia.

Romanowski's method of staining can be heartily recommended to show the finer structure of the malarial parasites. The technic is as follows: The slides are fixed either in alcohol or by heating to 105° or 115° C. for half an hour. They are then allowed to float in a solution of 1 part of concentrated aqueous methylene-blue and 2 parts of 1 per cent. aqueous eosin for two to three hours. They are next washed in water, and, if overstained, in alcohol. The stock solutions may be preserved for a long time. The eosin solution must be free from mold, whereas the solution of methylene-blue is said to be better after a little mold has developed on its surface. The required quantity of each solution is filtered off before using. When they are mixed, a considerable precipitate develops, which should not be filtered off. Romonowski claims that a new neutral color is formed in the mixture, which is supposed to stain the chromatin of the malarial parasites particularly intensely. The plasma of the parasite is stained blue, and the chromatin of the nucleus is violet carmin. The success of the stain seems to depend considerably upon the quality of the coloring matter. Höchster's medicinal methylene-blue and eosin are the best dyes. Sometimes the proportions of the solutions must be modified.[1]

To find malarial plasmodia quickly when they are but sparingly present, R. Ruge[2] recommends employing a thick layer of blood from which the hemoglobin has been removed before staining, in a similar manner to the method described upon p. 652. A cover-glass is thickly smeared with blood, dried, and then laid with the smeared surface down in a watch glass containing 2 per cent. formaldehyd and a ½ to 1 per cent. acetic acid. A few minutes suffices to fix the specimen and also to extract its hemoglobin. It is now stained with methylene-blue in the ordinary manner, and the plasmodia may be readily found, even though there be a number of layers of erythrocytes. This procedure has the advantage of employing more than twenty times the volume of blood utilized in the usual cover-glass smear. Ruge admits, however, that there is always a greater or less amount of precipitate, which an inexperienced observer might differentiate from plasmodia with difficulty. He also calls attention to the fact that the ring forms of the young malarial plasmodia (p. 657) are beautifully shown by this procedure.

Counting the plasmodia gives more or less information as to the severity of a malarial infection. Türk counts them in the same way as leukocytes, in a ½ per cent. solution of acetic acid, a method available only for the pigment forms. The time of counting should therefore be immediately before the attack—*e. g.*, in tertian fever, the evening before the day of fever. Türk's figures vary between 6700 and 16,800 per cubic millimeter.

The *diagnostic significance* of malarial parasites in the blood is absolute. The presence of one single parasite is sufficient for a diagnosis of malaria (the pigmented forms and the crescents cannot be mistaken). Negative results are not so certain, because many cases of malaria require a long search before a single parasite is seen. The blood should be examined both during and between the attacks. Even the most expert investigators have been sometimes compelled to make the diagnosis of malaria without finding the parasite. In these cases the parasites

[1] *Deutsch. med. Woch.*, 1903, No. 12, p. 205.

[2] See Mannaberg in *Nothnagel's Spec. Pathologie u. Therapie*, 1899, p. 34.

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are either very few in number or else develop in the tissues rather than in the blood (hypothetic). An inexperienced examiner should always mistrust a negative result and repeat his search. In case of repeated negative blood-examination, suspicion should be directed to the presence of one or more diseases which are easily confounded with malaria clinically—*e. g.*, acute sepsis, irregular cholelithiasis, ulcerative endocarditis, etc.

The blood of severe malaria shows in addition the characteristics of anemic blood—namely, the diminution of hemoglobin and of the number of red cells. Poikilocytosis is by no means uncommon. There is usually no leukocytosis, but rather a leukopenia. The presence of pigment in the white blood-corpuscles or pigment floating free in the blood is of considerable diagnostic importance if the parasites cannot be found. The red blood-cells which are invaded by the parasites may be altered in various ways. The ordinary types usually decolorize the cells gradually, until finally all that is left is an indistinct stroma surrounding the parasite. The blood-corpuscles may be considerably enlarged by the tertian parasites, which differ from the quartan variety in this respect. In the malignant types the infected blood-corpuscles shrink and become darker, and, according to Mannaberg, resemble the color of old brass (brassy bodies.

The majority of investigators have concluded that the various forms of malarial parasites found in the blood represent various developmental phases not only of the same, but also of different species. Each type of malaria which is characterized clinically and endemiologically represents a separate species of parasites with a definite cycle of development. The reasons for these views may be found in Mannaberg's monograph. He describes the following five species of parasites:

1. The parasites of quartan fever.
2. The parasites of the common tertian fever.
3. Pigmented quotidian parasites.
4. Non-pigmented quotidian parasites.
5. Malignant tertian parasites.

Those of 3, 4, and 5 are malignant varieties with crescents, which probably explains the difficulty with which these cases are influenced by quinin.

R. Koch, on the other hand, considers these three groups of parasites as one species, which is to blame for the malignant summer fevers (æstivo-autumnal) of southern Europe, especially of Italy, and of the tropics. Koch therefore distinguishes only the parasites of *quartan*, *tertian*, and *tropic* fever. He shows that a fresh infection of the parasite of tropic fever always produces attacks of a pronounced tertian type (corresponding to malignant tertian parasites). When the disease has persisted for some time and the natural course has been more or less influenced by treatment with quinin, there may develop from the tertian type either a quotidian type, an irregularly remitting, or a continued fever. According to this view there is no true quotidian parasite. Koch's simplified classification is based more or less upon his view that the difference in the pigment which gives rise to the distinction between the so-called pigmented and non-pigmented quotidian parasites is an artificial condition depending upon the method of preparation. When dry preparations were made skilfully and rapidly he found that the young parasites of tropic fever were, as a rule, without pigment or had only very fine granules, and, at any rate, were free from lumps of pigment, even when the fine pigment lent a brownish diffuse hue; lumps of pigment were found only when the parasite underwent subdivision or death. If a wet preparation was allowed to stand for any length of time, lumps of pigment were apt to develop. Koch therefore maintains that the only correct method of examination is by properly prepared dry slides. He considers that the ring-shaped young parasites found with this method (quartan and tertian as well as tropic fever) represent the correct morphologic conditions. Other investigators have observed these ring forms, to be sure, but have considered them partly as artificial products, partly as the result of the circular arrangement of the stained substance and not of the whole protoplasm.

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For determining the various varieties, Koch lays special stress upon the size of the parasite, and the presence of crescents so characteristic of the tropic fevers. He makes the following statements in regard to this matter: The young circular parasites of tertian and quartan fever have a diameter of about one-quarter to one-third that of the red blood-cells. In size and shape they resemble the fully developed parasite of tropic malaria so completely that they cannot be differentiated. But, as a rule, in tertian and quartan fever scattered large pigmented parasites are found in addition to the small circular ones, which makes the diagnosis easy. If the latter should be absent, the determination of the body temperature at the time of examination will furnish sufficient explanation of the significance of the preparation. If the temperature is low and the fever has ceased, the parasites must have completed their cycle of development. The small types found must, therefore, represent the fully developed smaller parasites of the tropic malaria. If, on the other hand, the temperature is high and the patient is in the early stages of an attack, then these small ring bodies must be young parasites of the tertian and quartan groups which have not yet reached their full size.

The variations of sporulation also serve to distinguish the individual varieties. This is seen in Plate 7 better than it can be described. It is of some clinical interest to be able to tell the time of an attack merely from the period of sporulation (Figs. 5 to 8 and 16). It usually takes place three to five hours after the spore forms have appeared.

The *crescents* are said to be formed in the blood eight days after infection. If these persist in the blood along with the other forms of the parasite, after cessation of fever, recurrences may be expected. There is ordinarily no fever when only crescent forms are present.

The significance of the so-called *flagellate forms* has recently been explained. The investigations of Ross, MacCallum, Sakharoff, Koch, *et al.*, with the hemosporidia species halteridium and proteosoma, which occur in birds' blood and which are closely related to the malarial parasites of human blood, have shown that, besides the endogenic cycle of development culminating in sporulation, there is a second sexual cycle of development. Koch divides this into the following stages: 1. Separation of the parasite from the red blood-corpuscle. Differentiation into male and female elements. The thread-like formations which were formerly considered flagella develop from the interior of the male. They separate, propel themselves independently, and play the part of spermatozoa. 2. Fructuation by penetration of the spermatozoa into the female plasmodia. This takes place in the stomach of a mosquito which has sucked the blood from some infected bird. 3. Change of the impregnated female parasites into worm-like bodies. 4. Penetration of the stomach-wall of the mosquito by the latter and their change to coccidia-like spheres. 5. Formation of sickle-shaped bodies in these spheres. 6. Deposit of these, fully developed and liberated, in the poison and salivary glands and perhaps in the other organs of the mosquito. 7. Transmission of young parasites again to birds by fresh bites of the mosquito.

It has been shown that these biologic facts are also true for the parasites of human malaria, especially since the transmission of the disease by mosquito bites, an old and popular belief, has recently been definitely proved experimentally.

It is of considerable pathologic interest to know that the action of quinin results in a necrosis of the parasite. This may be recognized in stained preparations by the disappearance of the nucleus. Very young parasites are more susceptible to its action, which accounts for the rule that it is best to give quinin three or four hours before an attack is expected. The crescents are almost immune to the direct action of quinin, as shown by microscopic examination, so that the action of repeated doses of quinin in malignant types resembles more or less a process of fractional sterilization. The crescents themselves are not disturbed, but the plasmodia developing from them are killed by the repeated doses. In exceptional cases, however, the crescents are not so resistant.

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PARASITIC WORMS IN THE BLOOD.

Of the two helminthes inhabiting the human blood in the tropics, Distomum hæmatobium (Bilharzia hæmatobia) and Filaria sanguinis, only the latter is of diagnostic importance in blood-examinations. The blood of patients infected with filaria (tropic chyluria) contains numerous small thread-like worms, the filaria embryos, 0.21 to 0.36 mm. long and 0.004 to 0.0075 mm. broad (Fig. 205). Strange to say, they are usually found only at night.

CONDITION OF THE BLOOD IN THE MOST IMPORTANT BLOOD-DISEASES.

THE ANEMIAS.

The condition ordinarily called *anemia* really should more correctly be termed oligochromemia; either merely the quantity of hemoglobin in the blood may be decreased, or the number of erythrocytes may also be simultaneously diminished. An actual loss of blood, and then probably merely for a short time, occurs only when the anemia is produced by hemorrhage. In all anemias undeveloped erythrocytes may be present in the blood, as the result either of a disturbed or of an incomplete development. The subdivisions of anemia vary considerably. The following arrangement would seem to correspond best to the actual conditions:

THE SO-CALLED PRIMARY ANEMIAS.

Chlorosis.—The chief characteristic of the blood in chlorosis is a diminution in the amount of hemoglobin. This is not infrequently as low as 20 per cent. The number of red blood-corpuscles is also usually diminished, and sometimes very considerably (1,500,000); but it is more or less characteristic that the diminution of hemoglobin in chlorosis is more marked than the diminution of the number of red cells.

The *color index* (blood-corpuscle quotient of value) is therefore less than 1. The pallor of an individual blood-corpuscle may sometimes be recognized microscopically. Severe cases of chlorosis exhibit poikilocytosis, microcytes, nucleated red cells, and the disintegrating forms of red blood-cells described by Maragliano. Granular degeneration and polychromatophilic changes in the erythrocytes are absent. The number of white corpuscles and of blood-plates is usually within normal figures. A diagnosis of chlorosis cannot always be made from an examination of the blood alone, as in many cases it depends considerably upon the clinical picture. Chlorosis is a disease occurring during the period of development, especially in women, and is probably to be explained by the fact that the amount of blood formed is not sufficient for the demands of the growing organism.[1]

The urine of chlorotics is pale and contains but little urobilin. This favors the theory just given, and indicates that the destruction of red blood-corpuscles is limited, for in some other types of anemia, especially in so-called *pernicious anemia*, the increased destruction of red cells is manifested clinically in a dark urine, which contains a good deal of urobilin. The patient is usually well nourished, and may even be increased in weight. This makes the peculiar clinical picture of chlorosis.

v. Noorden demonstrated that this abundance of fat in chlorotics is not due to the diminution of the oxygen content of the organism from deficiency in the

[1] The greater frequency of chlorosis in women must be associated with the peculiarities of the female sex. Apparently, disturbances somewhere in the generative organs inhibit the stimuli which affect the blood-forming organs, upon which is dependent the normal blood-formation of the female. The conception that blood-formation is intimately connected with the female sexual organs has much in its favor, as it is reasonable to assume that the female body, which loses blood during menstruation, a process peculiar to the sex, must have an adaptive mechanism in the genitalia which serves to stimulate blood-formation.

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hemoglobin of the blood, but to the relatively diminished exercise enforced upon the patient by the disease.

Simple Primary Anemia.—Under this term we may include all cases, except *chlorosis* and so-called *pernicious anemia*, where there is a diminution of the hemoglobin or red cells, either of one or both at the same time, and where no other disease is present which can cause the conditions. Simple primary anemia, like chlorosis, can be considered a disease of the blood-forming organs; it differs from chlorosis in that it does not affect growing individuals, and is not, therefore, a disease of development. The fact that the condition of the blood in simple primary anemia is identical with that of chlorosis supports this view.

To distinguish simple primary anemias from pernicious anemia, see below.

So-called Pernicious Anemia.—As indicated by its name, this disease is malignant, and is associated with an unfavorable prognosis. This view, however, does not entirely explain the situation, for the severity of the symptoms is a relative affair, and undoubtedly many cases of so-called pernicious anemia have recovered. It would seem more appropriate to consider the characteristic feature of the clinical picture to be not only the inusfficient blood-formation, but also the abnormal destruction of blood-cells, which does not occur in chlorosis and in the so-called simple anemias. This peculiarity must ultimately depend upon some especially severe disturbance of blood-formation, not only quantitative but qualitative, as is evident from the peculiar condition of the bone marrow (megaloblastic), and the presence of megaloblasts in the blood. At the present time an effort is being made to explain pernicious anemia as a megaloblastic degeneration of the bone marrow. The cases to be mentioned further on do not, however, support this view, because megaloblasts were absent in the bone marrow, although all the other clinical signs of pernicious anemia were present. The destructibility of red blood-corpuscles in pernicious anemia is usually indicated clinically by the marked poikilocytosis, the presence of basophilic degeneration of the red cells, and the presence of urobilinuria and icterus. Anatomically, there is a deposit of iron in the liver and kidneys, as shown by Quincke. The granular basophiles and the polychromatophilic changes in the red cells (see p. 637 *et seq.*) which occur so frequently in this disease can no longer, as formerly, be considered as degenerative changes. There are, to be sure, less severe signs of red-cell degeneration in other types of anemia; for instance, in severe chlorosis.

The number of red cells is usually considerably more diminished in pernicious anemia than in chlorosis and simple primary anemia. This diminution is generally more marked than the reduction of the hemoglobin, which probably means that a portion of the hemoglobin of the destroyed blood-cells or the superfluous material for the formation of hemoglobin is made use of by the remaining blood-cells (or is present free in the plasma). On account of this relation between the amount of hemoglobin and the number of red cells, the color index is above 1. This is partly due to the intense coloring and partly to the increase in size of the individual blood-cells. In a case reported by Quincke the number of red blood-cells sank to 143,000 per cubic millimeter.

Micro- and macrocytes and poikilocytes are found in great numbers. It is usually possible to find granular erythrocytes, although in very small numbers; and red cells with polychromatophilic changes are more or less abundant. The presence of megaloblasts in the blood, however, is specially characteristic of pernicious anemia. They are usually very scanty and require patient searching. They are found only exceptionally in large numbers, and then only in the agony of death. Normoblasts may also be found.

The number of leukocytes is usually diminished in pernicious anemia. As the function of the bone marrow is diminished, this decrease is at the expense of the polynuclear leukocytes, so that the lymphocytes seem to be relatively increased. According to Ehrlich and Lazarus, they may constitute 62 per cent. of the total number. Strauss[1] regards the decrease of the polynuclear leukocytes

[1] Strauss and Rohnstein, *Die Blutzusammensetzung in den verschiedenen Anämien*, Berlin, 1901.

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as characteristic of true pernicious anemia, as compared with anemia following carcinoma, in which the percentage of the polynuclear cells is usually greater than that of the mononuclear, and in which there is frequently present a pronounced polynuclear leukocytosis. The latter peculiarity, however, is not always present in the anemia of carcinoma, and may sometimes be observed during a temporary improvement in pernicious anemia. In pernicious anemia the blood-plates are diminished in number.

Individual cases have been observed where, clinically, the megaloblasts have been absent from the blood and the bone marrow has not been found red at the autopsy (aplastic type of pernicious anemia). It is not quite clear yet whether these cases belong properly to megaloblastic pernicious anemia. The etiology of this disease is by no means uniform; it has been known to occur after repeated severe loss of blood, from carcinoma of the stomach, from bothriocephalus, and in absolutely unfathomable causes, so the difficulty of sharply distinguishing it again.

This fact suggests that pernicious anemia may possibly be due to a gradual case in severity of an ordinary secondary anemia. The fact that the blood in

FIG. 236.—Pseudoleukemia (New York City Hospital).

is of megaloblastic type certainly indicates that the ordinary influences, their increased action, exert some specific action on the bone marrow t yet understood.

leukemia.—This is the designation given to those cases of general osis in which, as a result of a general proliferation of the lymphatic here is an enlargement of the spleen and lymphatic glands analogous id in true lymphoid leukemia, and in which the blood shows changes, characteristic changes of lymphoid leukemia. The most striking and change in the blood in these conditions is oligochromemia, although absent. A more important characteristic is the relation of the leuko- umber is normal or slightly increased, but there is a relative increase hocytes—*i. e.*, there is a slight degree of absolute lymphocytosis. tic finding is indicative of what Türk fittingly designates as a sub- ndition of the blood. He differentiates pseudoleukemia from lym- ia, in which the lymphocytosis is so marked that the total number seems to be considerably increased, and an actual lymphemia is

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brought about by the flooding of the blood with lymphocytes. It cannot be said, however, that a sharp boundary exists between pseudoleukemia and lymphoid leukemia, for the former may pass into the latter. When the chief enlargement was in the spleen, we formerly spoke of pseudoleukemia lienalis or anæmic splenica; while if the increase in size mainly affected the lymphatic glands, we referred to the disease as lymphatic pseudoleukemia, malignant lymphoma, Hodgkin's disease, or anæmia lymphatica. It would be wise to drop these names, since they refer to immaterial differences and do not characterize the particular affection. It seems best to the author to classify every case of lymphomatosis, as advised by Türk, according to its acuteness, its anatomic malignancy or benignancy, and the alymphemic, sublymphemic, or lymphemic condition of the blood, and to tabulate it according to the following scheme without giving it a name, since names frequently do more harm than good in pathology.

A Classification of the Lymphomatoses
(From Türk, with slight modification).[1]

Chronic Benign[2] *Growth.*	*Acute growth.*			*Chronic Malignant*[2] *Growth without blood-changes.*
	a. Benign.	*b.* Partly malignant.		
1. Alymphemic (so-called malignant lymphoma.	1. Alymphemic.	Alymphemic.	This also includes chloroma.	1. General (lymphosarcomatosis).
2. Sublymphemic (pseudoleukemia).	2. Sublymphemic.	Sublymphemic.		2. Regional or local lymphosarcoma.
3. Lymphemic (chronic lymphoid leukemia).	3. Lymphemic.	Lymphemic.		

Clinical experience, particularly that of Türk, indicates that all of the clinical conditions in this classification pass over into each other, so that the differences between them are simply those of gradation.

This classification of the lymphomatoses probably also includes the splenomegalies, Banti's disease, and (according to Bleichröder[3]) certain forms of cirrhosis of the liver.

From a practical standpoint, it is still difficult to differentiate the alymphemic lymphomatoses of chronic benign growth, which were previously designated as malignant lymphomata, from similar conditions dependent upon tuberculosis. A rather plausible conception of these tubercular cases assumes that they are combinations of tuberculosis with lymphomatosis or with malignant lymphoma. If this be true, the question is not that of differentiating the so-called malignant lymphoma from tuberculosis, but of discovering some sign by which we may know that the malignant lymphoma has become combined with tuberculosis.

SECONDARY ANEMIAS.

These include all anemias which can be traced definitely to some other definite disease—that is, all the anemic conditions in diseases which produce cachexia, disturbances of nutrition or loss of blood (phthisis, cancer, ulcer of the stomach, ankylostomiasis, bothriocephalus). We cannot sharply separate secondary from primary anemias, as we do not yet definitely know the cause of the disturbance in the blood-making organs which is assumed to be responsible for the primary anemias. The knowledge gained concerning the production of the typical picture of pernicious anemia by the bothriocephalus indicates that perhaps other cases of pernicious anemia may be secondary to other disorders. For instance, the severe anemia so frequent in carcinoma of the stomach has not yet been definitely distinguished from pernicious anemia, although, as a general rule, the megaloblasts usually present in pernicious anemia are absent in the anemia of carcinoma, and a polynuclear leukocytosis is the rule. Nevertheless, in some

[1] *Wien. klin. Woch.*, 1903, p. 39.

[2] The terms benign and malignant are used here in a pathologico-anatomic sense, and refer to aggressive growths which proliferate into and destroy the neighboring tissues.

[3] *Virchow's Archiv*, vol. clxxvii., 1904.

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cases of carcinoma both megaloblasts and leukopenia have been found, just as in pernicious anemia. We have little accurate information as yet in regard to the real condition of the blood in the secondary anemias. A pale appearance does not constitute an anemia at all, as we have already mentioned, for the percentage of hemoglobin and red blood-corpuscles may be normal.

Anemias Following the Loss of Blood.

This type of anemia is the only one which has been accurately studied.

Acute Loss of Blood.—After an acute hemorrhage the blood becomes rapidly diluted, owing to the absorption of lymph and water. Although the hemoglobin and the number of blood-corpuscles may be normal immediately after a hemorrhage, all the characteristics of oligochromemia soon appear, and persist until a normal condition has again been reached. The oligochromemia reaches its maximum within a few hours after a slight hemorrhage, but after severe hemorrhages not until about nine days. Evidence of a regeneration of the blood-corpuscles can be found almost as soon as the plasma begins to be replaced. According to the investigations of Ott and Laache, the number of red blood-corpuscles is normal much sooner than the percentage of hemoglobin. This produces a condition similar to chlorosis. Normoblasts may be found from the second to the third day on. The regeneration is associated with an active posthemorrhagic leukocytosis, chiefly polynuclear. The number of blood-plates is increased and the coagulation hastened after acute losses of blood. Mikulicz considers that major surgical operations will not prove successful if the hemoglobin sinks below 30 per cent. This assumption is rather arbitrary, and the percentage of hemoglobin which would justify an operation might very well be placed a little higher, especially if the narcosis is to be a prolonged one, as narcosis exercises some detrimental influence upon the composition of the blood.

Chronic Loss of Blood (Hemorrhoids, Menorrhagia, Chronic Gastric Ulcer, Carcinoma, Ankylostomiasis, etc.).—Generally speaking, the condition of the blood here is very similar to that several days after an acute loss. The number of red blood-corpuscles is, as a rule, not diminished to so great an extent as the hemoglobin, probably on account of the processes of regeneration. The anemia following chronic loss of blood differs from that caused by acute loss of blood in that the former, provided it is severe and prolonged, presents a poikilocytosis, just as does pernicious anemia. This is in all probability due to the continued loss of plasma and the permanent hydremic nature of the blood-plasma. Another difference is that the leukocytes are not increased in the severe cases of anemia following chronic hemorrhages, and so the picture approaches more and more that of pernicious anemia. The number of polynuclear leukocytes is an approximate measure of the regenerative activity of the bone marrow in the anemias following hemorrhage. The presence of nucleated blood-corpuscles, which generally belong to the normoblastic type, is another index.

ERYTHEMIA OR POLYCYTHEMIA.

Numerous descriptions have recently appeared, the first coming from French authors,[1] of a peculiar affection which consists essentially of a more or less marked increase of the erythrocytes, associated with splenic enlargement, and, further, is characterized clinically by a marked redness or cyanosis of the face, headache, attacks of vomiting, slight albuminuria with casts, and the presence of blood in the sputum, vomitus, and feces. The autopsy findings indicate that in addition to the increased number of erythrocytes there is also a true plethora, since all parts of the body seem to contain an excessive quantity of blood. The heart is normal or somewhat enlarged. In several cases the blood-pressure was found to be increased. The disease appears in middle life and lasts for many years. As yet there is no accurate information as to any change in the bone marrow. Careful

[1] Rendu et Widal, *Bull. et mém. de la soc. méd. des hôp.*, vol. iii., Sér. 1899, p. 528. Further references in Türk's article, *Wien. klin. Woch.*, 1904, Nos. 6 and 7.

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examinations of the blood in these cases show that the number of erythrocytes may be increased to 12,000,000 per cubic millimeter, and the hemoglobin to 200 per cent. The number of leukocytes varies. Türk always found leukocytosis in his cases. In one case myelocytes were observed. The specific gravity of the blood is increased. It is still doubtful whether all cases answering to this description are one and the same affection. Türk believes that, in some of the cases at least, there is a primary hyperplasia of the erythroblastic bone marrow with functional hypertrophy. In favor of this conception is the frequently occurring leukocytosis and the finding of myelocytes. Türk has suggested the name of erythemia or erythrocythemia for this disease, and contrasts it with myeloid leukemia.

LEUKEMIA.

The chief characteristic of leukemia is a considerable and permanent pathologic increase of the mononuclear leukocytes in patients who do not exhibit, aside from the changes in the blood-forming organs (the bone marrow, spleen, and lymph nodes), any other lesions which may be considered primary.

Formerly leukocytosis was separated from leukemia in a very arbitrary way according to the degree of increase of the white blood-corpuscles. If the proportion of white to red cells reached from 1 : 50 to 1 : 20, the affection was called leukemia. This method of differentiation was shown to be useless in the early periods of leukemia and in cases of severe leukocytosis associated with marked anemia. When Ehrlich emphasized the frequency of the eosinophilic leukocyte in leukemic blood, it was at first believed that he had found a reliable point of differentiation between leukemia and leukocytosis, but it did not prove to be a correct one. Since the morphology of leukocytes has been more accurately studied, we have found that the difference between leukemia and leukocytosis is neither one of gradation nor one depending upon the tinctorial properties of the leukocytic granules; but that the true difference lies in the fact that in leukemia the increase is chiefly in the mononuclear type of leukocytes, which are always in excess of the polynuclear type, except in the rare and transitory conditions which will be mentioned later, while in the different varieties of leukocytosis, except in a few cases to be subsequently mentioned (p. 666), the polynuclear cells predominate, as in the normal. Thus, it is usually easy even in early cases to distinguish leukemia.

The other characteristics of the blood in leukemia are as follows: In very advanced cases the blood-drop is peculiarly whitish, clay-colored, or milky, and upon coagulation a thick grayish-white film is deposited above the layer of red blood-corpuscles (the so-called buffy coat containing the white blood-corpuscles). Coagulation is considerably retarded, and in advanced cases the clot is of a soft, jelly-like consistence. The specific gravity of the blood is diminished and the amount of water contained increased. The blood feels peculiarly sticky. The number of red cells is frequently diminished, as is the percentage of hemoglobin. These latter conditions are not constant, especially in the early stages—*e. g.*, leukemic blood is not always pale. Where the hemoglobin is considerably diminished, poikilocytosis and nucleated red blood-corpuscles are found (normo- and megaloblasts). The white cells are usually very considerably increased, not uncommonly to 500,000 per cubic millimeter. All the normal types of leukocytes are present, as well as the pathologic variety described on p. 642.

Leukemia was formerly divided into splenic, myelogenous, lymphatic, and intermediate or mixed types, according to the changes in the organs. To-day the condition of the blood decides to which type a given case belongs.

The changes in the viscera cannot be sharply separated; hence the associated alterations of these organs is of no particular value for properly classifying the disease. For instance, in a lymphatic leukemia, when the enlargement of the lymph glands controls the clinical picture, it is very common to have similar changes in the spleen and bone marrow without the corresponding blood-changes even when the spleen is considerably enlarged. Purely splenic leukemia is equally hard to distinguish. According to modern investigations, the spleen has

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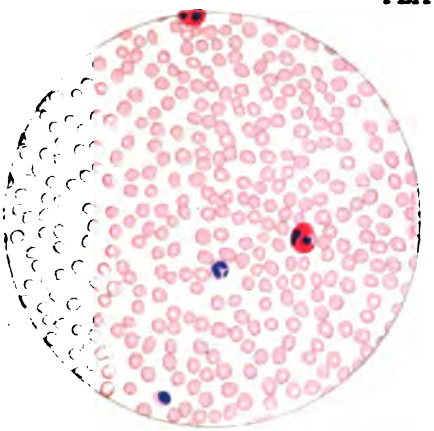
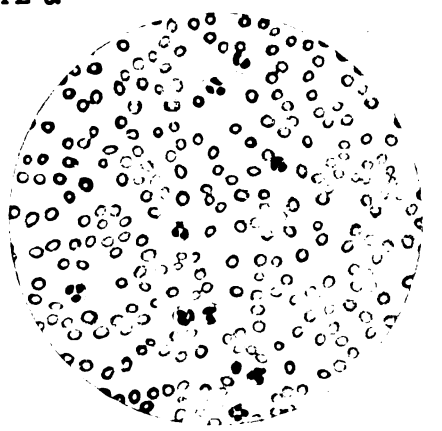
PLATE 8.

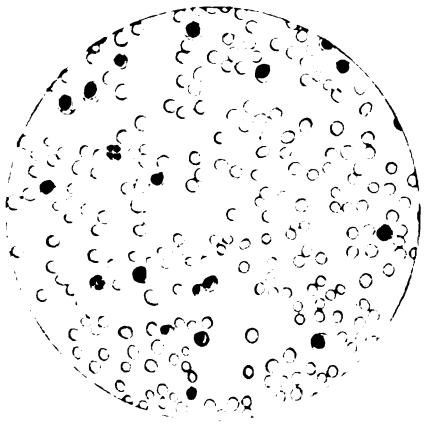


Normal blood. In the field of vision a lymphocyte, a polynuclear cell, and an eosinophile one. The nuclei of all the white cells dark blue, the eosinophile granulations a brilliant red (Rieder).

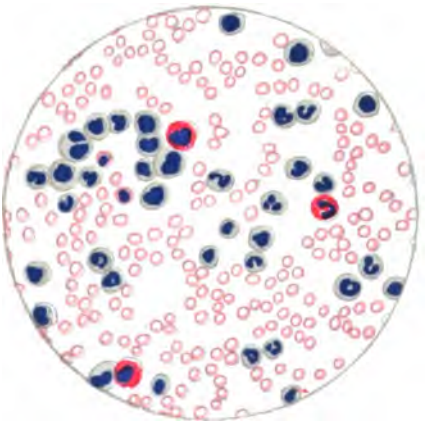


Inflammatory leukocytosis. Marked increase of polynuclear leukocytes. Representation of their neutrophile granules (Rieder).

Heart-disease cells from fresh sputum-preparation (Jakob).



Lymphatic leukemia. Almost all the white blood-corpuscles uninuclear (lymphocytes); most of them very small (Rieder).



Lienomyelogenic leukemia. Most of the white cells are uninuclear; many are strikingly large, with large, plump nucleus. Several eosinophile cells. One nucleus contains red blood-corpuscles (Rieder).

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little or nothing to do with the formation of blood. Besides, in certain leukemias with enormous tumors of the spleen, apparently justifying the term splenic leukemia, not only has the bone marrow been found altered, but the increased leukocytes present in the blood are similar morphologically to those derived from the marrow. Finally it had been demonstrated that the so-called splenic leukemia is also myelogenous; hence the separation of myelogenous leukemia, in the old sense of the word, has no foundation.

Ehrlich and Lazarus recommend subdividing leukemia into two types based upon the blood-examination: lymphatic leukemia or lymphemia and myelogenous leukemia or myelemia. The associated changes in the organs may be described in connection with these terms; for instance, a lymphemia with splenic tumor, or a myelemia with a splenic tumor and swelling of the lymph nodes. The characteristics of the blood in lymphemia and myelemia are represented in Plate 8, Figs. 4 and 5, after Rieder.

The finding of protozoa in the leukocytes in leukemia by Löwit and Mannaberg[1] has not yet been generally confirmed, and for this reason cannot be discussed in detail.

There has recently been a growing tendency among hematologists to give up the sharp distinction between the so-called lymphatic and myelogenous leukemias. This seems to be justified, since, on the one hand, the bone marrow doubtless also contains lymphatic constituents, and consequently forms lymphocytes; and, on the other, the lymphatic glands in myelogenous leukemia have frequently been found undergoing myeloid transformation, so that it must be assumed that they are also implicated in the production of the blood-picture in myelogenous leukemia. The majority of authors, moreover, assume that lymphatic leukemia occurs only when there are also anatomic changes in the bone marrow, and that when these are absent the disease remains in the stage of pseudoleukemia (p. 661). In this connection Pappenheim suggests employing the expressions lymphatic and myelogenous to denote the place of origin, and characterizing the blood-picture as lymphoid or myeloid according to the predominance of lymphocytes or the specific elements of the bone marrow. The question of terminology is a most difficult one, owing to the occurrence of transition forms and to the fact that it is frequently impossible to determine whether certain elements originate in the lymphatic glands or in the bone marrow. For this reason the old subdivision of leukemia into lymphemia (lymphoid or lymphatic leukemia) and myelemia (myelogenous or myeloid leukemia) will be retained here.

LYMPHEMIA (LYMPHATIC LEUKEMIA; LYMPHOID LEUKEMIA).

Chronic lymphatic leukemia is characterized by a more or less marked increase of leukocytes, usually over 100,000, and by a predominance of the lymphocytes, especially the smaller variety. In a few instances, however, the large lymphocytes are more abundant. Both are as normally devoid of granules. The relative number of lymphocytes may reach 99 per cent. of the total number of leukocytes. The other leukocytes are, of course, always diminished relatively as compared to the total number, and they may even be absolutely diminished. The blood usually contains a number of eosinophiles and mast cells, but the myelocytes which are characteristic of *myelemia* are very rarely present. As in *myelemia*, the number of red blood-corpuscles may remain normal for a considerable length of time, but later there is generally a moderate decrease. Nucleated erythrocytes (normoblasts) are found only in small numbers. The percentage of hemoglobin corresponds approximately to the number of erythrocytes. As death approaches the number of lymphocytes may increase considerably. If the condition is complicated by an acute infectious disease, a polynuclear leukocytosis may temporarily take the place of the lymphemia. The lymph

[1] The findings of Mannaberg refer to lymphemia, those of Löwit to both forms of leukemia.

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glands are usually more or less swollen, although the spleen and the liver may also be enlarged and the bone marrow affected. Lymphatic leukemia needs to be studied more accurately. It is much less common than myelemia, and is still little understood hematologically.

The cases of **acute leukemia** are usually grouped with the lymphatic type. This peculiar clinical picture presents the signs of a very acute anemia accompanied by hemorrhagic diathesis. The disease generally runs a very acute and lethal course. Large numbers of cells like large lymphocytes are found in the blood. They are noticeably different from the ordinary large lymphocytes, however, especially on account of the marked lack of chromatin in the nucleus, so that it is possible that they may be undeveloped bone-marrow cells (Nägeli's myeloblasts). In some instances small lymphocytes predominate. As a rule, the number of red blood-corpuscles and the percentage of hemoglobin are considerably diminished. Nucleated red blood-cells are found only in small numbers. Cases of acute leukemia are not common, and but few have been investigated from the standpoint of modern hematology.

Cases of acute myelemia or myeloid leukemia have recently been described (see p. 667), a fact which must be borne in mind in making a differential diagnosis of acute cases.

MYELEMIA (MYELOGENOUS LEUKEMIA; MIXED LEUKEMIA; MYELOID LEUKEMIA).

This variety of leukemia (Plate 8, Fig. 5, and Plate 6, Fig. 2) has been much more extensively studied than lymphemia. The only constant visceral changes are alterations in the bone marrow. The spleen is usually considerably increased in size (compare Fig. 112). The lymph glands and the liver are usually enlarged. The blood-picture is very characteristic, and in itself diagnostic and positive. Ehrlich and Lazarus mention the following anomalous conditions as determinative for the diagnosis:

1. Not only polynuclear cells, but also their early developmental stages, the mononuclear granular leukocytes, must be found in considerable numbers in the blood.

2. All three types of granular cells—the neutrophiles, eosinophiles, and mast cells—but chiefly the first, must be increased.

3. Atypical cells must be found; for instance, dwarf types of the different varieties of white blood-corpuscles, and leukocytes with mitoses.

4. Lastly, the blood must contain nucleated red blood-corpuscles, chiefly of the normoblastic type.

The name "mixed leukemia" is preferred by some authors because the lymphocytes are also increased in this form, and even in those cases in which there are no changes in the lymphatic glands. This fact at once becomes intelligible if we assume with Pappenheim that the bone marrow also forms lymphocytes.

The mononuclear neutrophilic granular cells are the main characteristic of myelemic blood—the "Markzellen," marrow cells or myelocytes, as they were named by Ehrlich. It is not uncommon to find 100,000 myelocytes per cubic millimeter in a case of myelemia. In pronounced cases these cells, as a rule, predominate, although the polynuclear cells are also increased. All other varieties of the white cells appear in insignificant numbers, compared with the myelocytes. In certain stages of the disease, however, the myelocytes may be numerically less than the polynuclear neutrophiles, so that the blood resembles that of a leukocytosis plus myelocytes. This reverse of the typical picture of the blood is observed chiefly with intercurrent febrile diseases, and may easily give rise to diagnostic and prognostic errors, especially as the total number of leukocytes may be considerably diminished. We have seen the blood-picture of a myelemia temporarily change to that of a pronounced leukopenia without any known cause—*i. e.*, without intercurrent disease.

However, in all of these conditions, which probably represent but transitory episodes in leukemia, the absolute number of myelocytes is still considerably

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increased; and even if the blood-picture does resemble a simple neutrophilic leukocytosis, the number of the myelocytes is much more significant than in leukocytosis, where, according to Türk, they hardly ever exceed 1000 per cubic millimeter. In such exceptional cases an absolute increase of the other granular leukocytes, especially the eosinophiles and mast cells, will decide the diagnosis in favor of myelemia. The percentage of eosinophiles and mast cells need not be increased, so that it is necessary to consider the absolute numbers. Myelemia, even in atypical cases, does therefore differ very decidedly from neutrophilic leukocytosis. It also differs from the eosinophilic leukocytosis described upon p. 649. The total number of leukocytes in eosinophilic leukocytosis is only moderately increased; the eosinophiles are exclusively polynuclear; both mast cells and neutrophilic myelocytes are practically absent. The normal figures (see p. 640) will enable one to judge of any increase in the individual types of leukocytes.

In some few cases the neutrophilic granules of the mark cells and of the polynuclear leukocytes are either very slightly developed or even for the time being almost entirely absent. In cases of this sort reported in recent literature,[1] the examinations were made shortly before death. We observed the same condition temporarily in a case which was at the time improving considerably under arsenic.

In leukemic blood which has been kept moist for some time Charcot's crystals (Fig. 211, *e*) appear quite regularly. This condition is probably connected in some way with the abundance of eosinophilic cells in myelemia, Charcot's crystals being supposed to be derived from these cells.

After Jolly demonstrated ameboid movement in mononuclear leukocytes, Ehrlich and Lazarus concluded that myelemia, like leukocytosis, depends upon some chemotactic influence which produces an emigration of undeveloped elements from the bone marrow. As against this view, which places leukemia and leukocytosis in close relationship, we know that myelemic metastases are found in the spleen, in the liver, and in many other organs, a fact which would seem to indicate rather that the changes in the bone marrow are more or less analogous to tumors, and to suggest the idea that the cells are washed out mechanically from the bone marrow.

In reference to the occurrence of acute myelemia, the clinical picture of which practically agrees with that of the chronic form, the reader is referred to the monograph of Fr. Billings and J. A. Capps.[2]

THE SO-CALLED LEUKANEMIA.

Under this name Leube,[3] and subsequently Leube and Arneth,[4] and Luce[5] have described blood-affections in which the phenomena of myelogenous leukemia and pernicious anemia occurred simultaneously (myelocytes, megaloblasts, red metaplasia of the bone marrow). There is no doubt that these cases should be regarded as a species of myelogenous leukemia.

OSMOTIC PRESSURE OR MOLECULAR CONCENTRATION OF THE BLOOD.

In contrast to cryoscopy of the urine, which received a rather adverse criticism upon p. 548 *et seq.*, we possess in cryoscopy of the blood an extremely valuable method of examination. It furnishes important conclusions, particularly in the diagnosis of functional diseases of the kidneys. It cannot be replaced by the determination of the specific gravity of the blood-serum, since to get results analogous to those obtained from the osmotic pressure, the albumin must be removed from the blood-serum, and the complete removal of such a

[1] Ehrlich and Lazarus, "*Die Anämie*," part 1, in *Nothnagel's Spec. Pathologie u. Therapie*, 1898, p. 126.
[2] *Amer. Jour. Med. Sci.*, September, 1903.
[3] Leube, *Deutsch. Klinik*, 1902, No. 42.
[4] Leube and Arneth, *Deutsch. Arch. f. klin. Med.*, vol. lxix.
[5] Luce, *ibid.*, vol. lxxvii, parts 3 and 4.

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large quantity of albumin is possible only after a preceding dilution. Moreover, in view of the small quantities of blood usually at our disposal, the specific gravity must be determined by the rather troublesome capillary-pyknometric method of Hammerschlag (see p. 612).

The nature of the osmotic pressure and its measurement by determinations of the freezing-point, or cryoscopic examination, have been explained on p. 545 *et seq.* In reference to the method of determining the freezing-point, the reader is also referred to what has previously been stated in detail (p. 546 *et seq.*). Only those features which are peculiar to the cryoscopy of the blood will now be discussed.

The determination of the freezing-point of the blood is usually carried out by withdrawing rather a large quantity of blood (usually about 20 c.c.) from the patient by venesection (see p. 610) and allowing it to coagulate. The separation of the serum from the coagulum may be accelerated by loosening the latter from the sides of the vessel. This serum is then employed for the examination. The freezing-point, or osmotic pressure, of the serum is practically equal to that of the plasma, since the fibrin, in consequence of its great molecular weight and of the small percentage present, exerts practically no influence upon the lowering of the freezing-point. Since the osmotic pressure of the serum is equal to that of the plasma, it must also be equal to that of the whole blood or of the defibrinated blood, since the blood-corpuscles must necessarily possess the same osmotic pressure as that of the plasma. It consequently follows that the osmotic pressures of the serum, of the plasma, of the whole blood, and of the defibrinated blood are identical. Nevertheless, we do not recommend the employment of the whole blood, because it can be used only in a coagulated condition, and with this semi-solid mass, even though it be stirred, we have no guarantee that the temperature is absolutely homogeneous. It is only when a sufficient quantity of serum cannot be obtained that the coagulated whole blood may be employed, and then it should be well broken up by the platinum stirrer after its introduction into the freezing-apparatus and before the determination is made. It would be better previously to defibrinate the blood. This may readily be accomplished immediately after its withdrawal by stirring it in a glass vessel for some time with the platinum stirrer belonging to the freezing-apparatus. In every case all of the hemoglobin should be changed into oxyhemoglobin before coagulation and separation of the serum, since the blood obtained by venesection always contains a large quantity of CO_2, and since the variable quantities of CO_2 found combined or free in the blood are dependent upon the degree of oxidation of the hemoglobin, so that this naturally has some influence upon the osmotic pressure. Constant results will be obtained only when the blood employed is saturated with oxygen. This saturation with oxygen is most easily insured by allowing the blood from the cannula to trickle in a thin layer along the sides of the receiving vessel. The contact of the blood with atmospheric air is sufficient to convert all of its hemoglobin into oxyhemoglobin; the area of contact may be increased by catching the blood in a funnel introduced into the mouth of the receiving vessel, and then markedly tilting the funnel so that the blood will spread out in a thin layer upon its inner surface and slowly trickle into the receptacle. Where the blood has been defibrinated, the requisite stirring is sufficient to convert the reduced hemoglobin into oxyhemoglobin. Some investigators also pass oxygen into the blood, but this seems to the writer to be superfluous and too complicated for clinical purposes. It might also be noted that it does not matter whether or not some of the erythrocytes remain in the serum (as frequently happens), since defibrinated blood and serum have the same freezing-point.

The normal freezing-point of the blood is $-0.56°$ C.

The most important result of the determination of the freezing-point of the blood is that it gives definite information in reference to the sufficiency or insufficiency of the elimination of urinary solids by the kidneys. When the method is accurately carried out with due regard to the previously mentioned precautions, among which the writer would lay special stress upon the saturation of the blood with oxygen, an elevation of the freezing-point amounting to

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0.01° to 0.02° must be regarded as pathologic. Considerable elevations of the osmotic pressure of the blood are most frequently encountered in uremic conditions, when freezing-points of −0.65° and −0.7° C. are by no means rare. This evident disturbance of the elimination of urinary solids undoubtedly has some relation to uremia; but the idea that an elevation of the osmotic pressure is constantly present in uremia is incorrect, as is also the converse—that uremic symptoms must be present when the osmotic pressure is increased. From this it follows that uremia is not dependent upon the elevation of the osmotic pressure alone, but must be due to the retention of some special substances.[1] This retention cannot be recognized from the freezing-point of the blood when the retained substance is compensated for, from an osmotic standpoint, by the elimination of other solids. Disregarding the question of uremia, the determination of the freezing-point of the blood furnishes the most important data for the estimation of the work done by the kidneys in a given case of nephritis, and consequently justifies important conclusions in reference to treatment. The author would call special attention to the fact that, while all conclusions in reference to the renal function based upon cryoscopic examination of the urine are inaccurate except in extreme cases, a renal insufficiency may be surely concluded from a distinct elevation of the osmotic pressure of the blood, which, for example, would contra-indicate the extirpation of one of the kidneys.

Very considerable elevations of the osmotic pressure of the blood have also been observed in cardiac cases with broken compensation.[2] Since uremic symptoms, in the ordinary sense of the word, are scarcely ever present in these cases, it follows that uremia does not essentially depend upon the elevation of the osmotic pressure of the blood.

The osmotic pressure of the blood may be lowered in the retention of water as the result of nephritis, in case this retention is not compensated for from an osmotic standpoint by the simultaneous retention of urinary solids, as is usually the case. If the loss of osmotic pressure be more than compensated, cryoscopy is of no value for the recognition of hydremic plethora, and we must then employ what is really the best method for this purpose, the determination of the relative quantity of hemoglobin in the blood.

The lowering of the osmotic pressure of the blood, indicative of the retention of water in febrile diseases, is also of general pathologic interest.[3]

INVESTIGATION OF THE VISCOSITY OR OF THE INTERNAL FRICTION OF THE BLOOD.

From the physico-chemical investigations of Ostwald[4] and the physiologic study of the question by Hüthle,[5] C. Hirsch and C. Beck[6] have worked out a clinical method for determining at the bedside the viscosity or internal resistance of the blood. The apparatus employed is illustrated in Fig. 237. The method depends upon measuring the time required by a known quantity of blood to flow through a capillary tube under a definite pressure. The experiment must be carried out with a constant temperature, since the temperature influences the viscosity. The apparatus is constructed as follows: It consists of the hand-bulb *A*, of the calcium chlorid tube *B*, of the pressure flask *C*, which is protected against heat conduction and radiation by a felt jacket; of the manometer *D*, which is rendered more sensitive by being filled with benzol; of the water bath *E*, and of the actual measuring apparatus *F*. This measuring apparatus consists of a U-shaped tube, *XV*, which is dilated at *G*, below which it becomes capillary, to dilate again at the bend *U*, and pass into the ampulla *M*, in which the closed tube *V* is inserted by means of a ground joint. There is a mark both above and below the dilatation at *G*. The capacity of *G* is about ½ c.c., and the diameters

[1] From the constant findings of Strauss in his analyses of the blood in uremia, these retained substances are nitrogenous, and consist largely of urea.

[2] Landau, *Arch. f. klin. Med.*, vol. lxxviii., 1904. [3] See Landau, *ibid.*

[4] Physico-chemical measurements.

[5] *Pflüger's Archiv*, vol. lxxxii., parts 9 and 10. [6] *Münch. med. Woch.*, 1900, No. 49.

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of the different capillary tubes vary between 0.25 and 0.35 mm. During the course of the experiment this measuring apparatus is held by a support, as indicated in the figure, immersed in a water bath at a temperature of 38° C., and connected by the T-tube *T* and tubing with the pressure flask *C*, the calcium chlorid tube *B*, the hand bulb *A*, and the manometer *D*, as indicated in the illustration. The upper end of the closed tube of the measuring apparatus projects into the air above the level of the water. Before commencing the experiment, the measuring apparatus should be kept for some time in air at a temperature of 38° C., so that we may be certain that its temperature is 38° C. immediately after its immersion in the water. Beside the apparatus is a chronometer which registers fifths of a second. The experiment now proceeds as follows: The connection is broken at *P;* the tube leading to the pressure

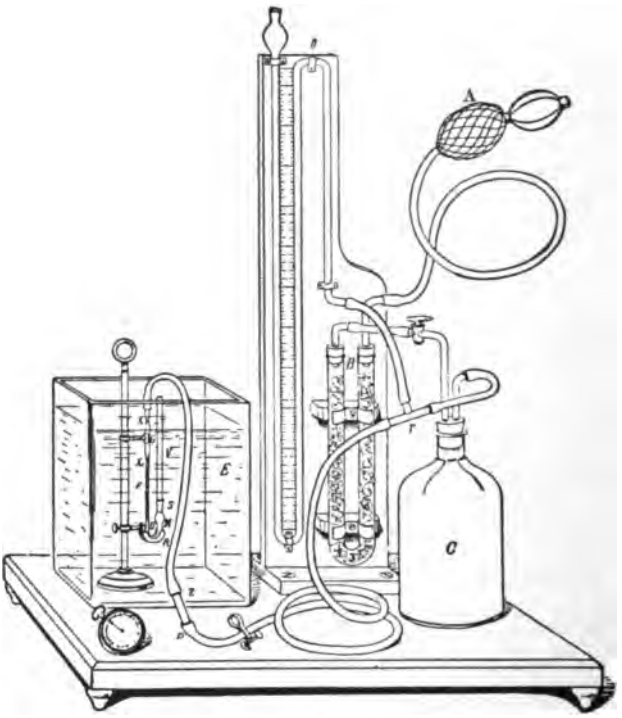

FIG. 237.—Apparatus of C. Hirsch and C. Beck for the determination of the viscosity of the blood.

flask is closed by a stopcock, and the desired pressure of 400 mm. of water, equal to 452 mm. of benzol, is obtained in the pressure flask by means of the hand bulb. The U-shaped lower portion of the measuring apparatus is now filled to the upper end of the ampulla (*M*) with blood, which is obtained fresh from an exposed vein in the arm by means of a pointed and bent glass cannula. The closed tube *V* is now quickly inserted. Suction is then made at *Z* until the blood is just above the mark *X*. The measuring apparatus is now connected with the pressure flask by the glass tube *Z P*. The stopcock is opened, so that the pressure forces the blood downward through the capillary tube. The chronometer is set going as soon as the upper level of the blood-column passes the upper mark *X*, and is stopped at the moment when it reaches the lower mark *X*. The experimenter notes the elapsed time, convinces himself that the pressure has remained constant, and immediately repeats the measure-

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ment. In this manner from two to six measurements may be made with the same blood. The manipulation must be done very quickly, so that it may not be interfered with by coagulation. According to Jakoby,[1] blood may be employed which has been rendered non-coagulable by the addition of leech extract or hirudin. This does not influence the result, while defibrinated blood exhibits a diminished viscosity. The apparatus is cleansed with soda solution and distilled water and kept in a dry place.

The necessary calculation is made by the formula

$$\eta = \eta_1 \frac{st}{s_1 t_1},$$

in which η is the desired coefficient of the internal friction of the blood, s the specific gravity of the blood, η_1 the coefficient of the internal friction of the fluid with which the blood is compared, s_1 the specific gravity of this fluid, and t and t_1 the flowing times of the two fluids. Freshly distilled anilin is selected for comparison, since its specific gravity is so close to that of the blood that it may be assumed to be equal to it. This simplifies the formula so, that

$$\eta = \eta_1 \frac{t}{t_1},$$

η_1, the coefficient of viscosity of anilin as compared with that of water (which serves as the unit in these investigations), is determined once for all, and it consequently follows that, in order to calculate η, we must simply determine the flowing-time of anilin $= t_1$. We then know the value of n_1 and t_1, and, as we have determined t for the blood, the formula gives us the value n; that is, the coefficient of the viscosity of the blood as compared with water. Since interest centers chiefly in relative values, it is still simpler for clinical purposes to select the viscosity of anilin as the unit. This gives us

$$\eta = \frac{t}{t_1}.$$

The most important conclusion arrived at by Hirsch and Beck in a large number of investigations[2] is that it is incorrect to suppose that the cardiac hypertrophy in cases of nephritis is due to an increase in the viscosity of the blood. Still further results may be expected. In all events, the viscosity of the blood is of great clinical interest, since, according to the laws of capillary currents as stated by Poiseuille and Hagenbach, it is an essential factor in the velocity of the blood-stream.

CHEMICAL EXAMINATION OF THE BLOOD.

(Percentage of hemoglobin, compare p. 616. Reaction of the blood, compare p. 613. Removal of the blood for the purpose of chemical examination, compare p. 610.)

IRON TESTS FOR THE BLOOD WITH JOLLES' FERROMETER.

A. Jolles[3] has recently succeeded in devising a method for determining the amount of iron in the blood by means of an instrument called a ferrometer. The method is of some value clinically, for it requires only small amounts (0.05 c.c.) of blood. Its principle is, briefly, as follows: A measured quantity of blood (0.05 c.c.) is removed with a special capillary pipet (such as is used in Gowers'

[1] "Sitzung der medic. Gesellsch. in Göttingen," am 10. Jänner, 1901. Ref. in *Deutsch. med. Woch.*, 1901, No. 8.

[2] See *Arch. f. klin. Med.*, vol. lxix.

[3] *Deutsch. med. Woch.*, 1897, No. 10; *ibid.*, 1898, No. 7, and *Pflüger's Archiv.*, vol. lxv.

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hemoglobin examination). It is reduced to ashes in a platinum dish, 0.1 of acid sulphate of potash is added, and the combination is heated and evaporated to dryness. The residue contains the iron of the blood in the form of an oxid of iron. This is dissolved in 10 c.c. of hot water and 1 c.c. of diluted hydrochloric acid (1 : 3), and 4 c.c. of ammonium sulphocyanid solution (7.5 : 1000) added, so that the total quantity is 15 c.c. This gives a red solution of sulphocyanid of iron, whose composition can be determined colorimetrically by comparing it with a solution containing a known quantity of iron. The latter is prepared in such a way that each cubic centimeter contains exactly 0.00005 of iron.[1] One c.c. of this standard solution is diluted to 10 c.c. with water, and then 1 c.c. of dilute hydrochloric acid and 4 c.c. of ammonium sulphocyanid solution are added just as above. The resulting solution is then, by means of the apparatus pictured in Fig. 238, compared colorimetrically with the sulphocyanid of iron solution derived from the blood. This apparatus consists of two glass cylinders, *C* and *C'*, of exactly equal caliber, *C* containing accurately 15 c.c., and *C'* about 16 c.c. Both cylinders are subdivided into 0.1 c.c., and both are closed at the bottom with glass plates, through which light is reflected from the plaster-of-Paris reflector, *R*, in the direction of their axes. The sulphocyanid of iron solution prepared from the blood is exactly 15 c.c., and so just fills the cylinder *C*, which is then covered with a glass disk, care being taken to avoid the formation of air bubbles. Fifteen cubic centimeters of the sulphocyanid of iron solution (prepared from the stock solution) are placed in cylinder *C'* for comparison. To avoid the development of a meniscus, which will disturb the reading, a floating stopper, made of aluminum and closed at both ends with parallel glass plates, is placed on the surface of the fluid in cylinder *C'*, avoiding air bubbles. A flat fluid surface is thus obtained. This contrivance always displaces a slight amount of fluid, which is the reason why cylinder *C'* must be made somewhat taller than *C*. Cylinder *C'* is provided with a lateral escape stopcock, *H*. Both cylinders can be easily removed from the base of the instrument for cleansing or filling. The tin jacket surrounding the cylinders shuts out any lateral illumination. The colorimetric comparison is made as follows: Both cylinders are filled as just described, and then the fluid in *C'* is allowed to escape through the stopcock *H* into the vessel *A* until the contents of both tubes, as seen from above by transmitted light, present exactly the same shade. When this point is reached, cylinder *C'* is removed and the level of the fluid noted. If the amount of fluid remaining in *C'* to the base of the floating cork were exactly 15 c.c., the amount of blood used (0.05 c.c.) would contain 0.00005 of iron—*i. e.*, 1 liter of blood would contain 1 gm. of iron. If, however, when the color in the two cylinders is alike, *C'* contains only 7.5 c.c. of fluid, the amount of iron contained is only half as much—*i. e.*, 0.5 gm. per liter of blood. A table to calculate the percentage of iron contained in the blood has been prepared by Jolles, and is included with the instrument. It takes about ten to fifteen minutes to calculate the amount of iron with Jolles' ferrometer.

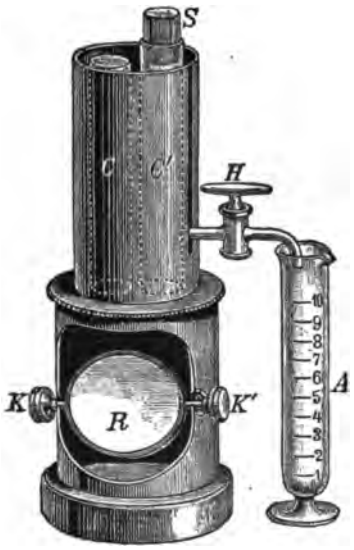

FIG. 238.—Jolles' ferrometer.

The same maker (Reichert) has recently placed upon the market an instrument which may be used either as a Fleischl-Miescher hemometer or as a Jolles'

[1] For the exact steps see above reference.

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ferrometer, since the intensity of the color of the sulphocyanid of iron solution is estimated by means of the glass wedge of the hemometer.[1]

Jolles believed at first that his method was an accurate means of determining the amount of hemoglobin indirectly, for he assumed that hemoglobin contained 0.42 per cent. of iron, and that all the iron present was contained in the hemoglobin. But in more recent communications[2] he has abandoned this theory, as his investigations have led him to conclude that the blood contains considerable quantities of iron, probably in the shape of nuclein, outside of the hemoglobin. This idea corresponds to the views of Biernatzki and Jellinek. Moreover, it is likely that hemoglobin itself does not always contain a constant amount of iron. The great variations (which have been reported in literature) as the results of analyses of hemoglobin crystals in one and the same animal species would seem to suggest this.

Although these statements might give rise to the thought that the estimation of the iron in the blood might acquire a clinical significance independent of that of the estimation of the hemoglobin, the investigations of Krüss[3] and Schwenkenbecher[4] would make this seem very doubtful, since these authors reached the conclusion that the double sulphocyanid of iron producing the red color in Jolles' method becomes so markedly dissociated by dilution that the red color decreases in intensity more rapidly than does the quantity of iron in the solution. In other words, iron cannot be accurately estimated by this colorimetric method.

THE BLOOD IN CARBON DIOXID POISONING.

In marked cases of carbon dioxid poisoning the naked eye can probably appreciate the change of color in the blood. It is noticeably bright red, and almost fails to exhibit the difference between venous and arterial blood. The presence of carbon dioxid in the blood is demonstrated usually by means of the spectroscope. If a few drops of blood containing carbon dioxid are diluted with water, the mixture will show in the spectroscope two bands, between the green and yellow, very much like those of oxyhemoglobin (Fig. 167, 1). They are, however, very slightly displaced toward the violet end of the spectrum. They differ from the oxyhemoglobin bands by not disappearing upon the addition of ammonium sulphid, whereas the oxyhemoglobin bands are replaced by a simple band of reduced hemoglobin (Fig. 167, 2). Oxyhemoglobin bands disappear very slowly after adding ammonium sulphid to the blood, and may be temporarily reproduced if the liquid is shaken with air, owing to the concentrated action of the oxygen.

Another test for carbon dioxid in the blood consists in adding a little 10 per cent. caustic soda or potash solution to the blood contained in a porcelain dish, and then gently warming. If carbon dioxid is present in any appreciable quantity, the mixture will become very brilliant red, while normal blood will turn to a dirty greenish brown.

It is advisable to perform both spectroscopic and chemical tests and compare them with the normal blood for a control, because when only a slight amount of carbon dioxid is present in the blood, quantitative differences are important in forming an opinion.

[1] S. Jolles, *Berlin. klin. Woch.*, 1899, No. 44, p. 965. [2] *Loc. cit.*

[3] *Colorimetrie und quantitative Spectralanalyse in ihrer Anwendung auf die Chemie*, Hamburg and Leipzig, 1891, p. 176.

[4] *Arch. f. klin. Med.*, vol. lxxv., parts 3–5.

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We must not overrate the diagnostic value of these investigations. The human body reacts so intensely to carbon dioxid gas that symptoms of poisoning may be very pronounced without its being possible to demonstrate the presence of this poison in the blood either chemically or spectroscopically.

THE BLOOD IN METHEMOGLOBINEMIA.

The blood contains methemoglobin in various types of poisoning, especially from chlorate of potash and antifebrin. Its presence is demonstrated by means of the spectroscope after sufficiently diluting the blood with water (see Fig. 167, 3).

THE BLOOD IN HYDROGEN SULPHID POISONING.

In severe cases the color may be a dirty green, and may show the characteristic bands of sulphohemoglobin besides the oxyhemoglobin bands. The former resemble the bands of methemoglobin, but are situated a little more toward the violet end of the spectrum (Fig. 167, 3).

THE BLOOD IN HEMOGLOBINURIA.

Hemoglobinuria may appear independently as the so-called periodic hemoglobinuria (Lichtheim), or especially in certain kinds of poisoning and after burns. It is always produced by the red blood-corpuscles becoming dissolved within the vascular system. Hemoglobinuria is therefore always associated with hemoglobinemia or methemoglobinemia. Free hemoglobin and methemoglobin can be readily demonstrated in the blood by allowing several cubic centimeters of blood to coagulate. Under normal conditions the serum will only be stained slightly yellow (luteïn), provided that the blood-clot has not been interfered with mechanically. In hemoglobinuria, on the other hand, the serum will be more or less of a ruby red, owing to the presence of hemoglobin in solution; or brown, if methemoglobin is present. The serum, however, must be absolutely clear, for if it is cloudy the color may be due to an admixture of blood-corpuscles with the serum, from some disturbance during coagulation. In doubtful cases the microscope will decide the question. The stained serum will give the characteristic bands of oxy- or methemoglobin, one or both, in the spectroscope (Fig. 167). In periodic hemoglobinuria the blood shows during the attack a peculiarity first described by Hayem, and subsequently corroborated by Chvostek.[1] It coagulates very rapidly, but the clot dissolves again after some little time. In fresh preparations disintegrated, deformed, or decolorized blood-corpuscles may be found under the microscope (shadows, p. 638).

PRESENCE OF URIC ACID IN THE BLOOD.

Physiologically, the blood does not contain any appreciable quantity of uric acid. In chronic nephritis and acute articular rheumatism mere traces only have been found; but in gout, at least in the early stage of

[1] *Ueber das Wesen der paroxysmalen Haemoglobinuria*, F. Deuticke, 1894.

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the attack, the amount of uric acid contained in the blood is so considerable that Garrod devised a comparatively simple method, the so-called thread test, to demonstrate its presence.

According to Garrod, 30 to 35 c.c. of blood are allowed to coagulate; 10 c.c. of the serum which accumulates during the next few hours over the clot is mixed with 100 c.c. of acetic acid. A fine linen thread[1] is placed in the mixture, which is then covered so as to prevent evaporation, and allowed to stand.

If the blood contains at least 0.0025 per cent. of uric acid, characteristic crystals (Fig. 186) will appear on the thread in the course of one or two days. In doubtful cases the murexid test (p. 556) may be applied to these crystals.

Garrod indirectly demonstrates an increase of uric acid in the blood in gout by performing this test with the serum of an artificially produced blister.

WIDAL'S SERUM TEST IN TYPHOID FEVER.

This test has its origin in the fact that the blood-serum of patients who are convalescing from typhoid, or of animals immunized to typhoid, according to the investigations of Grüber, Durham, and Pfeiffer, influences the typhoid bacilli in a manner in such a way that the latter lose their motility and form conglomerations which may be recognized microscopically. A similar phenomenon is observed when cholera-immune sera are added to cultures of cholera bacilli. This phenomenon has been attributed to the presence of peculiar hypothetic substances in the immunized serum, which have been called agglutinin or glabrificin by Grüber, and bacteriolysin by Pfeiffer. These substances are not identical with the bactericidal and immunizing substances of immune serum.

Based upon this observation, Widal devised a peculiar diagnostic method for recognizing typhoid, for he noticed that this peculiarity is shown not only by the serum of convalescents, but also by the serum taken during the disease, and quite early in the attack. He proposed[2] two methods for testing the serum in doubt of the questionable typhoid cases, the so-called microscopic and macroscopic methods. To prevent evaporation, a few drops of freshly drawn blood are placed in a closed tube of narrow caliber, in which the blood may be preserved for use. The serum which separates by spontaneous coagulation is employed.

In beginning Widal demonstrated the reaction microscopically by simply adding a drop of the serum to a drop of typhoid broth culture, of not more than two to twenty-four hours' growth, upon a slide. Under the microscope the bacilli in the broth culture must be very active. If the reaction is positive, the bacilli will cease to move within from a few minutes to half an hour and become agglutinated, while the intervening fluid will be free from bacilli. The second method depends upon the macroscopic appearance of a typhoid broth culture to which the serum of the patient in question has been added. The controlled culture is evenly clouded; but a culture to which a moderate amount of typhoid serum has been added will clear up, on account of the agglutinated bacilli settling to the bottom of the tube.

Experience, however, has been demonstrated in the Bern Clinic that this macroscopic reaction is not nearly so reliable as the microscopic test.

[1] It is better not to use a thread, but to separate from it one of the elementary fibers of which it is composed.

[2] *La Semaine Méd.*, 1896, No. 33, p. 259; also numerous tests of the method in the same year of this periodical; also tests by the Germans: Stern, *Centralbl.* 1896, No. 49; Breuer-Lichtheim, *Berlin. klin. Woch.*, 1896, Nos. 47 and 48. F. Widal and M. A. Sicard, *Ann. de l'institut Pasteur*, 1897, vol. xi., No. 5.

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To obtain reliable results, the microscopic reaction had to be modified, since it was found necessary to take into account the dilution of the serum. For it was soon discovered that in low dilutions the serum of some people not suffering from typhoid had a moderate power of agglutinating typhoid cultures. It was therefore found necessary to determine that a certain very small quantity of the serum would exert this action in order to consider the test positive for the diagnosis of typhoid.

Widal's reaction is performed quantitatively in the Bern Clinic as follows, a convenient and reliable method: In 4 or 5 glass staining dishes we place 10, 20, 50, 70 or 100 drops respectively of a typhoid culture not over twelve or fourteen hours old, by means of a glass pipet with a long, fine point. But 1 drop is allowed to escape at a time. The bacilli of the culture must be known to be very active. A drop of the suspected serum is added to each dish from the same pipet (after it has been washed). The culture and the serum are now thoroughly mixed in the dish with a platinum wire. The serum dilution is therefore 1: 10, 1: 20, 1: 50, 1: 70, etc. A microscopic preparation from each of these mixtures and from the typhoid culture for the purpose of control are examined immediately and after a known interval with an oil-immersion lens. The more concentrated specimens are, of course, examined first. In some cases of typhoid the agglutinating action of the serum may be so marked that even with a dilution of 1: 100 the typhoid bacilli will lose their power of motion and will adhere in clumps immediately after the addition of the serum. When the agglutination is less vigorous, the reaction may not be manifest for a considerable time, or it may occur only in the more concentrated dilutions. Before an absolute diagnosis of typhoid fever is made from the serum action alone, agglutination should take place within an hour with a dilution of the serum of 1 : 50. The sooner clumping occurs, the safer one is in expressing an opinion. It is useless to extend observations beyond an hour, because by using the more concentrated solutions we can get sufficient information regarding the less pronounced agglutination power in less than an hour. If the period of observation is limited in this way, the microscopic preparations may be left to themselves without fear of having them dry up. In case of any doubt, it is always easy to make fresh slides from the staining dishes.

Different typhoid cultures vary in their susceptibility to clumping, so that each observer should make himself familiar with the peculiarities of his own cultures. This explains variable results reported.

In this connection the author will record what he believes to be a new observation, made in his clinic during an epidemic of typhoid fever. Early in the disease the serum of the patients produced more marked agglutination in weaker than in stronger concentrations; but this condition was reversed during the course of the disease. This indicates that, in addition to the agglutinative effect of the serum, there is a counteraction against agglutination early in the disease, which may be overcome by the more marked dilutions. Since agglutination is doubtless indicative of an injury to the typhoid bacilli, this phenomenon is to be regarded as an expression of a favorable influence of the serum upon the growth of the bacilli, which disappears during the course of the disease, and which may be overcome by the more marked dilutions. Further investigations are now under way in order to determine whether these differences between the effects of concentrated and dilute serum mixtures are of value from a prognostic standpoint. At all events, the observation is of practical importance, since it demonstrates that a negative result should never be assumed simply because agglutination is not obtained by a concentrated serum, but that an opinion should be withheld until the test has been made with a much diluted serum. It also shows the necessity of trying the different concentrations in every case.

In order to have fresh typhoid cultures always at hand, it is best to transfer the glycerin-agar cultures every eight or fourteen days, as otherwise the cultures will degenerate and die off. A bouillon culture is prepared from the water of condensation of the agar culture twelve to fourteen hours before performing the test and placed in an incubator. The water of condensation always

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contains very active bacilli. Stock cultures, after being grown for a little while, are best preserved at the temperature of the room. Authentic typhoid cultures are best obtained from the spleen of a fresh typhoid corpse (with the ordinary precaution to avoid contamination—*i. e.*, singeing the surface).

Attempts have recently been made to substitute for the original Widal reaction analogous reactions with dead preserved typhoid bacilli, and also reactions of precipitation with chemical extracts of cultures of typhoid bacilli. Until the results obtained are shown to be as reliable as those obtained from the original procedure, they will not be described in detail in this work. These modifications would certainly have the advantage of rendering the procedure more accessible to the practising physician.

EXAMINATION OF THE MOUTH AND PHARYNX.

INSPECTION is the most important method for examining the mouth and pharynx. For proper inspection the patient should open his mouth as wide as possible, and then retract the tongue from the place to be examined. The pharynx and soft palate are usually seen best if the base of the protruded tongue is depressed with a spatula, with the handle of a spoon, or with the finger of one hand, while the examiner steadies the patient's head at the same time with his other hand. Care must be taken not to apply the depressor too far back, for if the posterior portion of the base of the tongue or the pharynx is touched, gagging results and renders the examination difficult. The posterior wall of the pharynx can be seen better when the patient says "Ah," and so elevates the soft palate. It is sometimes very difficult indeed to examine patients' mouths if their consciousness is at all disturbed, and it is almost impossible to examine the mouths of unruly children. Sometimes closing the nostrils will make such patients open the mouth, while at other times we have to forcibly separate the jaws with a spoon, a depressor, or a mouth gag. With children, whether we employ the aid of a depressor or not, we should look quickly the moment the child opens his mouth to cry.

Palpation with the finger often assists in determining the condition of portions of the mouth which are situated farther back. Palpation of the nasopharynx or of the entrance to the esophagus to hunt for foreign bodies, retropharyngeal abscesses, or adenoids must be done very quickly, because it is very disagreeable to the patient and can be borne but a short time.[1] Unruly patients and children are apt to bite the examiner's finger unless the cheek is pressed in between the patient's teeth with the free hand. This is a better method than to employ appliances for protecting the fingers. The forefinger is better adapted for palpating adults, and the little finger for small children. The introduced finger is hooked upward or downward, according to the region to be

[1] [The application of a solution of cocain (2 to 4 per cent.) to the back of the pharynx will deaden the sensibility of the parts and allow a more careful digital examination.—ED.]

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explored. The entire pharyngeal cavity can be palpated from the top of the larynx below to the nasopharyngeal space above.

Lips.—The examiner should notice their color (pallor, cyanosis),

FIG. 239.

FIG. 240.

FIG. 239.—Hutchinson's teeth in hereditary syphilis: Two upper middle incisors (second dentition) with deep transverse and longitudinal grooves; also concave edge of middle incisors. The length of the teeth is normal, but the width is diminished; hence, broad interspace in middle.

FIG. 240.—Upper middle incisors (second dentition) immediately after eruption; also four lower incisors. Lower surface of upper incisors is rough, due to prominent points of dentin. Upper teeth short and directed away from each other, forming a broad interspace. On the four lower teeth are numerous excrescences, due to deficient formation of enamel. Base of excrescences everywhere at same level.

the presence of aphthous patches, mucous patches, fissures (rhagades), or their scars, which leave diverging folds at the corners of the mouth (children with hereditary syphilis), and herpes labialis.

FIG. 241.—Hutchinson's teeth: The upper middle incisors (second dentition) and all four lower incisors are characteristic enough. The photograph was taken from a colored child, six years of age, who had been under Dr. McConnell's observation at the Vanderbilt Clinic for a year. She has sabre shins, and when first brought to the clinic presented a rash. An older sister has similar teeth. The mother has had two miscarriages since the birth of her last child. The original of this photograph has improved upon biniodid of mercury.

Teeth.—The condition and the time of appearance of the first and second sets are important in childhood.[1] In adults we should notice the state of preservation of the teeth. Poor teeth not infre-

[1] [Note irregular dentition as indicating nasal deformity and former adenoids.—ED.]

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ly cause digestive disturbances. They are quite commonly due ne general disturbance. Special attention should be paid to

42.—All four upper incisors approach the type of Hutchinson's teeth. The lower incisors ically normal. In this case the upper middle incisors are not characteristically peg- ut the groove upon the cutting edge is a significant feature. No other facts relating se are known. It is probable that the teeth should be classified as Hutchinson's teeth.

d "rachitic teeth." They are characterized chiefly by transverse gitudinal grooves with uneven enamel, which rapidly wears off unt of its irregular distribution. These changes are most marked

3.—Irregular teeth simulating Hutchinson's teeth in a patient at the New York City who exhibited at the time this photograph was taken a well-marked secondary syph- ion.

cisors. In hereditary syphilis the teeth are often deformed very in rachitis. Some of the teeth formations which are observed lisease are by no means characteristic of syphilis alone, but occur

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FIG. 244.—Irregular teeth in a young boy with a high-arched palate. Although the two upper middle incisors are slightly wedge-shaped, there is not sufficient ground for considering them of the Hutchinson type. The irregularities are probably due to an inflammation of the gums during the second dentition (Dr. W. L. Stowell, Randall's Island Hospital).

also in rachitis and other general disturbances, or even when the teeth buds have been damaged by stomatitis. Hutchinson considers the type

FIG. 245.—Irregular teeth, not Hutchinson's teeth (New York City Hospital).

of teeth shown in Figs. 239, 240 as pathognomonic of hereditary syphilis.[1] Other authors consider the concave dents (notches) in the lower margin

[1] "Hutchinson's Teeth."—These were first described in the *Transactions of the Pathologic Society of London*, July, 1858. In the original description he says: "There is a peculiar condition of the teeth resulting from hereditary syphilis, the most frequent conditions being the following:

"(*a*) *Smallness.*—They are small, stand apart, are rounded and peggy.

"(*b*) *Notching.*—They usually exhibit at the border a broad, shallow notch (sometimes two or three serrations).

"(*c*) *Color.*—They have a dirty-grayish surface without polish, and are rarely smooth.

"(*d*) *Wearing Down.*—They are deficient in enamel and are soft, and therefore liable to premature wearing down.

"(*e*) These signs almost exclusively apply to the incisors and canines."—[ED.]

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of the upper middle incisors the characteristic feature (Fig. 239, *a*). The term "Hutchinsonian teeth" should be applied more especially to those showing this peculiarity. These are observed chiefly in the second dentition. After the twenty-fifth year this concavity is usually worn away.

It is important to be familiar with the normal periods of the appearance of teeth, so as to be able to recognize anomalies in the formation of teeth and to attach a proper significance to the symptoms oftentimes connected with their eruption in early childhood.

A. Vogel makes the following statements (Fig. 246):

FIRST DENTITION.

First Group: The two lower middle incisors appear almost simultaneously between the seventh and the ninth month.

Interval of three to nine weeks.

Second Group: The four upper incisors appear between the eighth and the ninth month, and follow one another rapidly within a few weeks, first the middle and then the lateral ones.

Interval of six to twelve weeks.

Third Group: Six teeth appear almost at the same time between the twelfth

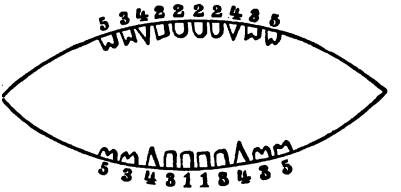

FIG. 246.—Sequences of teeth with first dentition (A. Vogel).

and the fifteenth month. These are the first four molars and the two lower lateral incisors. Usually the anterior molars of the upper jaw appear first, then the lower lateral incisors, and finally the anterior lower molars.

Interval to the eighteenth month.

Fourth Group: The four canines appear between the eighteenth and the twenty-fourth month.

Interval of two to three months.

Fifth Group: The four second molars appear between the twentieth and the thirtieth month. This ends the first dentition, and the child then has the twenty "milk teeth."

SECOND DENTITION.

During this period the milk teeth are supplanted by permanent teeth, and the three posterior molars in each jaw appear. The latter are not present during first dentition. A permanent set of thirty-two teeth then takes the place of the twenty milk teeth. Second dentition begins in the fifth or sixth year of life with the appearance of the first molar teeth in each side of the jaw. The milk teeth now begin to fall out in the same order that they appeared. Each tooth as it falls out is supplanted immediately, or very soon, by the corresponding second tooth. The second molars appear during the twelfth year, and the third molars, or wisdom teeth, appear from the sixteenth to the twenty-fourth year, or sometimes still later.

Gums.—The gums are swollen and bleed easily in acute and chronic mercury-poisoning, after the use of iodids and bromids, and in scurvy.

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The bluish-gray "lead line" on the gums, due to a deposit of sulphid of lead in the mucous membrane, is of great diagnostic importance in chronic lead-poisoning. This line should not be confused with a discoloration of the teeth themselves at the junction of the gums. The latter is not uncommon in careless individuals and those who smoke a great deal. This discoloration is visible up to the margin of the gum, and may simulate the blue appearance of a lead line very accurately. It is very easy to tell these conditions apart by pushing the corner of a stiff piece of paper beneath the margin of the gum. A true lead line will become more distinct than ever, but the other will disappear entirely. A bluish discoloration sometimes appears at the margin of the gums, due to venous congestion, not only in general circulatory disturbances,

FIG. 247.—Lead line: Note the interrupted fringe-like line just at the edge of the gum (photographed from a water-color drawing; kindness of Dr. R. C. Cabot).

but in some local inflammations of the gums, and may lead to error. Employing the piece of paper as above directed makes the gum anemic by the pressure exerted and renders the distinction easy.

Tongue.—The way the tongue is protruded is sometimes more or less characteristic. Very ill or stupefied patients push it out with a trembling, and leave it out until told to withdraw it. Injuries or scars on the tongue from biting are of great diagnostic importance in epilepsy.

We should examine the tongue of patients with bulbar symptoms very closely for any noticeable atrophy. This atrophy may become very marked indeed in progressive bulbar paralysis, and the tongue may exhibit pronounced fibrillary contractions. Some healthy individuals present a slight fibrillary twitching of the tongue.

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The so-called "coat" of the tongue is of some diagnostic importance. The tongue is covered in all dyspeptic conditions and in fever. This coating is usually associated with some disturbance in the appetite. However, some healthy individuals with a good appetite always have a coated tongue. Acute and chronic gastric catarrh is almost always associated with a coated tongue; but in ulcer of the stomach the tongue is often not coated and there is no disturbance in the appetite. The cause of the coat is an overgrowth of epithelium of the mucous membrane of the tongue, but it is not yet fully understood. It is very often explained as being due to the fact that in people who do not eat, the lingual epithelium is not removed in a normal way by friction. This explanation, however, does not correspond to clinical experience. Probably some trophic disturbance of the mucous membrane of the tongue is produced by the condition of the digestive organs.

Frequently in patients with high fever the tongue is not only coated, but dry, on account of the diminished secretion of saliva. Small fissures of the mucous membrane are rather common in these cases and may cause some bleeding. The blood-crusts which form in this way, combined with the dried brownish epithelium of the tongue, produce a dark-brown or blackish coating. This always indicates that the patient's condition is serious. It may be found on the lips at the same time.

The coating of the tongue is often stained by certain foodstuffs, drinks or medicines (milk, red wine, coffee, cocoa, chocolate, licorice, fruit, tobacco, etc.).

The presence of fungi must not be confused with an ordinary coating of the tongue. Fungi may be found on the tongue, as well as elsewhere on the mucous membrane of the mouth, and occur in the form of large thick patches. Fresh patches are easily recognized by their snow-white color and round margins. The older patches often lose this white color, and are of a peculiar dirty-gray appearance. The situation of these mycotic patches on the margin of the tongue and their abundance in other parts of the mouth distinguish them from a simple coated tongue. Fig. 223 represents the microscopic appearance of thrush.

The tongue should be examined for *aphthous patches* and *syphilitic sores*. The peculiar circular thickenings of the epithelium, with slight inflammatory signs, so-called "*leukoplacia buccalis*," must not be confounded with either of them. Leukoplacia, sometimes called "*psoriasis linguæ*," is much more extensive. Its nature is not as yet well known. Whitish, thickened spots of epithelium, produced by the irritation of bad teeth or of too much smoking, may resemble very closely syphilitic mucous patches.

The nature of the so-called *black hairy tongue* is utterly unknown.

The erythematous tongue of scarlet fever, with its red and swollen papillæ, is very characteristic. It is familiarly known as the "*strawberry tongue.*"

[Virchow's smooth atrophy of the base of the tongue may be of some value in diagnosing late syphilis. It consists of an atrophy of

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the lingual tonsillar tissue, and can be appreciated by palpation better than by inspection.—Ed.]

Soft Palate; Tonsils; Pharynx.—The various kinds of acute angina are to be considered in this connection (*angina simplex, lacunaris, necrotica, phlegmonosa,* and *diphtheritica*); furthermore, the different varieties of chronic pharyngitis (*pharyngitis sicca, granulosa,* etc.). (See text-books of Special Pathology.)

Examination for Diphtheria Bacilli.—In doubtful diphtheric membranes only a microscopic demonstration of Löffler's bacilli (see Fig. 248) will prove the existence of true diphtheria. These bacilli are sometimes found in the membrane of true diphtheria in great numbers. Dry preparations may be made from the membrane in the same manner as described for the sputum, and then stained with the ordinary anilin stains, preferably by Gram's method. But cultures are generally necessary. According to the investigations of the Bacteriologic Institute at Bern,[1] Löffler's horse-serum and glycerin-agar (7 per cent. glycerin) tubes are the best media for growing diphtheria bacilli.

Fig. 248.—Diphtheria bacilli (× 1000) (after Weichselbaum. Culture).

The media can be inoculated from the surface of the affected region of the pharynx by means of a sterile platinum loop or swab. The round colonies of diphtheria bacilli, transparent at first, gradually become opaque. They may be seen after the tube has remained in an incubator about twelve hours. The colonies must be identified by preparing and examining microscopic slides, as macroscopically they may be confounded with those of other bacteria, especially streptococci. It is diagnostically important to remember that diphtheria bacilli are generally present in the pharynx in cases of laryngeal diphtheria, even when the pharynx itself is not visibly affected.

THE PRACTICAL VALUE OF THE BACTERIOLOGIC EXAMINATION FOR DIPHTHERIA.

Diphtheria may be recognized clinically with a considerable degree of accuracy in the vast majority of cases. At times, however, demonstrating the presence of diphtheria bacilli may be very important.[2] Where there are clinical reasons for making a diagnosis of diphtheria (epidemics), we should not rely absolutely upon the result of a single negative bacteriologic examination (even when Löffler's modified serum is used).

Negative results may easily be due to accidental conditions in inoculating the

[1] G. Michel, "Das Wachsthum der Diphtheriebacillen auf verschiedenem Sera und Glycerinagar," J. D., Bern, 1897 (*Centralbl. f. Bakteriologie*). The author concludes that Löffler's modified horse serum is the best medium of the five he examined, and that glycerin-agar is next. Löffler's ox serum and the pure ox or pure horse serum without any addition are considerably inferior. These results suggest that many statements in literature regarding false diphtheria are doubtful. Löffler's modified horse serum frequently gives a negative result, whereas from the same case diphtheria bacilli grew on one or the other inferior medium. Perhaps the most logic conclusion is that one negative examination does not prove anything.

[2] "Deucher (Bern), Zur Klin. Diagnose der Diphtherie," *Correspondenzbl. f. Schweizer Aerzte*, 1895, No. 16, p. 485.

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medium ; or, in case the specimen is sent to a central laboratory, the bacilli may have become less virile from drying. Again, other bacteria may easily outgrow the diphtheric in the culture tube. The use of antiseptics (gargling, swabbing) in diphtheria is often responsible for a negative culture. Finally, for unknown reasons, certain diphtheria bacilli (discovered in the Bacteriologic Institute at Bern) refuse to grow well upon the media which are generally supposed to be best. Control examples have proved beyond doubt that these factors may influence the result of bacteriologic examination. A physician may err if he relies absolutely upon the bacteriologic examination, especially if attempted only once. Since the introduction of serum treatment this is a serious matter, from a therapeutic standpoint, and it may be still more disastrous from a prophylactic one.

A positive result must also be carefully interpreted, for we know that virulent diphtheria bacilli are not infrequently found in perfectly healthy people, in ordinary cases of mild coryza and rhinitis, and in cases of pharyngitis which do not even produce a rise in temperature. It is hardly justifiable to administer antitoxic serum to these cases, because, as is well known, the serum does not remove the bacilli. It is perfectly possible that at some later date a severe case of diphtheria may develop in the individual. A logical and practical deduction is that the serum treatment should not be confined to cases where the bacteriologic examination is positive, but should be employed whenever the case presents a perfect clinical picture of diphtheria, or when there are severe symptoms and diphtheria bacilli are present in numbers, although the case may not resemble diphtheria clinically.

The bacterial examination would be of much greater clinical value if in every case smears could be made directly from the pharynx, then fixed, stained, and examined under the microscope. The chief error in the modern culture method is that mere chance in inoculation may alter the numeric relation of the bacteria, and some species may be entirely suppressed. In one case the diphtheria bacilli may be concealed by other bacteria and be totally overlooked, and in another a few diphtheria bacteria may overgrow everything else, although in the actual illness they may be of little or no etiologic significance. In a dry preparation the conditions are represented as they actually exist. In the bacteriologic diagnosis of the upper air passages, just as in the bacteriologic diagnosis of peritonitis or cystitis, culture results may be misleading. It seems probable to the writer that when dry preparations are granted their proper place in the diagnosis of angina, the noticeable contradictions between clinical conditions and bacteriologic examinations will disappear. But the seat from which a dry preparation is made must be selected far more intelligently than in the present culture examination method—that is, when the diseased region is easily accessible, a slide must be prepared from it, and when diphtheria is in the larynx alone, more than the saliva must be examined. Especial attention should be paid to the tonsils and to the soft palate. Sometimes it may be necessary to make several preparations, just as it is in the examination of sputum for tubercle bacilli. In making cultures, the importance of selecting the proper regions is often forgotten. In addition to the factors already mentioned, this neglect may explain the unsatisfactory lack of harmony between the clinical diagnosis and the bacteriologic examination. Dry preparations made from material sent for examination do not always fulfil the above demands, because the material may not be suited for microscopic examination.

The occurrence of diphtheria bacilli in the throats of healthy people or of those without clinical diphtheria or without croup has caused a great deal of confusion. The term "pseudodiphtheria bacilli" has been applied to such organisms, but their characteristics really do not differ from those of the true diphtheria bacilli. Virulence can be tested upon animals only after the bacteria have been altered biologically by making cultures. Hence it is clear *à priori* that animal experiments do not prove anything regarding the bacterial virulence in man. Besides, it has even been demonstrated that diphtheria bacilli taken from the buccal cavity of healthy people, although usually considered pseudodiphtheria bacilli, may sometimes be just as virulent for animals as true diphtheria bacilli taken from patients suffering from diphtheria. The different influences of diphtheria serum upon animals inoculated with true and false diphtheria cannot be

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used as a differential point, because such a differentiation begs the question, taking it for granted that the cultures which act differently to the serum must of necessity belong to different species. The numerous efforts to separate the pseudo-diphtheria bacilli from the true bacilli have been absolutely fruitless. It seems to the author better to assume that the variability of the attacks of diphtheria in man is due to individual differences in species and to the continual reinoculation. The general endeavor, however, is to continue the search for new points of differentiation. It seems doubtful if this effort will meet with any success.

One of the most recent points of distinction is the double stain recommended by Neisser.[1] He found that if dry specimens are prepared from a Löffler's serum culture ten to twenty hours old at 35° C., stained for one to three seconds with acetic acid methylene-blue, washed in water, and then stained with aqueous vesuvin [2] three to five seconds, certain portions of the organisms, the so-called isolated polar bodies, stain blue. He claims that true diphtheria bacilli exhibited this peculiarity, but not the pseudo-bacilli.

This is in itself very interesting; but it is quite incomprehensible to the writer that these authors fail to recognize that they argue in a circle in all these endeavors to discover points of differentiation between true and false diphtheria bacilli. Because the older differential characteristics have proved unreliable, they unconsciously fall back upon the clinical diagnosis by assuming that the true diphtheria bacilli must come from a case that is clinically diphtheria. Neisser's results, we believe, prove nothing more than that the polar stain is present in serum cultures made from clinical diphtheria, but is absent in cases which cannot be considered diphtheria from a clinical standpoint (healthy people, follicular tonsillitis, and doubtful clinical cases). According to the author's opinion, this peculiarity does not prove anything about the differentiation between true and false diphtheria bacilli, nor does it indicate the diagnosis in any given case. In typical cases there is no need of this criterion. The test does not decide whether these diphtheria bacilli called false by Neisser are anything more than varieties of so-called true diphtheric bacilli. Biologic variations responsible for the lack of a clinical picture of diphtheria may, of course, be equally responsible for the absence of his reaction. Neisser considers his stain is available only for very young cultures upon Löffler's serum, which, of course, means that this criterion is not adapted to differentiate variations of species. The emphasis placed upon the age of a culture and the statement that false diphtheria bacilli when old may react in the same way show how little valuable the method really is. The writer's critical objections to the bacteriologic methods of making diagnoses in diphtheria should by no means influence the reader's opinion regarding the etiologic significance of diphtheria bacilli in relation to serum treatment.

Virulent diphtheria bacilli may be found in the fluids of the mouth months after diphtheria has disappeared. Theoretically, this fact is very important for prophylaxis, although it makes it very difficult or even impossible to keep up a proper quarantine. It is hardly possible to isolate the majority of convalescent diphtheria cases. The scheme at the end of this book shows how diphtheria cases are examined at Bern.

The presence of streptococci and staphylococci is of interest in the etiologic study of the different types of sore throat. They may often be present at the same time with diphtheria bacilli (compare Figs. 218 and 219). Fränkel's pneumococcus (Fig. 214), the Micrococcus tetragenus (Fig. 217), the Micrococcus conglomeratus, and several others may also occur. All these bacteria frequently inhabit a normal mouth, so that little importance can be attached to their demonstration by culture methods as compared with the demonstration of their presence in large numbers in freshly prepared dry slides. (Regarding the method of preparation, see p. 596.)

[1] *Zeits. f. Hyg.*, 1897, vol. xxiv.

[2] One gram of methylene-blue (Grübler) dissolved in 20 c.c. of 96 per cent. alcohol, and then 950 c.c. of distilled water and 50 c.c. of acetic acid added.

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PLATE 9.

FIG. 1.

FIG. 2.

FIG. 3.

FIG. 4.

The pathognomonic sign of measles (Koplik's spots).

FIG. 1.—The discrete measles-spots on the buccal or labial mucous membrane, showing the isolated rose-red spot, with the minute bluish-white center, on the normally colored mucous membrane.

FIG. 2.—The partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

FIG. 3.—The appearance of the buccal or labial mucous membrane when the measles-spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthem is at this time generally fully developed.

FIG. 4.—Aphthous stomatitis, likely to be mistaken for measles-spots. Mucous membrane normal in hue. Minute *yellow points* are surrounded by a red area. Always discrete.

(The Medical News, June 3, 1899.)

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opharyngeal abscesses present a visible and palpable in the posterior pharyngeal wall. They may cause dyspnea. *phied tonsils* are evident to inspection. *Adenoids* can be felt in pharyngeal space. The mobility of the soft palate should often . For a *paralysis of the palate* or for *absence of the palate* steria), see section upon Rhinoscopy and Laryngoscopy.

t **Rhinopharyngoscopy.**—W. Lindt,[1] of Bern, has recently announced for direct inspection of the nasopharyngeal space. This is accomplished the soft palate forward and upward by means of the retractor depicted 9. With the illumination from an ordi- mirror, the posterior, lateral, and upper ne nasopharynx can thus be examined, asily if the head is bent slightly back- e anterior and posterior walls of the soft st be cocainized (2 to 4 per cent. solu- ensitive individuals (compare p. 696). od of examination is valuable espe- he purpose of detecting adenoids.

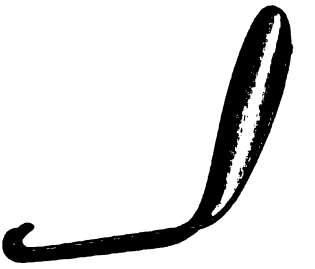

FIG. 249.—Lindt's palate hook for direct rhinoscopy.

Palate.—We should look espe- syphilitic perforation of the palate, ildren for the so-called Bednar's

al Mucous Membrane.—Besides the conditions of the membrane described in connection with other portions of the may look for cancrum oris, stomatitis, etc. A rare gangrenion of the mucous membrane of the cheek, called *noma*, is found in children. Koplik's spots, an early sign in measles, be remembered (see Plate 9).

tion of Saliva.—Increased salivation accompanies all types tis and chronic mercury-poisoning. (In one of the writer's persisted for one-half year after a single dose of calomel.) ion of saliva is diminished in *fever, diabetes mellitus, cholera, soning,* and in *facial* and *bulbar paralysis.*

XAMINATION OF THE ESOPHAGUS.

diagnosis of diseases of the esophagus, we should be on the listurbances in function, for pain upon swallowing, signs of regurgitation, in addition to the direct method of examinato be described. We must differentiate between regurgitated om the esophagus and vomitus (p. 361). The surgical rules

irecte Besichtigung und Behandlung der Gegend der Tonsilla pharyngea salpingopharyngea in ihrem obersten Theile," *Arch. f. Laryngologie,* vol.

s and mycotic patches are also sometimes seen. A high-arched palate sugted nasal septum and the former presence of adenoids.—W. S. B.]

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for palpation apply to the external examination of the esophagus. Attention should be paid to tumors which compress the esophagus, to swollen glands, to thyroid enlargements, to sensitiveness along the course of the esophagus, etc. Diverticula of the esophagus are characterized by their variations in volume and by the fact that they can be emptied.

Esophageal Sounds or Tubes.—The well-known whalebone sounds, to the tip of which an ivory olive of varying caliber may be screwed, or the hollow elastic English tubes of varying caliber are employed. Before introducing one of these instruments we should be sure that they are in perfect condition, as a faulty instrument may sometimes injure the patient.

The esophageal bougie is held in the right hand, close to the end, very much like a pen; the tip is smeared with a little oil or glycerin, and the patient, in the sitting posture, is told to open his mouth wide and bend the head back as far as possible. The tip of the bougie is introduced into the pharynx, covered by two fingers of the left hand, and pressed slightly forward. It is kept as much as possible in the median line. After passing the slight physiologic resistance at the level of the cricoid cartilage, the bougie will pass into the stomach with ease, provided there is no pathologic obstruction. Efforts to swallow should, if possible, be avoided, as they are unnecessary and only produce a gagging, on account of the contact of the soft palate with the bougie. It is important for the patient to breathe regularly. (Compare Stomach Examinations, p. 364.) If the examiner is awkward, or if there is a paralysis or lack of sensation in the pharyngeal region, a hard bougie will enter the larynx more readily than a soft one. But this fault can easily be avoided if the point of the bougie, while being introduced, is allowed to pass along the posterior wall of the pharynx. When hard hollow bougies are employed a peculiar whistling respiratory noise comes from the end of the bougie upon introduction (p. 365). An inexperienced operator may be alarmed by this, believing that the tube has entered the larynx. The noise is in reality produced by the respiratory variations of pressure in the interior of the thorax, which are transmitted in sufficient strength to the esophagus to produce an in-and-out current of air through the bougie, unless its opening is occluded by the esophageal wall (esophageal breathing). This can be easily demonstrated by placing a burning candle before the end of a stomach tube in position. The flame will flicker regularly with respiration. The greater size of the hard tubes and their resonance probably explain why this sound is much more intense when these are employed than when soft ones are introduced. The moment the opening of the bougie enters the stomach this noise ceases. If the bougie enters the larynx dyspnea follows immediately, and, in case the larynx is not anesthetized, violent attacks of coughing and severe pain.

Esophageal bougies will demonstrate the presence of stenoses by transmitting a sensation of resistance to their advance. The most frequent cause of stenoses of the esophagus is carcinoma; then come diverticula, foreign bodies (bits of bone, false teeth, coins, etc.), syphil-

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itic strictures, strictures from burns, compression of the esophagus from without by thyroid tumors, neoplasms, or aneurisms of the aorta, etc.

With signs of stenosis of the esophagus, it is a good plan to attempt a preliminary diagnosis regarding the nature of the disturbance before passing the bougie, taking the history and the general condition into consideration. If aneurism of the aorta is suspected the bougie should not be passed.

The bougie, particularly by indicating the location of the obstruction, may throw some light on the nature of the stenosis. The following data should be remembered: Distance from the incisor teeth to the entrance of the esophagus equals 15 cm. (6 in.); distance from the incisor teeth to the bifurcation of the trachea, about 25 cm. (10 in.); distance from the incisor teeth to the cardia, about 40 cm. (16 in.).

The consistence of an obstruction is also of considerable importance —*e. g.*, it may be shown by the noise produced by contact of the bougie against a hard substance, such as pieces of bone, coins, etc.

It is also important to determine the degree of stenosis. This is accomplished by trying a series of sounds of decreasing caliber until one is found sufficiently small to slip through the obstruction. In the patient's interest we should not employ any force while making this attempt.

It is also necessary to estimate the length of the obstruction. This is very difficult to determine with the ordinary stomach tubes, but quite simple if we employ the whalebone sound. For this purpose the latter is furnished with an "olive" (of ivory or bone) which can be screwed or fastened over one end of the sound. Varying sizes of "olives" are necessary, so that one can be selected which will pass through the obstruction. After slipping by the obstruction the sound is withdrawn until the lower edge of the obstruction can be plainly felt; then by comparing the position of the upper and the lower edge of the obstruction we can readily determine its vertical extent. In the same way multiple obstructions can be located and measured.

Repeated examinations of an esophageal stenosis sometimes reveal this peculiarity—at one attempt the obstruction can be overcome very readily, while at another it is very difficult or quite impossible. Perhaps this depends upon the fact that the esophagus may alter its condition quickly, due to the atrophy of ulcerations or to the persistence of food residue. Frequently, however, it depends upon some dilatation of the esophagus above the stenosis, so that the sound slips into a sort of pocket, and perhaps does not always meet the obstruction in exactly the same way. It may also be due to spastic conditions of the esophagus, which may be added to the anatomic obstruction by the irritation in sounding. It is also possible that a saccular "pressure diverticulum" crowds against the esophagus, and when filled compresses its lumen, and so stops the sound, although when the diverticulum is empty the sound readily slips by. However, before assuming the existence of such a diverticulum, we should always bear in mind that the saccular "pressure diverticula" are found only in the neck at the upper-

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most part of the esophagus, and if filled are usually palpable externally. "Traction diverticula" occur in the lower part of the esophagus. They cause no stenosis.

In sounding the esophagus the exhibition of a circumscribed pain at the moment when the tip of the sound passes over a definite place is of considerable diagnostic importance. Such a painful condition is observed in *carcinoma of the esophagus* and of the *cardiac orifice of the stomach*, sometimes, when there is no stenosis, in the rare affection *esophagitis*, and in the quite as rare *ulceration of the esophagus and of the cardiac orifice.*

With every attempt to sound the esophagus we should always carefully examine the sound as soon as it is withdrawn, and especially the window at the tip of the stomach tube if the latter is employed, in order to determine whether any fragments have been scraped off. Small bits of tissue are not infrequently found adhering to the window of the stomach tube in cases of carcinoma of the esophagus, and are often large enough to be sectioned after hardening in formalin, or with the aid of the freezing microtome (see p. 427, note). In this way the diagnosis of carcinoma may sometimes be confirmed. In "thrush" involving the esophagus, the fungous elements may be demonstrated microscopically in particles adhering to the withdrawn sound (compare p. 603, Fig. 223). The window of the sound frequently contains bloody mucous in carcinomatous and non-carcinomatous ulcerations of the esophagus.

Other methods of examining the esophagus are subordinate to the methods of palpation and of sounding. *Auscultation of the esophagus* has thus far furnished rather barren results. Hamburger and Zenker auscultate the swallowing murmur in the neck upon the left side of the trachea, and in the chest to the left of the spinal column, at the level of the eighth dorsal vertebra. Here, during the act of swallowing, we can hear a characteristic clucking and rattling murmur. With pronounced esophageal stenoses the swallowing murmur may either disappear entirely or may be delayed from the level of the stenosis downward. Meltzer[1] studied more minutely the murmur which arises upon the entrance of nourishment into the stomach, and which we hear best by auscultating with the stethoscope in the neighborhood of the xiphoid process. He arrived at the following conclusions: In normal individuals, six to seven seconds after the beginning of a separate act of swallowing fluid or gruel, we hear at that point a more or less distinct prolonged murmur, as if air or fluid was being squeezed through a sphincter-like opening (squeezing murmur). According to Kronecker and Meltzer's examinations, fluids are squirted into the lowest part of the esophagus at the very commencement of the act of swallowing. Hence we are entitled to conclude from the delay of the murmur at the cardiac orifice that, normally, what is swallowed in each separate act of swallowing remains six to seven seconds just above the cardiac orifice before it reaches the stomach. With a relaxed (insufficient) or paralyzed cardiac orifice, a distinct murmur can be heard immediately after the beginning of the act of swallowing (squirting murmur). If this "squirting murmur" is plainly to be heard, the later "squeezing murmur" is lacking. The former shows that the action of the mylohyoid muscle and the base of the tongue squirts the swallowed material directly into the stomach without any obstruction. In some cases both murmurs can be heard one after the other. Either one or the other murmur can be heard in all but very few cases. Swallowing warm fluids seems to make the "squeezing murmur" more distinct. It occurs earlier in weak individuals, perhaps

[1] *Centralbl. f. d. med. Wissenschaften*, 1883, No. 1.

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four seconds after the act of swallowing. If several swallows succeed each pidly, the result is not uniform. Either the "squirting murmur" becomes stinct with the rapid swallowing, even where it was not heard with a wallow, or we hear only a "squeezing murmur" six to seven seconds after swallow, or, finally, we may hear nothing at all. Meltzer considers the ice of a distinct "squirting murmur" as a trustworthy symptom of incy of the cardiac orifice. He found it in individuals who complained of ating their food with coughing, and exceptionally in patients with d recurring syphilis. Meltzer's swallowing murmur has thus far proved ed clinical value. Quincke[1] pointed to a decided objection in that the ng murmur is differently affected by the way in which the fluid is mixed during the swallowing and by the condition of fulness of the stomach.

ussion of the esophagus may in rare instances aid in the diagnosis of large re diverticula" of the esophagus. If the diverticulum is filled with air, nitic note can be elicited in the cervical region or at the superior thoracic . If the diverticulum contains food or liquid, the note will be dulled or ne variations depending upon the ingestion of nourishment are especially ristic and important for the diagnosis.

phagoscopy is the inspection of the esophagus with the aid of a specially ted instrument, a so-called esophagoscope, furnished with electric illumi- Further experience with this instrument may demonstrate its practical ut thus far its application is too troublesome and too heroic for most

likicz[2] has recently furnished interesting and important information in to the measurement of the intra-esophageal pressure, which indicates motor power of the esophageal musculature and the tonus of the cardia. essary to have a mercury manometer with an index of 100 mm.; the mercury manometer described upon p. 136 may be employed. A thin, hageal tube with an open end, and made of patent rubber (see p. 364) of resistance to overcome lateral compression, is introduced into the esophthat its open end is at the level of the manubrium. Before the introof the tube its outer end is clamped, and the clamp is not removed until has been connected with the manometer. When the clamp is removed ury usually falls, indicating that the end of the tube is within the air- g thoracic portion of the esophagus. When the patient swallows the rises, and this elevation, according to Mikulicz, should amount to about A diminution of this pressure may be due either to atony of the esophsculature or to a diminished tonus of the cardia, which prevents the n of a greater pressure. The tonus of the cardia is now estimated by ing the pressure at which fluids flow into the stomach. For this purube is inserted between the esophageal tube and the manometer, and its mb connected with a funnel by rubber tubing. Fluid at body temperhen poured into the funnel, and the pressure is read from the manometer oment the fluid commences to run into the stomach. In carrying out manipulation, the lower end of the esophageal tube must be pushed just above the cardia, otherwise the hydrostatic pressure of the column collecting between the opening in the end of the tube and the cardia lost. Since the tube and its connections with the manometer are filled , care must also be taken to bring the zero point of the manometer to of the cardia, since it is at this point that we wish to determine the According to Mikulicz, the initial pressure at which lukewarm, indifids flow into the stomach varies between 2 and 17 mm. of mercury. tance of the cardia is shown by the values obtained by this method. lly low or high pressures are indicative of corresponding variations in of the cardia. With due regard to the results obtained by this method, letermine whether a low or a high pressure during deglutition is due

[1] *Arch. f. exper. Path.*, vol. xxii., p. 395.
[2] *Deutsch. med. Woch.*, 1904, Nos. 1 and 2.

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simply to a diminished or an increased resistance of the cardia or to abnormal conditions of the esophageal musculature. After the introduction of the fluid, it is well for the patient to repeat the act of deglutition, when the pressure should be measured again.

This procedure may also be employed for the estimation of the capacity of the esophagus, which is of importance for the diagnosis of diffuse esophageal dilatation, a subject about which so much has recently been written. The greater the amount of fluid which can be poured into the esophagus before any of it escapes into the stomach, the greater is the esophageal capacity. If such a diffuse esophageal dilatation be due to cardiospasm, as is usually the case, it will be found not only that a large quantity of fluid may be introduced into the esophagus before any of it flows into the stomach, as indicated by a sudden fall of the mercury, but that the fluid does not enter the stomach until it is acted upon by an abnormally high pressure.

FIG. 250.—Laryngeal mirror.

Several other methods of esophageal investigation have recently been tried in order to gain information in reference to diffuse dilatation and deep-seated diverticula. One of these, for example, consists of the introduction into the esophagus of a rubber balloon, and its subsequent inflation. If this balloon can be markedly dilated by slight pressure, without annoyance to the patient, and at all points of the esophagus, a diagnosis of diffuse dilatation is indicated. The size of the rubber bag or the caliber of the esophagus may even be made visible by means of a skiagraph, a procedure which is facilitated by filling the balloon with a bismuth subnitrate suspension instead of with air. The esophagus itself may be filled with a mixture of potato soup and bismuth subnitrate, when a skiagraph will reveal the presence of diffuse dilatation or of esophageal diverticula.

Mention must also be made of Rumpel's experiment for differentiating spindle-shaped dilatations of the esophagus from deep-seated diverticula. Rumpel introduces a rather soft tube which, in addition to the end opening, has a number of lateral perforations in its lower half. When it is certain that the tip of this tube is in the stomach, a second tube is introduced into the esophagus alongside of the first one. Water is then poured into the esophagus through the second tube. If a spindle-shaped dilatation be present, the water immediately flows into the stomach through the lateral perforations in the tube first introduced, so that none of it may be recovered by means of the second tube. If there be a diverticulum, however, at least a portion of the water introduced may sometimes be siphoned out by means of the second tube. A number of difficulties are encountered in carrying out this experiment in practice. In the first place, it is not always easy to determine that the first tube extends into the stomach, since it may become bent upon itself either in a spindle-shaped dilatation or in a diverticulum. If a diverticulum be present, moreover, it is not such a simple matter to introduce the second tube into it. The first difficulty may be overcome by performing the experiment upon a full stomach, and introducing the first tube so far that all of the perforations are presumably in the stomach; the gastric contents are then siphoned out, indicating that the tube has really passed the cardia. The tube is now withdrawn until only the open end remains in the stomach, and the experiment is then carried out in the usual manner. To diagnose a diverticulum, it is necessary not only to introduce a tube into it, but also to recover through this tube a part or all of the fluid introduced.

In view of these difficulties and the further one of introducing a second tube alongside of one already in the esophagus, the author will also give a procedure

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suggested by Richartz[1] for the differentiation of spindle-shaped dilatations of the esophagus from deep-placed esophageal diverticula. The esophagus is first dilated and well irrigated, so that all particles of food are removed. A tube similar to that used by Rumpel, with an end opening and numerous lateral perforations, is then introduced almost to the cardia. This tube is connected with a funnel. The esophagus is then thoroughly irrigated with a solution of methylene-blue, the fluid being introduced and withdrawn a number of times, so as to be sure that a diverticulum, if present, is filled with the methylene-blue solution. If the patient strain slightly, some of the solution will be forced back into the funnel, proving that the tip of the tube is within the esophagus. The tube is now slowly pushed down into the cardia, allowed to remain there a short time, and then drawn back to its former position. In pushing the tube downward, the fluid in the esophagus runs into the stomach. Clear water is then introduced into the esophagus through the funnel and recovered by siphonage, and this procedure is repeated several times. If a fusiform dilatation be present, the water returns colorless; whereas if there be a diverticulum, the returning water will be stained from the retained methylene-blue solution.

Under certain circumstances, Rumpel's experiment may be simplified by introducing the perforated tube just to the cardia, filling the esophagus with water, and then pushing the tube downward into the stomach, when the water will flow into the stomach, provided that it has not been caught by a diverticulum. The tube is then withdrawn until the end is just above the cardia. If the patient, by straining slightly, is now able to empty a portion of the fluid introduced, it necessarily follows that a diverticulum must be present.

These and similar experiments may be carried out with variations dependent upon the individual case.

LARYNGOSCOPY AND TRACHEOSCOPY; AUTOSCOPY OF THE LARYNX AND TRACHEA.

EXAMINATION WITH THE AID OF A MIRROR.

SINCE the invention of the laryngeal mirror by Garcia, Türk, and Czermak, mirror examination has played the most important part in the diagnosis of diseases of the larynx and trachea.

The principle of this method of examination is as follows: A reflector (a perforated, slightly concave mirror), which is ordinarily fastened to the examiner's head so that the central opening is opposite one of his eyes, collects the rays of light from the source of illumination (Argand burner, Welsbach light, electric light, or student lamp) and throws them upon a small mirror, attached at an angle of about 45 degrees to a fairly firm handle, which the examiner holds at the back of the patient's throat (Fig. 250). The reflector and the laryngeal mirror are so arranged as to throw an intense illumination into the larynx, and at the same time reflect an actual mirror picture of the illuminated parts to the examiner's eye.

In the ordinary position the base of the tongue hides the opening of the larynx, so that it is necessary for the patient to protrude his tongue as far as possible, while the examiner at the same time holds it firmly in

[1] *Deutsch. med. Woch.*, 1904, No. 21.

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position with the aid of a napkin or towel wrapped about it. Fig. 251 and the following pages explain the methods of application of the laryngoscope. Fig. 252 shows the path of the rays of light in laryngoscopy and tracheoscopy. The latter figure clearly illustrates the fact that to see the larynx plainly the laryngeal mirror must be held more vertically and introduced farther backward; whereas, to observe the trachea, it must be held more horizontally and farther forward. The lower the handle is depressed, the more the parts which lie still farther backward come into the field of vision. The following sequence in the appearance of the parts in the field of vision is accomplished by gradually depressing the handle of the mirror: epiglottis and base of the tongue,

FIG. 251.—Technic of laryngoscopy.

anterior commissure of the vocal cords and anterior laryngeal wall, anterior tracheal wall, bifurcation, posterior tracheal wall, posterior laryngeal wall and arytenoid cartilage. Fig. 253, copied from Heitzmann, represents a normal laryngeal picture.

A few details for the practical employment of laryngoscopy: The source of light and the heads of the examiner and patient should be practically upon the same horizontal plane. The laryngeal mirror should be heated over the lamp immediately before each introduction, to prevent its being dimmed by condensed steam.[1] In warming the mirror the glass side should be held at some little distance above the flame, and the degree of warmth tested by touching the back of the mirror to the exam-

[1] The same purpose can be accomplished by coating the surface of the mirror with a very thin invisible layer of soap.

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iner's hand. Not only is it necessary for the examiner to hold the tongue firmly as well as carefully, but it should be actively protruded by the patient, as otherwise a little pain and a reflex bulging of the posterior part may be produced and render the examination difficult. In order to prevent gagging and choking movements, the pharyngeal parts should not, if possible, be touched with the mirror. The posterior pharyngeal wall and the base of the tongue are especially to be avoided. Touching the

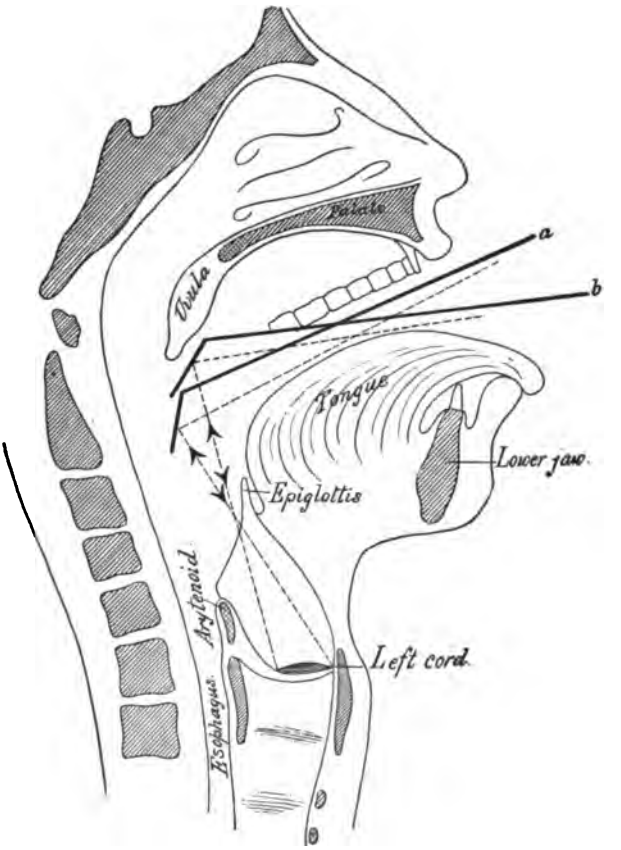

FIG. 252.—Diagrammatic representation of direction of rays of light in laryngoscopy and tracheoscopy (sagittal section of head and neck): *a*, Position of mirror for laryngoscopy; *b*, position of mirror for tracheoscopy.

uvula ordinarily causes less trouble, and, as it so often drops down in front of the mirror, one can sometimes simplify the examination by supporting and pressing it upward and backward with the back of the mirror. The examination is almost always facilitated by convincing the patient that it is not in any way painful, and that, even though at the beginning the procedure may produce gagging movements, quiet and patience will always accomplish the result. The patient should always breathe in quietly and regularly, and should intone with expiration either

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a prolonged "ah" or "a" (as in bake). This device elevates the epiglottis, and so permits the examination of the interior of the larynx. If the epiglottis is depressed enough to obstruct the view, intoning "ee" will usually elevate it sufficiently. It is rarely necessary to employ an instrument which has been constructed for the purpose of elevating the epiglottis, nor to cocainize the pharynx, provided one takes the time

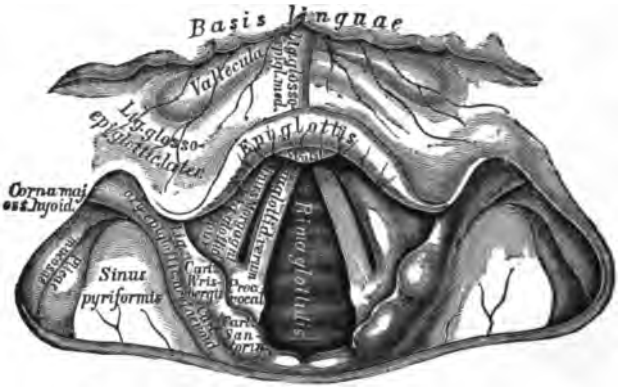

FIG. 253.—Normal laryngeal picture (after Heitzmann).

and patience to get the patient somewhat accustomed to the examination. If cocain is to be employed, the pharyngeal parts should be quickly painted with a brush dipped in 5 per cent. solution of cocain hydrochlorid, and the patient should be instructed to spit out the solution immediately after its application, in order to prevent intoxication. In

FIG. 254.—Paralysis of both thyro-arytænoidei int. (tensors), due to acute laryngitis. Position of cords during phonation (after v. Ziemssen).

FIG. 255.—Posticus paralysis. Bilateral complete paralysis of crico-arytænoidei postici (dilators) at moment of inspiration (after v. Ziemssen).

FIG. 256.—Arytænoideus paralysis. Paralysis of interarytænoidei transversi and obliqui (respiratory closers of glottis) in laryngitis. Position of cords in phonation with open glottis respiratoria (after v. Ziemssen).

FIG. 257.—Recurrence paralysis. Cadaver position of left cord (midway between adduction and abduction, with complete paralysis of laryngeus recurrens sin). Inspiratory position of right cord (after v. Ziemssen).

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FIG. 258.—Carcinoma of right false and true cords. Ant. commissure of cords pushed to left (after v. Ziemssen).

FIG. 259.—Pedunculated fibrous polyp arising from lower surface of left cord. Inspiratory position (after v. Ziemssen).

FIG. 260.—First stage of tuberculosis of larynx. Ulceration of right cord and swelling of interarytenoid region with formation of folds. May be early ulceration here.

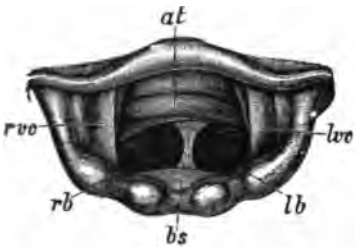

FIG. 261.—Tracheoscopic picture of anterior tracheal wall and of bifurcation: *at*, Anterior tracheal wall; *rvc* and *lvc*, right and left cords; *rb* and *lb*, right and left main bronchi; *bs*, seat of bifurcation (after Mackenzie).

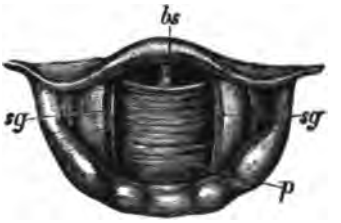

FIG. 262.—Tracheoscopic picture of posterior tracheal wall and bifurcation: *p*, Posterior tracheal wall; *sg*, regio subglottica; *bs*, seat of bifurcations (after Mackenzie).

examining the larynx we should pay especial attention to the mobility of the parts, especially the vocal cords, to their color, to accidental alterations in the surface (swelling, ulcerations, coating with mucus, etc.). It is impossible to go into a detailed description of the results of examination, and so the author will limit himself to appending the accompanying seven illustrations, which represent a few of the commonest and most important findings.

The same principles and rules apply to **tracheoscopy.** The patient should, however, sit considerably higher than the physician, so that the mirror can be held more horizontally and looked up into from below. Further, the patient should hold his trunk and neck quite straight, and at the same time bend his head forward at the atlanto-occipital joint (chin against the neck). This position brings the axis of the pharynx into a more favorable position for tracheoscopy. The trachea cannot be observed well in all individuals, but in many we can

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get a look as far as the bifurcation without any special difficulty. In such cases all sorts of tracheal stenoses can be examined and recognized. Fig. 261 represents the normal tracheoscopic picture with the anterior tracheal wall. Fig. 262 represents the same with the posterior tracheal wall (a different position of the mirror). Both represent the bifurcation of the trachea.

Inferior tracheoscopy or *laryngoscopy* means the examination of the trachea or larynx from a tracheotomy wound. For this purpose we employ a so-called "subglottic mirror," which is constructed in quite the same fashion as the laryngeal mirror, except that it is much smaller (diameter of 7 to 10 mm.). This is introduced through the tracheal wound, and the illumination is thrown in from the reflector in the same way as described above. Its principal use is to determine the causes which prevent the withdrawal of the cannula in croup operations.

DIRECT EXAMINATION OF THE LARYNX, TRACHEA, AND BRONCHI.

(Autoscopy; Orthoscopy; Direct Laryngoscopy and Tracheoscopy; Bronchoscopy.)

A. Kirstein[1] has described a new method of examining the larynx and trachea without employing a mirror. The base of the tongue is drawn forward with the aid of a spatula-shaped instrument, and the larynx and trachea are then examined directly. Kirstein described this method under a rather unfortunate term—"autoscopy" of the larynx and trachea. It would seem to the author more correct to name the procedure "orthoscopy of the larynx and trachea," or "direct laryngoscopy and direct tracheoscopy" (as opposed to indirect or mirror laryngoscopy and tracheoscopy). Kirstein employs for direct laryngoscopy the instrument pictured in Fig. 263, the so-called "autoscope"; but for most cases he has more recently substituted a simpler instrument, which is nothing but a tongue depressor of a particular shape (Fig. 265). The original "autoscope" is now recommended by Kirstein only for performing autoscopic operations for demonstration and for examining children.

The "autoscope" (Fig. 263, 1) consists of a grooved metallic spatula (*S*) about 12 cm. long and about 3 cm. wide. The groove varies from 1 to 2.5 cm. in depth. It is attached at a right angle to the handle (*G*), and well rounded off to prevent injury. The anterior end (*d*) of the normal spatula is quite sharply bent downward. Attached to the end of the spatula, next to the handle, is an electric lighting apparatus (not shown in the figure), so arranged that a cone of rays is transmitted, by means of a prism, along the groove exactly in its long diameter. A contact contrivance for the electric light is shown in the figure. The part *r*, which is movable, forms a kind of a roof or sheath to the grooved spatula, and so prevents the upper lip or the moustache from obstructing the view. The application of the instrument can be readily understood by studying Fig. 264.

The instrument is grasped by the handle and inserted so that the tip of the grooved spatula lies between the base of the tongue and the epiglottis. The base of the tongue is then hooked downward and forward by raising the handle of the instrument a little, and at the same time the epiglottis is slightly raised by pulling upon the median glosso-epiglottic ligament. Pressure upon the teeth must be avoided. If the epiglottis obscures the view into the larynx, cocain may be applied and the instrument introduced directly behind the epiglottis. For this purpose a differently shaped spatula (Fig. 263, 2) is employed.

[1] *Therap. Monatshefte*, 1895, vol. ix., p. 361; *Ibid.*, 1896, p. 370; *Encyklop. Jahrbücher*, 1896, vol. vi., p. 30.

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the instrument is properly introduced, the examiner looks down between teeth and the grooved spatula while illuminating the depths interior with

FIG. 263.—Kirstein's autoscope: 1, Ordinary spatula; 2, intralaryngeal spatula.

ectric light. Fig. 264 illustrates the position of the patient, with the trunk slightly forward and the head thrown back a little. This position brings the

FIG. 264.—Technic of autoscopy (Kirstein).

tis of the larynx and trachea in as direct a line as possible with the axis of the outh. More recent experience has led Kirstein to recommend the instrument

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pictured in Fig. 265 (a simple tongue spatula with a special groove) for most cases where direct laryngoscopy is advisable. The illumination is furnished by the ordinary head mirror. The advantage of Kirstein's method is that the parts are seen more naturally than by a mirror, with richer and more vivid shades of color. Nevertheless the author is convinced that direct laryngoscopy will never usurp the place of mirror laryngoscopy. The application of the instrument is much more disagreeable to most individuals than indirect laryngoscopy, and almost always necessitates the employment of cocain. Besides, we can rarely view the entire larynx, and still less the trachea. In many cases the anatomic position of the parts is such that we do not see any farther than to the epiglottis. The method furnishes very incomplete conclusions about the motility of the larynx, because the mobility of the parts is disturbed by the examination. Advantages of the method, however, are the directness of the picture and the ease with which the posterior parts of the larynx can be examined. These are very difficult to see in the ordinary laryngeal mirror, and, if seen, are sharply shortened; they are

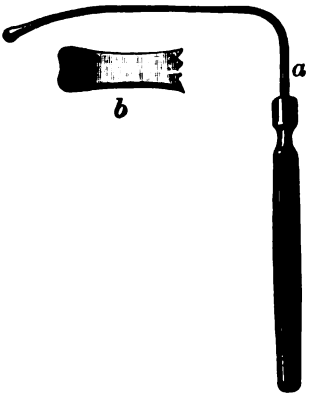

FIG. 265.—Kirstein's tongue spatula for autoscopy: *a*, Lateral view; *b*, end of spatula, seen from above.

very important in the diagnosis of initial tuberculosis affecting the arytenoid folds. Still other advantages are that direct laryngoscopy can be usually accomplished quite easily in small children; that in difficult cases the examination can be effected under chloroform anesthesia; that it decidedly simplifies intralaryngeal operations, and that it allows of such operations to be carried on under narcosis.

Kirstein has even succeeded in introducing a small so-called subglottic mirror into the larynx of patients who have been cocainized. He attached this with a long rod to the autoscope, and was able to examine the inferior surface of the vocal cords.

It must be acknowledged that considerable practice is necessary for orthoscopy as well as for the mirror examinations. To anyone who is accustomed to the indirect or mirror laryngoscopy, the direct picture is so unusual that it is quite difficult to orient properly the apparently transposed parts, and still more difficult to correct certain habits of movement in the application of instruments, etc.

BRONCHOSCOPY.

The procedure of bronchoscopy has recently been so perfected that it is possible to inspect the interior of the bronchi by means of an endoscopic appliance which may be introduced either through the intact larynx or through a tracheotomy wound. Although this procedure has already been proved to be of considerable value, particularly for the extraction of foreign bodies, it will not be described, since it is difficult of application by the general practitioner.

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COMBINED LARYNGOSCOPY.

Kirstein and, later, Leo have recommended a combination of direct and indirect laryngoscopy, under the name of "combined laryngoscopy," for cases which are very difficult to see. The base of the tongue is depressed with the spatula of the orthoscope, and then the laryngoscopic mirror is introduced much farther down into the depths of the throat opposite the epiglottis.

RHINOSCOPY.

THE nasal cavities can be examined from in front and from behind from within the pharynx. In the former case we speak of *anterior rhinoscopy;* in the latter case, of *posterior rhinoscopy.*

For **anterior rhinoscopy** we employ a nasal speculum to dilate the nasal openings a little, and then supply an illumination reflected from the head mirror into the nasal cavities directly from in front. In this way we can observe the nasal septum, the inferior and a part of the middle turbinate bone. Quite exceptionally we also can see a small part of the superior turbinate bone. A very desirable nasal speculum is pictured in Fig. 266. Catarrhal conditions of the nasal

FIG. 266.—Fränkel's nasal speculum (one-half natural size).

mucous membranes are readily recognized by *anterior rhinoscopy;* also the atrophied dilatation of the nasal cavities due to ozena, nasal polypi, and the peculiar vasomotor engorgement of the erectal tissues in nervous nasal affections and in hay fever. The latter is specially pronounced in the inferior turbinate bone.

Posterior rhinoscopy depends upon the same principle as laryngoscopy. A cone of rays is thrown from the reflector upon a small laryngeal mirror which is introduced behind the uvula, with its reflecting surface turned upward and forward. This mirror reflects the illumination to the posterior nares and transmits the reflected image of these parts to the observer's eyes. The path of the rays of light and the position of the mirror are shown in Fig. 267. In regard to the practical application of *posterior rhinoscopy,* the following differences between it and laryngoscopy should be noted: In the first place the tongue should not be protruded; we use a very much smaller laryngeal mirror, perhaps the smallest size; and we must be very careful not to touch any part of the pharynx-wall. The patient simply breathes naturally with a relaxed palate and does not intone. If the root of the tongue bulges

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upward, it may be depressed by means of a tongue depressor, held by the examiner's left hand, or sometimes by the shaft of the mirror.

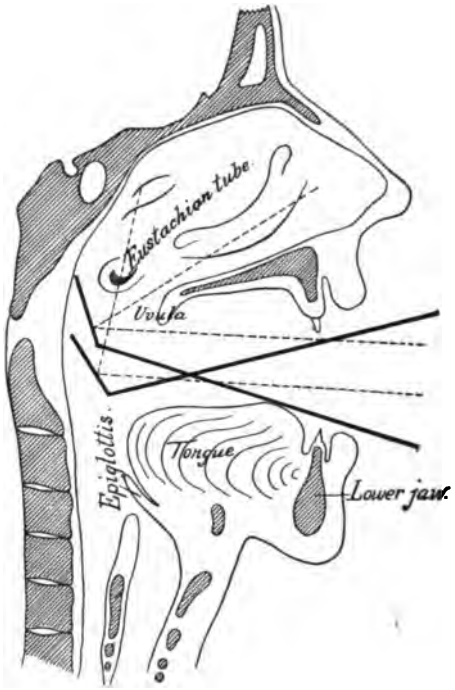

FIG. 267.—Course of light rays in posterior rhinoscopy. Sagittal section of head.

By accustoming the patient to this method, it will succeed just as well as in laryngoscopy, even in very difficult cases. If success is impossible otherwise, cocain may be employed.

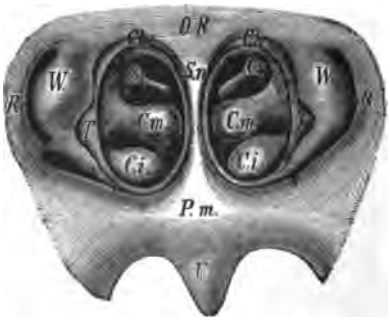

FIG. 268.—Normal picture in posterior rhinoscopy. Diagrammatic in that to obtain complete picture the position of mirror must be repeatedly changed: *S. n.*, Septum; *Ch.*, choana; *P. m.*, soft palate; *U.*, uvula; *C. i.*, lower turbinate bone; *C. m.*, middle turbinate bone; *C. s.*, upper turbinate bone; beneath each turbinated the corresponding fossa; *O. R.*, roof of pharynx; *T.*, opening of Eustachian tube; *W.*, promontory of tube; *R*, Rosenmüller's fossa (after Schnitzler).

Fig. 268 represents a normal picture in *posterior rhinoscopy*. The main object of posterior rhinoscopy is to observe: adenoid vegetations

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and tumors of the nasal pharynx, nasal polypi, inflammatory affections of the nasal cavities, alterations in the openings of the Eustachian tubes in middle-ear disease, etc. (Compare p. 687 in regard to completing posterior rhinoscopy by direct rhinopharyngoscopy.)

OPHTHALMOSCOPY.

WITHOUT going into the technic of ophthalmoscopy, a description of the more important changes in the eyegrounds will be given :

In Plates 10 and 11 several of these conditions are pictured. The drawings have been generally made in the upright image, though they have been reduced to the size of the inverted image.

Plate 10, Figs. 1, 2, and 3.—Various forms or stages of optic neuritis and choked disk.

Fig. 1. Beginning Optic Neuritis.—The disk is congested, the temporal margin is slightly veiled and swollen, the veins are moderately dilated and tortuous, the arteries are somewhat contracted.

Fig. 2. Pronounced Optic Neuritis.—The disk is distinctly enlarged, with ill-defined margins. It presents radial striations, and is opaque from exudates and hemorrhages; it is very congested and swollen. The veins are greatly dilated; the arteries very much constricted.

Fig. 3. Optic Neuritis of the Highest Grade, So-called Choked Disk.—The disk is ill-defined. It is prominent like a mushroom, and projects 2 to 3 mm. There is a difference in refraction of at least two dioptries between the apex of the swelling and the surrounding retina. The vessels make a sharp bend at the papillary margin. The disk at its margin is gray. In the center it is covered by a white exudate which hides the congestion. The veins are very much dilated and tortuous; the arteries are narrow. Both are covered by exudates in the center of the disk, and make their appearance at the margin. The central ends of the blood-vessels appear to taper. There are striated hemorrhages arranged radially on the disk and in the grayish surrounding retina.

Between Figs. 1, 2, and 3 there are only differences in grade. All three varieties may occur from local inflammations, as well as from an increase of intracranial pressure. Forms such as Fig. 1 are occasionally observed in hypermetropia and after overuse of the eyes following functional hyperemia. Pronounced forms like Fig. 3 are most frequently met with in conjunction with brain tumors and tuberculous meningitis following protracted and marked increase of intracranial pressure. Occasionally in these diseases forms like Figs. 1 and 2 are observed. The clinical conditions, therefore, speak against a sharp separation of the condition known as optic neuritis (Figs. 1 and 2) from that known as choked disk (Fig. 3).

Optic neuritis occurs in intracranial tumors (in 70 to 85 per cent. of the cases), in syphilis of the central nervous system (in 14 per cent. of the cases, presumably where an intracranial tumor or a basal gummatous meningitis is present), in tuberculous meningitis rarely, in purulent meningitis often, in primary internal hydrocephalus in case it leads to increased intracranial pressure, rarely in brain abscesses and internal hemorrhagic pachymeningitis, rarely in traumatic intracranial hemorrhages; furthermore, in certain affections of the orbit, especially tumors (in the latter case combined with exophthalmos, and, in distinction from the other cases, occurring on one side only), in polyneuritis, in chronic nephritis, especially contracted kidney; in diabetes mellitus, in scrofula, in disturbances of menstruation during pregnancy, and in labor; in chlorosis, in severe chronic and acute anemia, especially after gastric hemorrhages, and in acute infectious dis-

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eases. In the last-mentioned diseases, not depending upon intracranial lesions, the picture of ordinary neuritis or papillitis without pronounced swelling of the disk is common (Figs. 1 and 2).

For purposes of diagnosis it must be remembered that an optic neuritis, even in its most intense form, may be combined with unaffected vision. Hence the rule that in all cerebral affections the ophthalmoscope should be employed, even when no disturbance of vision is present. Furthermore, it must be mentioned that in brain tumors the size and the site of the tumor are not of exclusive importance for the development of a choked disk, but that the rapidity of growth and other unknown factors play as important rôles.

Fig. 4. Changes of the Eyeground in a Case of Severe Purpura Hæmorrhagica.—Extensive hemorrhages with inflammatory changes of the disk. The disk is not distinctly swollen. Its margins are completely lost from the exudation, and suffused with blood. There are numerous striated hemorrhages situated in the retina and arranged in a radiating fashion. The color of the hemorrhages varies from a pale red to a dark or even black red. Within the zone filled with hemorrhages, as well as in the region of the disk, the blood-vessels are scarcely visible. In the periphery of the eyeground the veins are thickened and tortuous, the arteries constricted (not visible in the figure). The picture resembles that of a thrombosis of the central vein. In this case the comparative improvement of vision spoke against this diagnosis. A moderate degree of neuritic atrophy remained.

Fig. 5. Albuminuric Neuroretinitis.—This occurs in the various forms of chronic, more rarely acute, nephritis, generally in contracted kidney. The disk presents the signs of a neuritis. It is blurred, not especially swollen, hyperemic, ill-defined. The veins are dilated, the arteries contracted. There are radiating hemorrhages in the disk and in the surrounding retina, with numerous white patches (fatty degeneration), especially in the neighborhood of the disk and in the peculiar characteristic star-shaped figure about the macula lutea. The changes in the region of the macula lutea furnish the anatomic explanation for the usually very pronounced loss of sight. It should also be mentioned that in nephritis these changes of the retina may occur without changes in the disk (pure albuminuric retinitis), as well as neuritic changes of the disk without changes of the retina (albuminuric neuritis). The changes of the retina and of the optic nerve in diabetes mellitus are often like those found in nephritis.

Fig. 6. The Eyeground in Pernicious Anemia.—The fundus appears unusually pale, and is covered with numerous irregular retinal hemorrhages and a few white patches. In one place, as occurs frequently, a white spot occupies the center of a hemorrhage. The hemorrhages and the blood-vessels have turned out somewhat too dark in the drawing. The former appear so pale in the pronounced anemia that they are recognized with difficulty and are frequently overlooked. This condition occurs in a variety of severe forms of anemia, most pronounced in the so-called pernicious anemia (compare p. 660), but is also found in bothriocephalus and ankylostomum anemia and in leukemia. The changes are not so intense and usually not so pronounced in the severe anemias of certain cancers of the stomach, and are practically absent in simple chlorosis.

Fig. 7. The Eyeground in Hereditary Syphilis.—Perivasculitis of the retinal vessels and choroiditis. Perivasculitis is especially characteristic for syphilis. The vessels, especially the arteries, appear to be outlined in white, owing to thickening of their walls. In one place the changes are so pronounced that a vessel is converted into a white strand, through which the blood-current can no longer be observed. The picture presents the characteristic spots of choroidal atrophy, and pigment deposits in the choroid. The macula lutea is especially plainly visible.

Fig. 8. Choroidal Tubercle in Acute Miliary Tuberculosis.—The choroidal tubercles are characterized as ill-defined white spots which become more intensely white, usually of an absolutely round form, measuring one-quarter to one-half a papilla in diameter. In the later stages they are larger, and their site is independent of the course of the blood-vessels. If they are situated in the region of a

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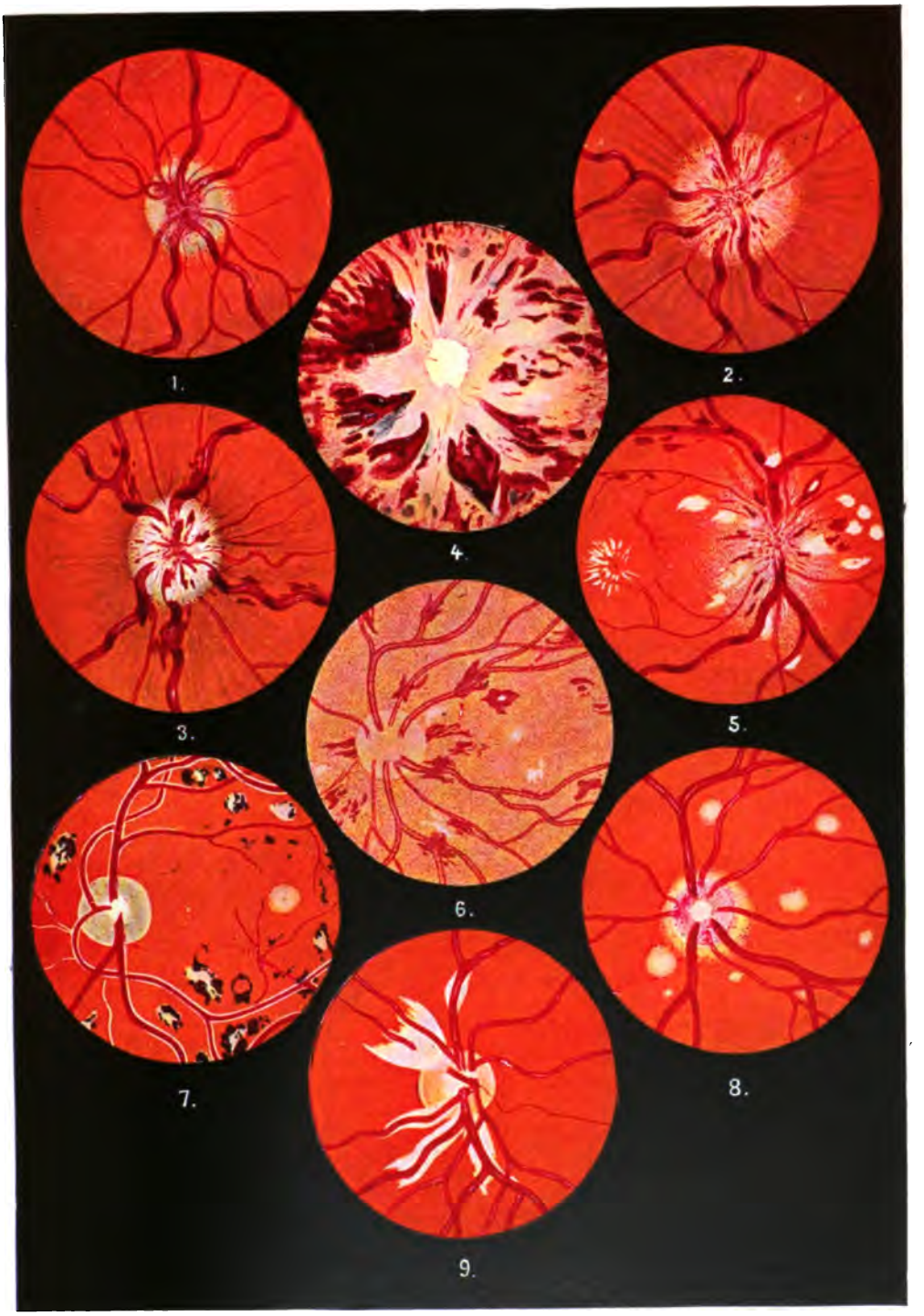
PLATE 10.



1, Early stage of optic neuritis; 2, optic neuritis; 3, choked disk; 4, neuroretinitis hæmorrhagica in purpura; 5, albuminuria neuroretinitis; 6, hemorrhagic retinitis in pernicious anemia; 7, syphilitic chorioretinitis; 8, miliary tubercle of the choroid; 9, medullated nerve-fibers.

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blood-vessel, they are covered by the latter. The drawing shows recent and old tubercles. Especially characteristic in cases where the nature of these structures is not clear is their sudden appearance and increase within a few days. They can be distinguished from retinal spots by their usually circular form and the ill-defined margins of the choroidal tubercle. In the case depicted in the figure, in addition to the miliary tubercles, there was a complicating tuberculous meningitis, which explains the hyperemic and blurred appearance of the disk (beginning neuritis). Contrary to a well-spread belief, the presence of choroidal tubercles is a rarity in uncomplicated tuberculous meningitis. Choroidal tubercles in almost all cases point to the presence of a general miliary tuberculosis. The characteristic condition of a tuberculous meningitis is, on the other hand, optic neuritis.

Fig. 9. Medullated nerve fibers of the retina, presenting a white, glistening figure, beginning at the disk and striated radially. In the upper part of the picture a retinal blood-vessel is partly covered by this white structure; otherwise, the eyeground is normal. This condition is without functional importance. It must, however, be recognized in order to distinguish it from pathologic changes.

Plate 11 gives a summary of the ophthalmoscopic pictures occurring in the various forms of optic atrophy, which it is of great diagnostic interest to distinguish.

Fig. 1. Simple Atrophy of the Optic Disk.—The disk is white and shining, the margins are unusually sharp, the light scleral ring is visible especially on the temporal side, the lamina cribrosa appear as a glistening network, including angular, grayish areas representing bundles of atrophic nerve fibers. The excavation of the atrophic disk is shallow, like that of a plate, and therefore recognized with difficulty. The color of the disk is usually most pale about the entrance of the blood-vessels and in the temporal half of the disk. The caliber of the blood-vessels usually remains normal. The very small blood-vessels which furnish nutrition to the disk are usually very few and thin. The large vessels, especially the arteries, usually diminish in size only after the atrophy has existed for a long time. The atrophic excavation of the disk develops generally only in the later stages, beginning at the margin of the disk and gradually proceeding to the center.

To distinguish this simple atrophy from inflammatory atrophy, it is of importance to note the normal condition of the blood-vessels, the visibility of the lamina cribrosa, the well-defined margins of the disk with distinct scleral ring.

Simple atrophy occurs most frequently in tabes dorsalis and in progressive paralysis; furthermore, in the so-called gray degeneration of the optic nerve without spinal or cerebral symptoms. In some cases of multiple sclerosis simple optic atrophy has occurred; in others neuritic atrophy. It should further be mentioned that simple atrophy of the optic nerve may occur as an interruption in the course of the optic nerve or of the optic tract, as in chronic hydrocephalus, where the distended infundibulum presses upon the chiasm.

Fig. 2. Atrophy of the Optic Nerve after Embolism of the Central Artery, Following Ligature of the Common Carotid in a Case of Pulsating Exophthalmus.—Complete blindness. The disk is grayish white. The margins have a distinct scleral ring; the lamina cribrosa is visible. In the center the central canal appears as a grayish dot. The vessels are contracted to mere threads.

Fig. 3. Atrophy (Primary Atrophy) of the Optic Nerve in Glaucoma Simplex.—Complete blindness. The disk is grayish white; the lamina appear in the center. The entire disk is deeply excavated. The vessels make a sharp bend at the margin and disappear in the depth. A few again appear at the bottom of the excavation; some are broader and lighter in color. The veins are somewhat dilated, the arteries slightly contracted. There is a yellow area with several pigment spots about the excavated disk (halo glaucomatosus).

Fig. 4. Neuritic Atrophy of the Optic Disk.—Incomplete blindness. The disk appears dull white; the nasal half is still slightly red in color. The margins of the disk are soft, ill-defined, without distinct scleral ring. The lamina cribrosa are not visible. The vessels are moderately contracted, especially the arteries. Some are surrounded by narrow white lines characteristic for sclerosis

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of the vessel-wall. Occasionally pigment is present in the form of small spots at the margin of the disk. These features (ill-defined margins of the disk, contraction of the blood-vessels, lamina cribrosa and scleral ring not visible) differentiate this form from that of simple atrophy.

Any inflammation in the course of the optic nerve may lead to neuritic atrophy. In some of the cases of multiple sclerosis, as has been above stated, the atrophies observed have been of an inflammatory type. The illustration is of one of these cases.

Fig. 5. Papillitic Atrophy.—Blindness. The disk is dull white and uniformly discolored. The margins are even less sharp than in Fig. 4, passing over into the choroidal changes which surround the disk (the pigment is often lacking; in other cases it is deposited irregularly). The lamina cribrosa are invisible. The vessels are distinctly contracted. These changes represent simply a higher grade of neuritic atrophy (Fig. 4), which is spoken of as papillitic atrophy, because in its occurrence the disk is included in the neuritic process. Consequently papillitic atrophy is a frequent sequence to true optic neuritis, especially in brain tumors. As the choked disk proceeds to atrophy, the disk remains swollen for a length of time, the vessels show a distinct bend, as in Fig. 3, Plate III., while it gradually assumes more and more the character of papillitic atrophy (whitish discoloration, constriction of the blood-vessels). In this condition the color of the disk is often a dirty grayish yellow.

Fig. 6. Papillitic Atrophy after Thrombosis of the Central Retinal Vein, Following Chronic Meningitis.—The disk is discolored, grayish white. Its margin on one side is distorted. It is surrounded by extensive changes in the choroid and pigment accumulations. In the center the central canal appears as a grayish dot. The vessels are converted into thin white strands (progressive organized thrombosis). In this form the condition of the blood-vessels is characteristic.

Fig. 7. Retinal Atrophy in Old Chorioretinitis.—The latter affection in this case was the result of unusually prolonged lactation, presumably developing upon a syphilitic basis. In any case, it resembled certain late stages of syphilitic chorioretinitis. Incomplete blindness. The disk is of a dirty-yellowish-gray color. The margins in this case are sharply defined; sometimes, however, they are blurred. The lamina markings are not visible. The vessels are unusually narrow. The retina and the choroid are very much changed, the choroidal vessels presenting evidences of sclerosis. The intravascular spaces are dark. There is moderate pigment accumulation in the retina.

Similar changes of the disk to those which have just been described may occur in retinitis pigmentosa and in other chronic inflammatory processes of the retina, as well as in detachment of the retina. The retinal and choroidal changes are characteristic in differential diagnosis.

Fig. 8. Atrophic Discoloration of the Temporal Half of the Disk in Alcohol Amblyopia (Central Scotoma for Green and Red; Vision Very Much Reduced).—The temporal half of the disk is grayish white, the margins are sharp, the scleral ring is distinct, the lamina are not visible, and the vessels are of normal caliber.

This is a case of atrophy of the papillomacular bundle of the optic fibers. As it occurs through toxic influences (especially alcohol, rarely tobacco, stramonium, carbon disulphid and chloral), 16.5 per cent. of alcoholic subjects and 65 per cent. of patients with alcoholic amblyopia show these changes. Similar changes have been observed after diabetes mellitus and after taking cold. It is important to remember that in 1 per cent. of persons with normal sight a somewhat similar pallor of the temporal half of the disk can be observed.

While in this illustration toxic atrophy of the temporal half of the disk presents the characteristics of a simple atrophy, it may in other cases show the peculiarities of an inflammatory atrophy (indistinct margins, slight sclerosis of the vessels, as in Fig. 4). Both forms are frequently spoken of as retrobulbar neuritis, from the supposition that the cause of the changes is a retrobulbar optic neuritis. This, however, is not quite correct, as an inflammation of the optic nerve behind the eyeball should lead to changes in the entire disk, in the form of a simple atrophy (descending atrophy) or of a neuritic atrophy.

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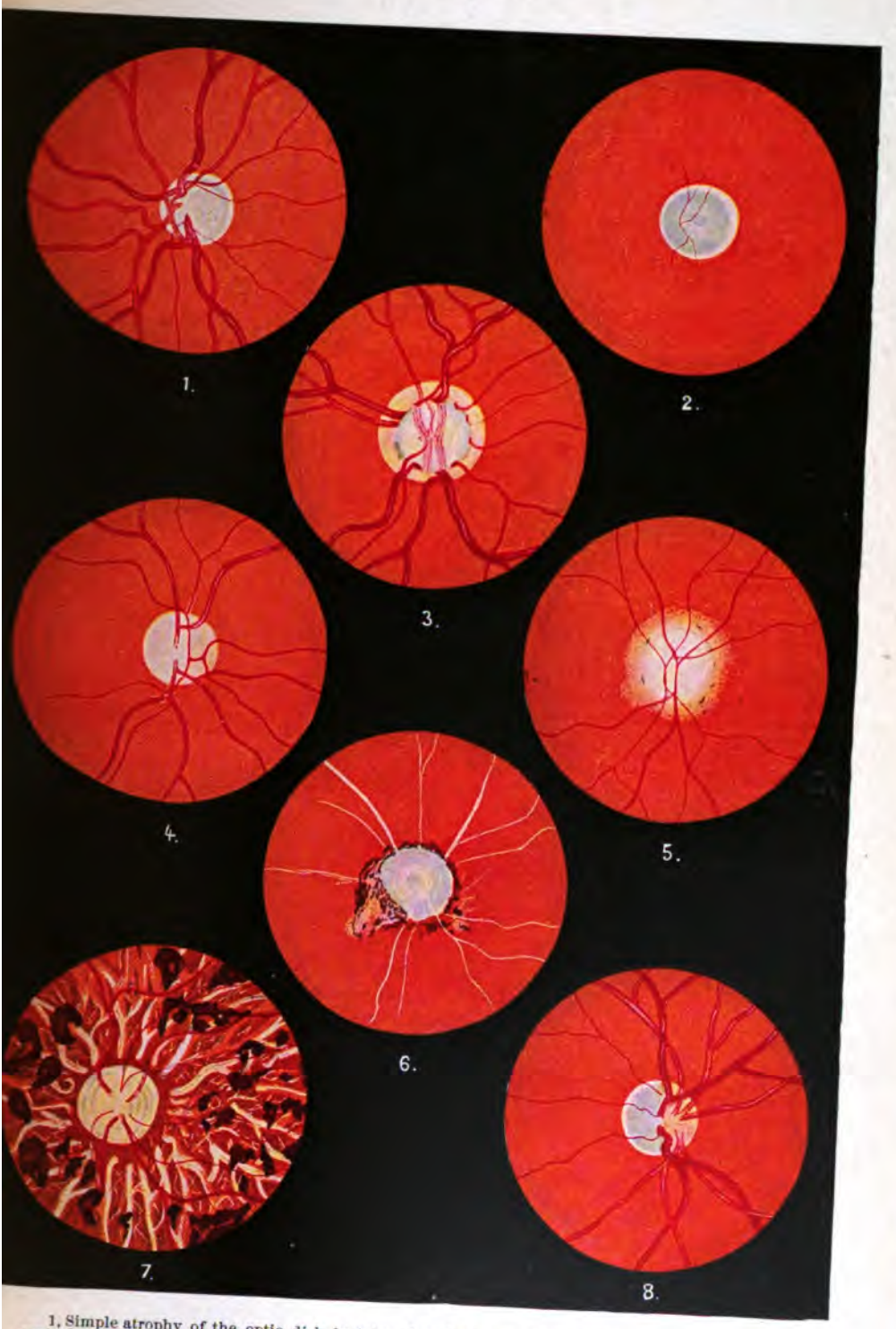

1, Simple atrophy of the optic disk in tabes dorsalis; 2, simple atrophy in embolism of the central artery; 3, pressure atrophy in glaucoma simplex; 4, neuritic atrophy; 5, papillitic atrophy after choked disk in gumma of the brain; 6, papillitic atrophy after thrombosis of the central retinal vein; 7, retinal atrophy from chorioretinitis following prolonged lactation with a probable syphilitic basis; 8, atrophy of temporal portion following retrobulbar neuritis from alcoholism.

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EXPLORATORY PUNCTURES AND HARPOONING.

EXPLORATORY PUNCTURES.

By an *exploratory puncture* we mean the introduction of a fine hollow needle, attached to a test puncture or aspirating syringe, into a diseased area, and a subsequent aspiration in order to examine the character of the material withdrawn, and especially to determine the presence or absence of collections of fluid.

SYRINGES FOR EXPLORATORY PUNCTURES.

The appropriate syringe is a little larger than the ordinary hypodermic syringe, and is furnished with a longer and rather coarser needle. The hypodermic syringe may also be used if such a large needle be fitted to it tightly. A syringe which will contain 5 to 10 c.c. of fluid is large enough. It should furnish adequate aspiration force to withdraw even quite solid fragments of tissue and sufficient fluid for a satisfactory chemical or bacteriologic examination. The essentials for a serviceable exploratory puncture (or aspiration) syringe are the following: The glass cylinder should have a uniform caliber, so that the piston fits accurately throughout its path. The packing must be absolutely tight, so that if the end is closed and the piston withdrawn the latter will slip back quickly into its former position by the force of suction. The needle must be fitted on absolutely tight, so that the same test can be successfully applied after the needle is screwed to the syringe. If the material withdrawn is to be examined bacteriologically, the syringe must be carefully sterilized. This can most easily be done with the glass syringes furnished with a metal mounting, asbestos packing, and asbestos valves, as they can be boiled. The most perfect are furnished with a contrivance for compressing the asbestos-packing valves against the cylinder, so that it can be made as tight or as loose as is desired. The cannulas or needles should be of an appropriate size. This is not always the case with all the varieties on sale. They should be 6 to 7 cm. ($2\frac{1}{2}$–$2\frac{3}{4}$ in.) long (not including the connecting portion) and about 1 mm. ($\frac{1}{16}$ in.) external diameter, so that the puncture shall be sufficiently large. Such fine needles practically obviate the possibility of any danger.

METHOD OF MAKING EXPLORATORY PUNCTURES, AND THE GENERAL RESULTS OBTAINED.

Before each attempt the needle must be carefully disinfected according to the strictest surgical rules. This means that after being used it should be carefully washed in water, and then either boiled, or left in 5 per cent. carbolic solution for at least three hours (being careful that the solution fills the bore of the needle). Just before using the needle, a few moments' immersion in strong carbolic solution will be sufficient. The needle should be freed from carbolic solution by squirting sterile

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water through it, otherwise the carbolic might produce a cloudiness in the fluid withdrawn, which might lead to error. For a bacteriologic examination the syringe must also be disinfected quite as carefully. For this purpose the syringes with asbestos valves, as already mentioned, are the most convenient. They can be boiled readily. The asbestos valves must then be loosened a little by means of the contrivance described above, as otherwise they are apt to swell a little too much. Ten minutes' boiling in water, or, better, in a 1 per cent. soda solution, is sufficient. Air bubbles should, of course, be expelled both from the syringe and the needle.

Immediately before the puncture is attempted, the spot selected must be very carefully disinfected (soap, alcohol, 1 per cent. sublimate solution). After the puncture the spot should be covered with a piece of adhesive plaster moistened in sublimate.

The skin should first be stretched tightly and the needle inserted perpendicular to the surface, and not too quickly, so as to avoid any bony parts by slightly altering the direction. During the introduction of the needle we should pay special attention to the resistances met with at the different levels. We are then able to select the appropriate moment for aspiration; this should be when we feel that the needle has passed all obstructions, entered a cavity, and is freely movable. Such palpation with the needle should never be neglected. Sometimes, however, a slight amount of fluid is obtained while the needle seems to be sticking into perfectly solid tissue. We should not desist if we obtain no fluid by aspiration, provided we have good reason to believe that fluid is there. Frequently we have penetrated too far or not far enough, and a slight push or withdrawal of the needle will be sufficient to obtain fluid. In other cases we withdraw the needle partially and then point it in another slightly different direction. At the same time we must be careful to notice any chance movements which may be imparted to the point of needle by parts which have been punctured or touched, such as the lungs, diaphragm, liver, spleen, heart. Such movements are of the greatest importance for diagnosis. If the first puncture is unsuccessful and the examiner is still convinced of the presence of fluid in the vicinity, the procedure should be repeated at an adjoining spot after he has again more carefully examined the locality. In every case where we find fluid, it is advisable to aspirate a sufficient quantity to perform all the necessary examinations. If the needle enters solid tissue, and we wish to examine this tissue histologically, we can generally obtain a specimen by pulling the needle back and forth a few millimeters, twisting it at the same time so that it acts a good deal like a cork borer. Then with sufficiently strong aspiration we can suck out a fragment of the tissue large enough for microscopic examination. In this attempt the needle must be very carefully withdrawn after the aspiration. Its contents, which often consist of quite small tissue fragments, can usually be forced out upon a glass slide by a vigorous push of the piston. The fragments may then be teased apart and examined microscopically. Whenever in any exploratory

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puncture aspiration brings no fluid, we should never neglect to examine the contents of the needle carefully in the way described. Aspiration frequently obtains only tiny flakes of pus from purulent infiltrated tissue or from thick collections of pus. Such flakes microscopically demonstrated are oftentimes quite sufficient for diagnostic purposes.

Where we obtain a large amount of fluid from an exploratory puncture, the first essential always is to examine this fluid macroscopically; thereby we determine definitely whether it is serous or purulent,[1] clear or turbid, colorless or colored, odorless or foul-smelling, and whether a purulent fluid contains the characteristic kernels of the actinomycosis (see p. 603). In turbid serous fluids the naked-eye appearances are often sufficient to decide between a purulent and a fibrinous turbidity, since the latter is characterized by the presence of flocculi.

In many cases a microscopic examination must be made. This will also furnish information in reference to the so-called chylous character of a fluid—*i. e.*, a turbidity caused by the presence of minute particles of fat. Chylous fluid in a cavity may be due to a number of different causes. As a result of a stasis of lymph in the thoracic duct, the fluid may become admixed with chyle by diapedesis without there being any solution of continuity of the tissues. In tuberculosis or malignant tumors of the pleura or of the peritoneum, the thoracic duct or some of the smaller lymphatic vessels may be opened up by the destructive process and the chyle poured out into the affected cavity. In all these cases, as in chyluria, the *admixture with chyle* may be recognized by the microscopic examination of the fluid. The fat is not present in large drops, but in the finest of granules, which in size somewhat resemble micrococci, and usually exhibit the so-called Brownian movement. In addition to the fact that they do not stain well in dry preparations (see p. 596), they may be differentiated from microorganisms by their great quantity, since such a large number of microorganisms could scarcely be present in a non-purulent fluid. They also show an additional peculiarity in that they tend to collect in a layer upon the upper surface of the fluid when it is allowed to stand. Chemical tests may also be applied after removing the fat from the fluid by agitation with ether. From a chemical standpoint it should also be noted that amounts of sugar greater than those found in the blood are not to be expected in chylous fluids, since sugar leaves the intestine through the veins and not through the lymphatics. True chylous fluids have also been found in cavities under circumstances which have not yet been explained, there being neither a lymph stasis nor a rupture of a chyliferous vessel. In these cases the affected serosa is possibly abnormally permeable to fat-particles, which, as is well known, are present in large numbers in normal blood after the ingestion of food.

[1] Although it seems very simple to differentiate a serous from a purulent exudate by means of exploratory puncture, errors may easily be made, since thin, purulent exudates exhibit a marked inclination to sedimentation, so that if the puncture be made high up, nothing but a clear serum may be obtained, the pus corpuscles having settled to the lowermost part of the cavity. In doubtful cases it is consequently advisable to make a second puncture at a lower level than that of the first.

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These true chylous exudates and transudates are not to be confounded with fluids in which the turbidity depends upon the presence of larger fat-globules. In the latter instance the finding of cells containing fat-droplets demonstrates that the admixture is dependent upon fatty degeneration of endothelial cells or of tumor cells.

Microscopic examination will also give information in reference to the *presence of leukocytes* and *admixtures with blood* which would escape macroscopic inspection (see also p. 714 *et seq.*), and to the presence of cholesterin crystals in old serous exudates (Fig. 211, *b*); it will also reveal the presence of *hematoidin crystals* (Fig. 211, *d*) in purulent collections (pleural empyemata, subphrenic abscesses, pulmonary abscesses, and biliary collections), and particularly the presence of *bacteria in exudates*. In the latter case cover-glass preparations should be made according to the rules previously given for the examination of sputum (see p. 696); it is best to select fibrinous flocculi obtained by centrifugation, since the bacteria are caught in the meshes of the fibrin network. When a serous exudate contains but few bacteria, their discovery may be facilitated by centrifugation in accordance to the method suggested by Ilkewitsch for the examination of sputum for tubercle bacilli. The nucleoproteid is precipitated by the addition of acetic acid, and the bacteria settle with the proteid. In addition to the foul odor, the microscopic demonstration of food particles and innumerable bacteria in the fluid obtained from the peritoneal cavity by exploratory puncture is of great importance for the demonstration of a perforation of the stomach or intestine. The microscopic demonstration of particles of tumors in fluids obtained by puncture is also of importance. They may be found as isolated cells differing from the normal endothelium or as peculiar cell conglomerations, the latter being evidence of a much more positive character.

When a serous fluid is obtained, it is sometimes of importance to determine by the examination of this fluid whether it be an *exudate* or a *transudate* (in cases where the determination is impossible by other means). This may be done by estimating the quantity of contained proteid, determining the specific gravity and the number of leukocytes, and demonstrating the presence of a proteid which is precipitated by acetic acid.

The proteid contained in the fluid withdrawn is most accurately estimated by weighing the precipitated proteid according to the method mentioned upon p. 505 for the urine. Esbach's method, although reasonably accurate for urinary examination (p. 505 *et seq.*) is not applicable for serous fluid. A very large number of statements are to be found in literature[1] upon the proteid content of exudates and transudates. The various authors practically agree that hydropic transudates contain much less proteid than inflammatory exudates. Nevertheless most observers contend that the distinction is not sufficiently sharp, and that the rules are not sufficiently general in their application to permit of a differential

[1] Compare for example Vierordt, *Daten und Tabellen*, 1888; Bernheim, *Virchow's Archiv*, vol. cxxxvii., and others.

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diagnosis between exudates and transudates. Runeberg,[1] however, contends very emphatically that the proteid content in serous fluid is of distinct diagnostic significance. He believes that the difficulties which have thus far militated against the diagnostic importance of the proteid content mainly depend upon the fact that various observers have contrasted merely inflammatory with hydropic affections, whereas in reality the distinction must also be made between (*a*) congestion transudates and (*b*) hydremic transudates. Besides, these authors have not heeded the possibility of a combined origin for these exudates, and have not taken the trouble to diagnose such causes. Runeberg considers that his experience shows that the estimation of the proteid content of the fluid is very essential in the diagnosis of such combined forms. He determined the proteid content as from 4 to 6 per cent. in inflammatory exudates (including tuberculosis and carcinoma of the serous membranes); whereas the proteid content in pure congestion transudates varied between 1 and 3 per cent., and in pure hydremic transudates from 0.1 to 0.3 per cent., scarcely ever exceeding 0.5 per cent. These figures may be employed in the diagnosis of fresh effusions, without any further criticisms; but in order to prevent incorrect conclusions it should be remembered that the proteid content falls after a time in old congestion transudates where they are under high pressure, where they are undergoing absorption, or where changes in the serous membranes, nearly related to inflammatory changes, are developed under the influence of chronic congestions (connective-tissue sclerosis, endothelial disintegration). In such cases we are in reality dealing with combined forms, and their occurrence will explain most of the apparent contradictions to the above-mentioned rules.

These combined forms are naturally much more difficult to classify; but as a matter of fact the proteid content of the fluid really enables us at least frequently to analyze clinically such a disease picture, and is especially valuable in the determination of the proper therapy. Alterations of the proteid content during the same clinical observation may furnish valuable conclusions. For example, they may suggest the appearance of a carcinomatous peritonitis as a complication of a portal stasis produced by carcinoma, or, again, the addition of congestion to a pure renal hydrops. The great difficulty in practically employing these important facts is the complexity of an exact quantitative proteid estimation; it is too difficult for bedside work. To overcome this difficulty Runeberg has adopted a method which, although it does not estimate the proteid content exactly, at the same time is sufficiently accurate for the purpose in view. He adds a few drops of nitric acid to the withdrawn fluid in a reagent glass, and then judges by the shape and consistence of the precipitate. In exudates which depend upon a local affection of the serous membrane (inflammation, tuberculosis, carcinoma), the precipitate forms thick, heavy plaques which quickly sink to the bottom of the glass; in congestion transudates,

[1] "Von der diagnostischen Bedeutung des Eiweissgehaltes in pathologischen Trans- und Exsudaten," *Berlin. klin. Woch.*, 1897, No. 33.

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abundant large flocculi, which ordinarily sink to the bottom also, but which are more loosely and more lightly precipitated; in pure hydremic transudates, merely a decided opalescence or small, loose flakes which float for a long time in the fluid. Evidently a certain personal experience, acquired from sharply defined cases, is essential for correctly differentiating the mixed forms. Runeberg also emphasizes in the differentiation, in addition to the proteid content, the reaction with acetic acid (see below).

The *specific gravity* of serous fluids is approximately proportional to the proteid content, and may therefore be utilized for the approximate percentage of the latter. According to Ruess,[1] the following figures represent the relation between the specific gravity and proteid content:

Specific gravity.		Proteid content.
1018	higher than	4.0 per cent.
1015	lower than	2.5 "
1012	" "	1.5 to 2.0 "
1010	" "	1.0 to 1.5 "
1008.8	" "	0.5 to 1.0 "

An ordinary urinometer (p. 451) is sufficiently accurate and the most convenient instrument to employ for estimating the specific gravity. A sufficient quantity of fluid is obtained, before the needle is withdrawn, by repeated suction and emptying of the syringe. If necessary the specific gravity may also be estimated from a smaller quantity of fluid by the pyknometric method described for the blood at p. 612, or by Hammerschlag's method, although the latter procedure is more difficult.

It is probable that on the number of leukocytes or the quantity of their products of decomposition in a fluid obtained by aspiration depends the amount of contained proteid which is precipitated by acetic acid. The demonstration of this proteid was first shown by Primavera and Rivalta [2] to possess a certain importance for the differentiation between exudates and transudates. An abundance of this proteid is evidence in favor of an inflammatory exudate and against a transudate. Rivalta's test is performed in the following manner: An exceedingly dilute aqueous solution of acetic acid (2 drops of glacial acetic acid to 200 c.c. of water) is prepared, and a drop of the fluid to be examined is allowed to fall into this solution from a glass rod. If the fluid contain a considerable quantity of the substance in question, the drop immediately sinks to the bottom of the acid solution, producing a turbidity resembling a cloud of cigar smoke. If the questionable substance be absent no turbidity appears; if present in slight amount the cloudiness is very slight and of gradual development. Following the suggestion of Paijkull, Runeberg demonstrates the presence of this substance simply by the addition of a few drops of acetic acid to the fluid. In inflammatory exudates a more or less marked turbidity appears, while in transudates this is very slight or entirely absent. The nature of this substance is still under discussion. Rivalta formerly believed it to be nucleo-albumin, but now [3] regards it as a mixture of

[1] *Cf.* Vierordt, *Daten und Tabellen*, 1888. [2] *Riforma med.*, April, 1895.
[3] *Policlinico*, 1904.

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euglobulin (paraglobulin) and pseudoglobulin. Umber[1] thinks it is a mucin, and designates it as serosamucin, while Stähelin[2] arrives at the conclusion that it is related more closely to the globulins than to the mucins. It would seem that the different authors are not working with the same substance, since Rivalta, for example, expressly states that it is soluble in a slight excess of acetic acid, while Umber declares the opposite to be the case.

Another important criterion for the inflammatory origin of a serous fluid is its content of fibrin ferment, which is manifested by the spontaneous formation of fibrin either before or after the withdrawal of the fluid.

The presence of *biliary pigment* in the fluid may also be important, particularly so when the biliary passages perforate into the peritoneal or pleural cavities. In addition to the characteristic yellowish-brown or greenish discoloration, the reactions for the biliary pigments may be demonstrated by the same methods as those employed for the urine.

For a *bacteriologic examination* of aspirated fluid the culture method to be recommended, because with serous fluids an ordinary microopic examination rarely furnishes a positive result. With a purulent udate, except with purulent tuberculosis, the ordinary stick or streak culation frequently furnishes positive results; but with serous exues, which contain very few micro-organisms, a much larger amount of d must be employed for inoculation, just as in the bacteriologic examion of the blood (see p. 652). A number of drops, from 5 to 10, may quirted from the syringe directly into culture tubes with agar-agar in bouillon. Plate cultures may be employed, as is mentioned in examination of the blood. In regard to the technic of culturing, text-books on bacteriology should be consulted.

as as well as fluid is sometimes withdrawn in punctures; for ex-, in abdominal punctures when the point of the needle penetrates omach or the intestines, in pyopneumothoraces, and in abscesses contain gas. This finding may have some diagnostic signifibut only when we are sure that the gas was aspirated through dle and that it was not admitted through an imperfect fitting of inge.

ddition to the determination of the presence of fluid collections, as their peculiarities and characteristics, exploratory punctures cially important in order to decide upon the spot for attempting tic punctures. Immediately before any therapeutic puncture we lemonstrate the presence of fluid by an exploratory puncture it the spot to be selected, and should demonstrate the free of the puncture needle in a cavity. Otherwise we run a causing some injury in a therapeutic puncture with the coarse more injurious instruments should we penetrate, for example, sions of the lungs or heart, or in abdominal punctures an overintestinal coil.

[1] *Zeits. f. klin. Med.*, vol. xlviii., parts v. and vi., p. 364.
[2] *Münch. med. Woch.*, 1902, p. 34.

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CYTODIAGNOSIS.

A method of investigation rejoicing in the promising title of cytodiagnosis has recently been introduced by French writers, particularly by Widal and his pupils. It consists of the study of the character and number of the cellular constituents of exudates and transudates, and of subsequent deductions from these studies as to the nature of the fluids.

The sediment obtained by gravity or centrifugation is either subjected to direct microscopic observation, or when greater accuracy is required, is utilized for the preparation of cover-glass specimens. In such dry preparations the relative numbers of the different varieties of cells may be observed, particularly of the leukocytes. For this purpose the movable stage or Ehrlich's ocular diaphragm (p. 644) may be employed.

If the exudate coagulate rapidly soon after its withdrawal, Widal advises that the coagula be broken up by agitation with glass beads before centrifugation. It is doubtful whether accurate results are obtained by this procedure, since a great many of the cellular elements will always remain embedded in the coagula. Instead of utilizing this procedure, the writer has several times prevented the coagulation of the fluid by aspirating it into a syringe half-filled with ammonium oxalate solution or leech infusion. The ammonium oxalate solution consisted of 2 parts of ammonium oxalate to 1000 parts of normal saline solution. The leech infusion was prepared by cutting the head of a leech into small pieces and rubbing them up in a mortar. This mixture was then diluted with cold normal saline infusion, allowed to stand for an hour, during which time it was frequently stirred, and finally filtered. Instead of this infusion, physiologic saline solution to which a small amount of hirudin (1 mg. to a few cubic centimeters) has been added may also be employed. The employment of these agents preventing coagulation is to be particularly recommended when the sediment is obtained by gravity rather than by centrifugation, since in the necessary interval some of the leukocytes may become disintegrated and be caught in the fibrin network.

The morphologic elements demanding our attention in cytodiagnosis are: erythrocytes, the various kinds of leukocytes, endothelial or epithelial cells, and the cells of tumors.

The microscopic study of the erythrocytes furnishes but little additional information to that obtained by a macroscopic inspection of the fluid. The presence of blood may be demonstrated also by chemical means (for methods employed see section upon Urinary Examination), but the microscopic demonstration is a more refined diagnostic procedure. Large quantities of blood are found in carcinomatous, tubercular, and nephritic exudates, in the transudates of marked venous congestion, and in all of the hemorrhagic diatheses.

The literature of cytodiagnosis is already quite extensive, and the greatest discussion has been in reference to the leukocytes contained in the fluid. It is to be supposed *à priori* that a large number of leukocytes speaks for an inflammatory origin of the particular fluid, and that the number of leukocytes is proportional to the intensity of the inflammation, as may be easily verified by studying the occurrence of suppuration in any serous exudate. Cytodiagnosis, however, has gone much farther, and just as certain blood-pictures have been accepted as characteristic of certain diseases of the blood and hematopoietic organs, definite cytodiagnostic formulas, based upon the nature and relative numbers of the leukocytes contained in the fluid in a cavity, have been suggested for the diagnosis of the underlying disease process. This is particularly noticeable in the attempts that have been made to differentiate tubercular exudates, non-tubercular exudates, and the exudates of malignant tumors, from the relative numbers of the polynuclear and mononuclear elements.

It would seem that these efforts have gone too far, and that the empiric or statistic findings have not been tested by our knowledge of general pathology. Although we know that the emigration of polynuclear leukocytes is one of the essential characteristics of inflammation, it has not yet been decided whether

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mononuclear cells or leukocytes ever pass out of the blood-vessels. It is generally supposed that by far the greater number of the mononuclear cells are the result of regenerative processes which are associated with and consecutive to the inflammation, and that they originate in the lymphoid tissue which is found disseminated throughout almost all of the viscera.

If we abandon the crude empiric or statistic standpoint from which the question of cytodiagnosis is usually considered, and which leads to erroneous conclusions because it does not lay sufficient stress upon the different stages and degrees of the inflammatory process, it would seem that a large number of polynuclear cells is indicative of marked and recent inflammations, while a considerable quantity of mononuclear elements signifies either a milder degree and later stage of an inflammation or the presence of a non-inflammatory process.

Accepting this general view, we shall readily understand that a preponderance of mononuclear cells has generally been found in tubercular exudates. This is explained by the fact that these tubercular exudates are usually of insidious development and accompanied by a slightly marked inflammatory process. Many of them may be transudates resulting from local disturbances of the circulation, in which there is an admixture of the mononuclear cells of the tubercular proliferation. In this connection it should not be forgotten that the tubercle is not an inflammation, although it may excite a more or less marked inflammation in its vicinity. It has also been established that acute tubercular inflammations of the pleura and of the peritoneum may furnish exudates in which there is a preponderance of polynuclear cells. Upon the other hand, in the later stages of non-tubercular inflammations of the serous membranes, it is undoubtedly true that the mononuclear cells are in the majority.

It consequently seems to the author that the predominance of either variety of cell in an exudate is less indicative of the etiology of the inflammation than it is of its stage and severity. It is a fact that mononuclear cells predominate in the majority of pleuritic inflammations of unknown origin; but if we assume from this that tubercular pleurisy is much more frequent than was formerly supposed, we base our assumption upon something which remains to be proved, and which, in fact, cannot be proved—namely, that a preponderance of mononuclear cells is pathognomonic of tubercular exudates. What is true of pleural and peritoneal exudates is also true of exudates in other cavities, and particularly so of the cerebrospinal fluid.

A great obstacle to the utilization of cytodiagnostic findings is that the fluid obtained by aspiration by no means accurately represents the characteristics of the same fluid within the body and at the moment of its origin. It is well known that the cellular elements of a fluid may be so influenced by sedimentation within the body that a serous fluid may be obtained by exploratory puncture, although a complete evacuation of the accumulation might demonstrate its purulent character (see footnote, p. 709). Since the various kinds of leukocytes sediment with different degrees of rapidity dependent upon their variations in size, it also follows that we cannot always depend upon their relative numbers as determined by a study of the evacuated fluid. The excessive formation of fibrin may also modify the findings, as a variable number of leukocytes are required to produce coagulation and still others are caught in the fibrinous masses, and it is by no means necessary that all varieties of the leukocytes should be implicated in the same proportions.

All these reasons lead the author to regard the conclusions obtained by cytodiagnosis as very deceptive, and to state that they should never be employed as the sole foundation of a diagnosis. In his opinion the establishment of rigid cytodiagnostic formulas is as much opposed to our knowledge of general pathology as it is to the first commandment of clinical diagnosis—that we should diagnose from all of the symptoms and never from a single one. Cytodiagnosis, like many other modern methods of investigation, rejoices in a loud-sounding title, which causes us to forget that it has to do only with the establishment of a single symptom, and it has consequently awakened false hopes which will never be realized. These conclusions have been forced upon the author both by his

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own experience and also by the contradictory results obtained by means of cytodiagnosis as they have been published.[1]

We must be equally cautious in basing conclusions upon the presence of endothelial or epithelial cells in the fluids obtained by aspiration. Their presence is indicative only of the desquamation of these elements, and such desquamation may be due to the most varied influences. They are found unassociated with leukocytes chiefly in transudates, although they may also occur in inflammations, particularly when the process is not sufficiently acute to produce large amounts of fibrin, which would destroy or entangle them in its meshes. We must also remember that the differentiation of certain forms of mononuclear leukocytes from isolated endothelial cells is by no means certain with the methods of investigation now at our command, particularly since the cells in an exudate are subject to manifold changes produced by osmosis and other causes.

The demonstration of specific tumor cells is very important for the diagnosis of exudates dependent upon the presence of malignant growths. They may sometimes be recognized when they are present in large numbers and when their mutual relations are undisturbed, but we must not forget that they are sometimes isolated; and that, upon the other hand, normal endothelial cells may be held together by fibrin, causing them to simulate particles of tumors. Isolated tumor cells may occasionally be recognized by their abnormal size and manifold outlines, but their differentiation from normal endothelial cells is nevertheless most uncertain.

OSMOTIC PRESSURE OF FLUIDS OBTAINED BY PUNCTURE.

The osmotic pressure of these fluids is obtained in a manner similar to that employed for the blood (see pp. 546 *et seq.* and 667). This method of study has not been crowned by practical results. Ketly and Torday[2] state that exudates and transudates cannot be differentiated by variations in their osmotic pressure. It may be said, however, that the lower the osmotic pressure of an exudate or transudate as compared with that of the blood, the greater is the likelihood of the speedy absorption of the fluid. In reference to the freezing-points of these pathologic fluids, it may be stated that they are exceedingly variable, and may be far above or below that of the blood. This is due not only to the initial differences in the qualities of the particular fluids, but also to the different stages of osmotic interchange at which they are obtained.

Despite Prof. Sahli's logical objections to the clinical value of cytodiagnosis, the editors insert in the following a note which was prepared by Dr. Musgrave for the translation of the last edition.

DIAGNOSTIC VALUE OF PLEURAL FLUIDS.

In the last three years many investigators in Europe have done a large amount of work with reference to determining the cause of pleural effusions, and two new and valuable methods of investigation have been published.

In 1900 Widal and Ravaut published the results of their researches based upon the differential enumeration of the white blood-corpuscles and endothelial cells found in the sediments of serous fluids, and as a result have formulated the following laws, or so-called cytologic formulas:

1. A predominance of lymphocytes means a tubercular effusion.
2. A predominance of polynuclear leukocytes means an effusion of an acute infectious origin.
3. A large number of endothelial cells, occurring especially in sheets or plaques, means a mechanical effusion or transudate.

They also divide tuberculous pleurisy into two classes: the primary form, in which there are no discoverable lesions of the lungs, and the secondary form, resulting from an underlying tuberculous lung focus.

[1] See the comprehensive presentation of Brion, *Centralbl. f. allg. Path.*, vol. xiv., p. 609, 1903.

[2] *Arch. f. klin. Med.*, vol. lxxix.

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In the secondary variety the lymphocytic formula, as seen in the primary form, does not usually hold good, but instead the sediment is almost entirely composed of necrotic cells and detritus. Of the distinguishable cells, a large propor-

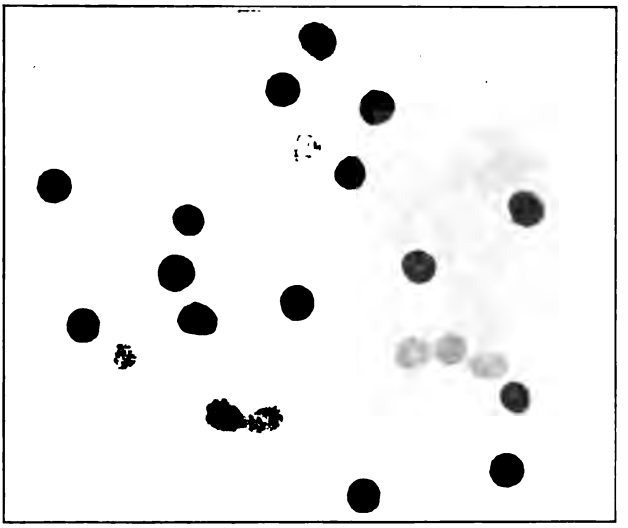

FIG. 269.—Lymphocytosis: Case of primary tuberculous pleurisy (Percy Musgrave; photographed by L. S. Brown).

tion may be polynuclear. This difference is probably due to a mixed infection. Of the acute infectious varieties, they note those due to the pneumococcus, streptococcus, and staphylococcus. In the pneumococcus variety they described the occurrence of considerable numbers of large cells having phagocytic prop-

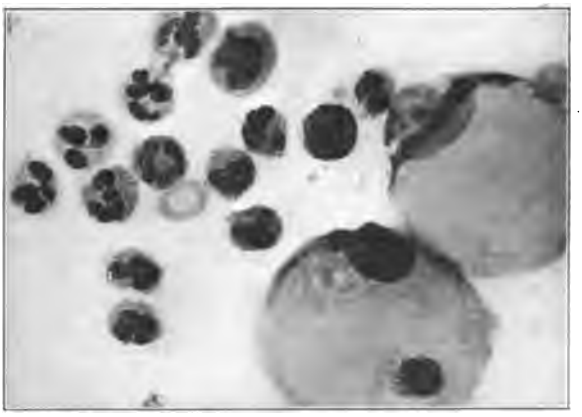

FIG. 270.—Acute infectious pleurisy, showing polynuclear leukocytes and large phagocytic endothelial cells (Percy Musgrave; photographed by L. S. Brown).

erties. In the streptococcus variety these cells are comparatively few in number, and the percentage of polynuclear leukocytes is greater.

These formulas have been extensively investigated by numerous observers, especially in France, and to a lesser extent in Germany, and have in the main

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been found correct. Certain modifications, however, must be taken in consideration. The chief of these modifications, which has been pointed out especially by Naunyn, is that lymphocytes may also predominate in transudates of long-standing or near the end of an acute infectious case where the process is subsiding or where the infection is a very mild one. The specific gravity and the amount of albumin aid us in eliminating the first mentioned of these conditions, and the clinical evidence usually gives a clue to the nature of the process in the second. Both of these conditions are rarely met.

In the first week or ten days of a tuberculous effusion polynuclear elements may predominate, but these rapidly disappear and give place to a purely lymphocytic formula.

The phagocytic cells mentioned above as occurring especially in the cases of pneumococcus origin are of very large diameter as compared with the other elements occurring in the sediment, and the writer has frequently found that they measure as much as 20 μ. These cells frequently contain partly digested polynuclear and other cellular elements, which can easily be seen included in their protoplasm. Their occurrence in large or increasing numbers is usually a good prognostic sign; and their absence, together with the presence of bacteria, is usually a warning of approaching empyema. From the observations which the

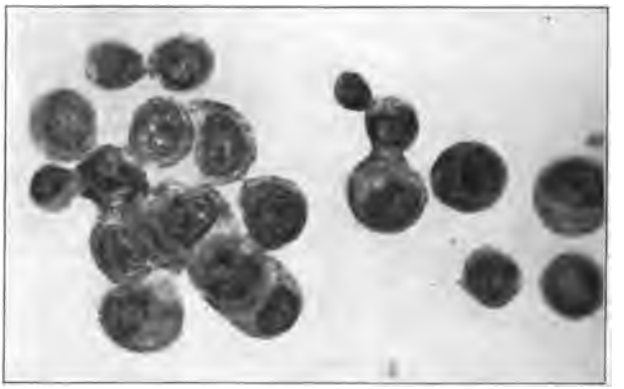

FIG. 271.—Endothelial cells from a pleural transudate due to cardiac disease (Percy Musgrave; photographed by L. S. Brown).

writer has been able to make, the presence of polynuclears in a proportion of over 90 per cent., and the presence of bacteria in most cases, precedes an empyema by only a few days.

Little need be said of the formula for the transudates except that the diagnosis is occasionally complicated by the presence of a considerable amount of blood, which raises the specific gravity and the amount of albumin. This blood is usually accidental.

In addition to these three classes of fluids we have the cancerous cases. These are difficult to diagnose, since the majority of pathologists agree, the writer thinks, in saying that it is difficult to differentiate the so-called cancer cells from endothelium, unless actual pieces of the cancerous tissue can be isolated. The iodin reaction (showing glycogenic degeneration) was thought at one time to be suggestive of the cancer cells, but this reaction is very commonly seen in the endothelial cells of simple transudates and in other conditions where endothelium is found.

The few cases of cancer that the writer has been able to study have shown a high specific gravity, 1018 or over, and an albumin content of over 1.5 per cent. The sediment has shown many more endothelial cells than are found in the tuberculous cases, and these cells are often found in plaques, which is very rare

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in tuberculosis. There is usually a much larger number of lymphocytes proportionally than in the simple transudates.

In addition to the above method of investigating pleural fluids, to which the writers have given the name Cytodiagnosis, Jousset has published a new procedure for the detection of tubercle bacilli in these fluids, to which he has given the name of Inoscopy, and which is of value in some cases.

The **technic of cytodiagnosis** is as follows: The fluid should be drawn with the usual aseptic precautions into sterilized flasks or tubes. If it is already clotted when received for examination, it should be shaken and stirred with a glass rod until the clot is thoroughly contracted, and the clot, or all clots of large size, should be removed. This is necessary because some of the cellular elements are entangled in the fibrinous meshes, and by contraction of the clot are set free. Widal and Ravaut accomplished the process of defibrination by placing sterilized glass pearls in the flask and shaking the fluid thoroughly. This the writer does not believe is essential for an accurate differential estimate of the cellular elements. The fluid is then placed in centrifuge tubes and centrifugalized for five minutes, at least. The supernatant fluid is decanted gently at first, and when only a small amount remains the tube is inverted for a few seconds. A few drops will adhere to the side. The sediment is stirred thoroughly with a small platinum loop, the sides of the glass being rubbed to remove adherent portions. When the sediment is thoroughly mixed with the few drops of fluid remaining after decantation, a drop of the mixture is removed with the platinum loop, and a cover-slip smear made. This is allowed to dry spontaneously or by gently heating the preparation. (Heating at the boiling-point will spoil the preparation.)

Many methods of fixing and staining these preparations have been used by the various investigators. Alcohol, ether, chloroform vapor, and osmic acid have been used as fixative agents. Ehrlich's triple stain, hemotoxylin and eosin, methylene-blue and eosin, Löffler's blue, and a great variety of other stains have been employed. Some writers have maintained that careful differential staining is not essential, but from the writer's observations he cannot agree with them, for the reason that careful differential staining of the protoplasm and granules of the polynuclear elements is essential, since the nuclei of these elements are often globular when the cell is degenerated, and they may be mistaken for lymphocytes.

The method of staining which has given the best results in the hands of the writer is practised as follows:

Cover the preparation with a staining fluid composed of:

Wright's blood-stain	3 parts;
Pure methylalcohol	1 part.

Allow this mixture to remain on the preparation twenty to forty-five seconds, then dilute it with 8 or 10 drops of water, and allow it to stand one to two minutes. Wash very gently, preferably by flooding the slide with a dropper. Do this four or five times, allowing the water to remain on the slide a few seconds each time. Vigorous or forcible washing will destroy the film and spoil the preparation. Dry the preparation by holding it between the thumb and forefinger and waving it through the Bunsen or alcohol flame. Do not attempt to blot the preparation or heat it above the temperature which the fingers will bear. Mount in xylolbalsam and examine with an oil-immersion lens.

Inoscopy is practised as follows:

1. The fluid should be drawn with aseptic precautions into sterilized flasks (Erlenmeyer flasks preferably). At least 100 c.c. should be taken, although results may sometimes be obtained with much smaller amounts. Allow the fluid thus taken to clot.

2. Shake the fluid gently to contract the clot as much as possible and then wash it, on a piece of sterile linen or fine gauze wrapped over the end of a funnel, until all the serum is washed away.

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3. Remove the clot or clots with a sterile spatula, and place them in a small flask with sufficient of the following fluid to digest them:

Pepsin		2 gm.;
Pure glycerin,		
Strong HCl	āā	10 c.c.;
Sodium fluorid		3 gm.;
Distilled water		2000 c.c.

The necessary amount of this fluid will vary, of course, with the size of the clot to be digested, but in most cases 20 or 30 c.c. are sufficient. A freshly prepared pepsin (HCl solution) apparently serves as well as the above fluid, but will not keep on standing as the above fluid does.

4. Place the above preparation in the incubator or oven until the clot is digested. A temperature of 37° C. for two or three hours will suffice, but the time is shortened if it is kept at a temperature of 50° C.

5. When the clot has disappeared, pour the mixture into centrifuge tubes and centrifugalize for five to ten minutes. Decant the supernatant fluid as described under Cytodiagnosis.

6. Make a cover-slip preparation and stain it for tubercle bacilli. Care, however, should be taken not to decolorize too long—one-half to three-quarters of a minute with Gabbet's solution is sufficient. Dry and mount.

The majority of the bacilli found by this method are shorter and broader, as a rule, than the tubercle bacilli usually seen in sputum, and some are paler red, but all forms occur. These bacilli may occur singly or in groups. The largest part of the sediment consists of undigested nuclei and a small amount of detritus.

Observations[1] made by the writer at the Laboratory of the Massachusetts General Hospital on 72 cases examined with reference to physical properties, albumin, animal inoculation, inoscopy, and cytodiagnosis resulted as follows: Fifty-one cases were classed as tuberculous; 46 of these were classified as primary and 5 as secondary (bacilli in the sputum). Of the 46 secondary cases, 50 per cent. (23 cases) were proved tuberculous by one or more tests—viz., guinea-pig inoculation, inoscopy, or operation. Animal inoculation was positive in 15 cases (32 per cent.), and inoscopy in 10 out of 22 cases examined by this method. One case was proved at operation. In 5 of the 23 unproved cases the examination of the fluid probably led to faulty conclusions. There were 12 acute infectious cases, all showing a predominance of polynuclear leukocytes. Six followed pneumonia, 1 infection of the lung, 3 started from unknown sources, 1 from trauma, and 1 from abscess of the liver. There were 5 cases of simple transudate; 4 due to cardiac disease and 1 to nephritis. There were 3 cancerous cases; 2 positive and 1 doubtful, all following cancer of the breast.

As a result of the study of this subject the writer thinks that we are justified in coming to the conclusion that routine and systematic examination of pleural fluids will aid us greatly in diagnosis and in determining the etiology of pleurisy. Of the methods of which he has spoken, cytodiagnosis is the only one which can easily be employed clinically, for the reason that animal inoculation, inoscopy, and culture methods can be employed only in the laboratory. Cytodiagnosis is not accurate in every case any more than any other clinical method, but the writer thinks that he is justified in saying that it is sufficiently accurate, especially when taken in conjunction with the history and bedside examination, to justify its use as a routine procedure. In some cases it has not only been of more than probable diagnostic value, but has given us positive and signal results. Routine examination of pleural fluids will not only enable us to study the question of pleurisy with effusion with reference to etiology and diagnosis, but will establish a basis upon which accurate prognostic statistics can subsequently be based.—Percy Musgrave.]

[1] *Trans. Mass. Med. Soc.*, 1904.

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DETAILS OF EXPLORATORY PUNCTURES IN DIFFERENT DISEASED CONDITIONS.

PLEURAL EXPLORATORY PUNCTURES.

The most important rules for aspirating the pleural cavity in order to determine the existence of exudates or transudates are similar to those for therapeutic puncture. We should not introduce the needle too near the upper or too near the lower border of the pleura. If we puncture too low, the needle frequently only goes through the pleural complementary sides stuck together with fibrin. If we puncture too high, we are apt to penetrate the compressed lung, for the latter often causes a dulness above the level of the fluid. Naturally there are no generally applicable rules for the place of exploratory puncture, but we must follow the results of physical examination and puncture at a point where we find decided dulness, diminished or bronchial breathing, and diminished fremitus. Generally speaking, however, in typical pleural exudations we select the fifth and sixth intercostal spaces in the anterior axillary line, and the area just below the angle of the scapula behind. In a test puncture of the pleura it is especially important to palpate carefully by means of moving the needle up and down, and so determine by its mobility whether it has penetrated a large cavity, in order that we may be enabled to select the best spot for a therapeutic puncture or for the drainage of a pleural empyema, and prevent penetrating the lung and the diaphragm. (Compare the following in regard to the distinction between pleural empyemata and intrapulmonary cavities.)

EXPLORATORY PUNCTURES TO DEMONSTRATE PULMONARY CAVITIES.

It is sometimes very desirable, especially for determining upon an operative procedure, to employ physical diagnosis, and more particularly exploratory punctures, in order to demonstrate pulmonary cavities (tuberculous cavities, bronchiectasies, and abscess cavities) and to determine their exact position and size. This naturally succeeds only when the cavities are filled with purulent secretion. It is therefore advisable to undertake the puncture at a time when the patient has not expectorated for a long period. If we succeed in aspirating purulent material, but only in small quantity, it may be obtained from a bronchus affected with a catarrh. It is important, therefore, to empty as large amounts of pus as possible, and to determine with slight movements of the needle whether the latter has penetrated a large cavity, an attempt that will succeed only when the cavities are situated superficially. The contents of pulmonary cavities are usually offensive in odor; for this reason the determination of the odor of the material withdrawn by the puncture, and its identification with the odor of the sputum, are sometimes of considerable significance.

Quite as important a question, in cases where we have determined the existence of a cavity, is whether the cavity is really situated within the lung or is part of a pleural empyema, the indications for operative interference often depending upon such a distinction. The depth from which the pus is withdrawn will often be an important consideration in settling this question. We determine this depth by successive aspirations, pushing the needle little by little back and forth. The characteristics of the pus may also sometimes be employed for determining the position of the accumulation; if it is rather slimy, it is probably partly derived from an intrapulmonary cavity, at least one lined with mucous membrane (bronchiectasis). It may also be noted that the pus of abscess cavities and tubercular cavities is not slimy. A mixture of air and pus argues in favor of an intrapulmonary cavity and against a pleural empyema; provided, of course, that the physical signs of a pneumothorax are absent and that we are sure that the needle is not stopped up and the air derived from a leak in the syringe. The air must also be withdrawn at the same time and from

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the same depths as the pus, and the amount of the withdrawn pus must prove the existence of a pathologic cavity. If this is not the case, we must always consider the possibility that the air simply comes from a bronchus. The microscopic examination of the purulent fluid furnishes certain conclusions about the nature of the cavity. Such an examination is performed like a sputum examination (elastic fibers, tubercle bacilli and other bacteria, crystals, etc.) (see p. 587 *et seq.*). The presence of elastic fibers argues especially against empyema and in favor of a destructive pulmonary lesion—*i. e.*, cavity formation.

EXPLORATORY PUNCTURE OF THE PERICARDIUM.

In exploratory puncture of the pericardium, injury to the heart is best avoided by introducing the cannula almost in the sagittal plane and directed but slightly toward the median line. The puncture should be made at the left border of the cardiac dulness and to the outer side of the apex beat, in case the latter be present. According to Döbert,[1] however, there are cases in which a puncture in this location gives a negative result, and in which the fluid may be more easily reached through the fourth intercostal space to the right of the sternum. The only cases in which this procedure is applicable are those in which there is a very pronounced and undoubted pericardial broadening of the cardiac dulness toward the right. In this situation the right ventricle and auricle are uncomfortably close, and their thin walls might easily be injured by the cannula. The cannula must be cautiously introduced, and the introduction must be suspended at the moment when resistance is no longer encountered or the heart is felt striking against the instrument. Since the danger of injuring a coronary artery or the heart itself (tearing of the thin-walled portions of the heart upon the cannula, Kronecker's "co-ordination center") is not to be despised and cannot certainly be avoided, the author does not advocate exploratory puncture of the pericardium for purely diagnostic purposes. He believes it should be employed chiefly in those cases of large pericardial exudate in which there is an urgent indication for the removal of the fluid, and where a point must be selected at which the cannula may readily enter the pericardial cavity.

EXPLORATORY PUNCTURES OF INTRATHORACIC AND ABDOMINAL CYSTS.

Both in solid tumors and in those with fluid contents exploratory puncture will afford useful conclusions, in the one case by the demonstration of characteristic morphologic tumor elements, and in the other case by the demonstration of fluid and the possibility of exactly examining it.

So far as the technical details are concerned, it is almost always a good rule to puncture tumors only in those places where the tumor lies superficially according to palpation and percussion. It is not wise to puncture through the intestine, although experience has shown that such a puncture with a very fine needle will probably not cause any damage.

To illustrate the diagnostic value of exploratory punctures in *intrathoracic tumors*, the author wishes to mention the results of aspirating a lung tumor at the Bern Clinic. Abundant myelin bodies were found in the fluid extracted. They showed in this case even more bizarre shapes than are found in the sputum (see Fig. 272). Dr. Zollikoffer proved that these myelin bodies, like those of the sputum, consisted essentially of protagon (Fig. 209, *g.*). The microscopic examination of the tumor showed that these bodies were situated exclusively in the remaining bronchi and alveoli. Their large amount should perhaps be attributed to the stagnation of the secretion associated with the tumor formation. Zollikoffer obtained similar results in studying anatomic preparations of pneumonia and tuberculosis. The appearance is therefore not specific of pulmonary tumors, although in none of the last mentioned cases was there such an abundance of the myelin bodies as in the pulmonary tumors. The author considers that their presence in

[1] *Berlin. klin. Woch.*, 1904, No. 18.

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the fluid withdrawn by puncture shows surely that the aspirated part belongs to the lung, which is significant in the local diagnosis of intrathoracic tumors, and in distinguishing pleural thickenings from pulmonary infiltrations and pulmonary tumors.

Only very fine needles should be employed to puncture abdominal cystic tumors, because, if no adhesions are formed, the fluid may trickle into the abdominal cavity and, if infectious, produce serious consequences.

To prove that a cystic tumor belongs to the gall-bladder, it is necessary to determine constituents of the bile in the fluid withdrawn (the bile-tinge to the color, the chemical determination of biliary coloring matter (p. 472 *et seq.*), the microscopic demonstration of cholestrin crystals (Fig. 211, *b*).). But it must be emphasized that closed-off gall-bladders frequently contain no more biliary constituents, cholestrin persisting the longest of any of them. If the aspirating needle strikes against a hard mass, this would be of considerable diagnostic importance.

The microscopic demonstration in the aspirated fluid from echinococcus cysts of *echinococcus scolices*, brood capsules, and the laminated remains of the membranes (see Fig. 204) is of diagnostic importance. (See also the plates in Küchenmeister and Zürn's *Die Parasiten des Menschen*, 2d ed., Plate III.) With good

FIG. 272.—Myelin kernels (protagon) obtained by exploratory puncture from tumor of the lung.

fortune we can sometimes obtain from the fluid in multilocular cysts both scolices and hooks, although, on account of the small size of the bladders, the fluid can be obtained only in traces. However, in this case we should not necessarily expect a positive result, because the majority of the bladders in multilocular echinococci are sterile. Provided the cortex or investing membrane is not inflamed, the echinococcus fluid is chemically characterized by the absence of proteid content and the presence of succinic acid. (For demonstration see below.) The thickness of the fluid—*i. e.*, the viscidity of the contents—is characteristic of ovarian cysts as contrasted with parovarian cysts. Oftentimes this cannot be determined by the fluid withdrawn from a very fine needle; in such cases the chemical demonstration of paralbumin proteid is in itself sufficiently characteristic.

The demonstration of pancreatic ferment in the fluid withdrawn is of importance in the diagnosis of *pancreatic cysts*. (For demonstration see below.) H. Zeehuisen[1] obtained the following results in this connection: The positive occurrence of trypsin in the fluid supports the diagnosis of a pancreatic cyst very decidedly, as does the demonstration of a fat-splitting ferment. The negative results to both tests do not, however, argue with certainty against pancreatic cysts. Diastatic action is useless for the diagnosis, as all sorts of fluids withdrawn possess

[1] *Centralbl. f. innere Med.*, 1896, vol. xl., p. 1017.

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this peculiarity. The occurrence of tyrosin and leucin crystals in pancreatic cysts is of diagnostic interest (Fig. 211, *c*). *Hydronephroses* and other cysts which are connected with the urinary tracts are oftentimes characterized by a considerable urea content.[1] (For demonstration see below.) However, if the cyst has been shut off for any length of time, urea may be absent.

Demonstration of Succinic Acid in Echinococcus Fluid.—Hoppe-Seyler[2] describes a method of demonstrating succinic acid in the blood, employing this method also with echinococcus fluid. The method is as follows: After the fluid has been carefully acidified with hydrochloric acid, it is freed from albumin by boiling. This is necessary only when the fluid comes from inflamed echinococcus sacs, because normally echinococcus fluid is free from albumin. The clear fluid is neutralized as carefully as possible with caustic potash, concentrated over a water bath till it is somewhat thickened, and completely precipitated with alcohol (alkaline salts of succinic acid are insoluble in alcohol). The precipitate dissolved in water, filtered, and compressed may show crystals of succinic acid alkaline salts. Succinic acid may be produced by adding to the aqueous solution a mixture of equal parts of alcohol and ether, to which is added hydrochloric acid. This mixture dissolves free succinic acid. Salkowski[3] mentions the following peculiarities of succinic acid for the purpose of identification: It forms four-sided needles, its melting point is 182, it is readily soluble in water and also in alcohol, and entirely soluble in ether. If heated in a glass tube the acid melts and sublimes, being partly changed to anhydrous succinic acid. When heated on platinum the acid folliculizes and forms vapors which tend to make one cough. If neutral lead acetate is added to the aqueous solution, lead succinate, a heavy crystalline precipitate, appears.

Demonstration of Paralbumin (Pseudomucin in Ovarian Cysts).—Salkowski gives the following rules: 1. A few drops of an alcoholic solution of rosolic acid are added to a small quantity (about 25 c.c.) of the fluid, which is heated to boiling, and very dilute sulphuric acid (tenth-normal solution) is added drop by drop until a yellowish tinge indicates that the reaction of the fluid is acid. The solution is again heated to boiling and filtered. If paralbumin is present, the filtrate will be clouded. 2. The same quantity of the cyst fluid is precipitated with three times its volume of 95 per cent. alcohol, filtered, washed several times with alcohol, and then shaken up in a mixture of one volume of hydrochloric acid and three volumes of water. The tube is then heated over a wire net to boiling, allowed to cool, and Trommer's test performed with a portion of the fluid without filtrating. After boiling, a specimen is cooled off by placing the test tube in water. Should paralbumin or mucin be present, a precipitate of red copper oxid will form. For the purpose of differentiating paralbumin (pseudomucin) from mucin, it should be remembered that the former cannot be precipitated from the cyst fluid by means of the acetic acid.

Salkowski's Test for Urea in Cysts of the Urinary System.—One hundred cubic centimeters of the fluid are carefully neutralized with acetic acid and then poured into 400 c.c. of 95 per cent. or absolute alcohol. After it has been shaken and stirred and has stood for several hours, the coagulum is washed off with alcohol and the extract evaporated over a low flame. The residue is treated several times with absolute alcohol, and the extract evaporated to dryness. A few drops of nitric acid are added to the residue after it has cooled off, and it is then allowed to stand in a cool place for twenty-four hours. As a general thing the nitric acid produces only a cloudiness at first, due to the fatty acids derived from the soaps which are almost always present. Little by little nitrate of urea is thrown down as a crystalline precipitate. This is characterized by the shape of the crystal (Fig. 13), and also by the fact that when the water is drawn off with filter paper, the crystal decomposes energetically or undergoes sudden combustion. Provided the quantity of nitrate of urea is sufficient, the

[1] Traces of urea also occur in exudates and transudates.
[2] *Handbuch der physiologisch-pathologisch-chemischen Analyse*, 6th ed., 1893, p. 52.
[3] *Practicum d. physiol. u. pathol. Chemie*, 2d ed., 1900.

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remaining portion may be utilized to obtain urea itself, which is tested for with the familiar reaction for this purpose.[1]

Demonstration of Pancreatic Ferments in Pancreatic Cysts.—For the purpose of demonstrating the tryptic, diastatic, and fat-splitting actions of fluid obtained by puncture, reference is made to the procedures on pp. 421 and 425.

PUNCTURE OF THE SPLEEN.

Puncture of the spleen, such as is recommended in typhoid in order to demonstrate typhoid bacilli microscopically in the material obtained and by means of culture, is not without danger. The small wound in the spleen in itself is of no particular importance, but the capsule may be considerably torn by the aspiratory excursions, as has been shown repeatedly on autopsy. This has often led to severe hemorrhage and peritoneal symptoms. The author warns against puncture of the spleen, especially since we have at the present time in Widal's serum test a method much more simple and far less dangerous. In any case, care should be taken to perform the puncture rapidly and to see that the patient does not breathe during the act.

EXPLORATORY PUNCTURE IN APPENDICITIS.

This method in suitable cases is devoid of danger and may be of practical value. It is self-evident that it cannot be applied in cases of diffuse resistance, especially when deep and when intestines intervene. There is little chance in these cases of obtaining pus, so that the procedure would be useless, to say the least, if not dangerous. When, however, there is a well-defined tumor, over which the percussion note is dull, an exploratory puncture may be performed without danger. Under these conditions one is sure to pass through already infected tissue, so that the risk of infection is unimportant. One may in this way favor external rupture of an abscess, as the author has personally observed. It may, however, happen, even in cases where the percussion note is dull over a tumor, that the needle passes through compressed intestines lying between the abdominal wall and the tumor mass. But this does no great harm, as is shown by the cases of puncture performed for therapeutic reasons in meteorism. The author would not, however, have it understood that he recommends exploratory puncture in appendicitis as a necessary and desirable routine measure for determining pus. On the contrary, he considers that the procedure is unnecessary for one of experience. He believes that the method has a didactic value chiefly for those physicians who are not familar with the nature of appendicitis and who believe that all cases of appendicitis suppurate. It may also serve to show that quite extensive atrophies may be present in very innocent-appearing cases. Besides this, exploratory puncture may be of a certain amount of value in demonstrating pus to people who otherwise refuse operation. The negative result, of course, proves nothing.

LUMBAR PUNCTURE.

Lumbar puncture attained diagnostic importance for the first time in the hands of Quincke. This author showed that it was very easy to enter the spinal column and the dura mater by means of a small aspirating needle introduced between the vertebral arches. Fig. 273 represents the arrangement of the lumbar vertebræ of a child twelve years of age, and shows that the interval indicated by the shaded area is quite sufficient for this procedure. The spinal cord reaches only to the second lumber vertebræ, so that it escapes injury. There is also little danger of injuring the cauda equina, because the fibers are sufficiently movable to escape the cannula, and injury to a few fibers would not be productive of any serious trouble. The technic is as follows: The patient is placed on his side and the body bent forward as much as possible, so as to increase the distance between the arches. It is not well to try lumbar puncture with the patient sitting up—

[1] See Salkowski, *Practicum d. Physiol. u. Pathol. Chemie*, 2d ed., 1900, p. 162.

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at least when performed for therapeutic reasons—as certain disadvantages have been observed due to the change of pressure. Quincke recommended that the puncture be performed between the second and third or third and fourth lumbar vertebræ.

Subsequent experience has shown that the space between the fifth lumbar vertebra and the sacrum serves just as well and may even be preferable, as the morphologic constituents of the cerebral spinal fluid, such as pus cells, blood, and tubercle bacilli, tend to gravitate toward the lowest portion of the dural sac, and might escape observation should the puncture be performed higher. The fluid at times runs clear at first, to become cloudy later. For the purpose of avoiding error, it is well to count the vertebræ not only from the seventh cervical spine down, but from the sacrum up. The needle, to which an aspirating syringe is attached, is introduced under the usual antiseptic precautions in a chosen space, and, according to Quincke, a few millimeters from the median line, so as to avoid the dense ligamentum interspinosum. The point of the needle, however, is directed toward the median line. According to Quincke, the distance to the dural sac in a child two years of age is about 2 cm., and in an adult 4 to 6 cm. It is just as well to introduce the needle in the median line, and it would appear to the writer that this procedure is somewhat easier, because of the topographic relation. It should be remembered that in a child the spinal processes are short, whereas in an adult they are longer and are directed somewhat downward. It therefore follows that in children the needle may be introduced midway between the spinous processes, whereas in adults it is best to keep close to the lower margin of the upper process. In adults it may be also necessary, if the needle is introduced in the median line, to direct the point slightly upward.

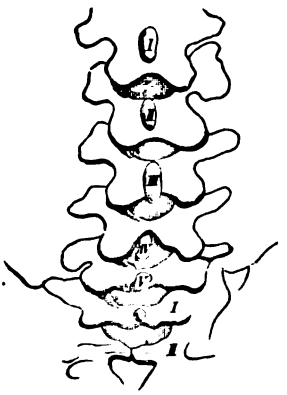

FIG. 273.—Lumbar vertebra of twelve-year-old child (Quincke).

Lumbar puncture is of diagnostic importance in two ways: It enables one, first, to determine the pressure of the cerebrospinal fluid; and secondly, to determine its peculiarties. It is self-evident that the pressure of the cerebrospinal fluid is immediately influenced by removal of a portion of it, so that tests to determine pressure should be performed before drawing off any of the fluid. An approximate idea of the pressure may be obtained by allowing the fluid to squirt out through the needle after removing the aspirating syringe. If the pressure is low the fluid appears drop by drop; if higher, the drops come faster; and if extreme, the fluid may escape in a stream. If the pressure, however, is to be measured accurately, the above experiment should not be performed. The simplest and most accurate way is to attach the needle by means of a tube filled with a 1 per cent. solution of carbolic acid, to a small mercury manometer of 1 mm. caliber. The pressure is estimated in the usual way. The portion of manometer above the level of the quicksilver, forming the connection between it and the carbolic acid tube, must, of course, also be filled with the fluid. If correct results are to be obtained, the manometer must be filled with mercury to the zero point, and must be held in such a manner that this point is on a level with the point of the aspirating needle, which is possible with ordinary manometers only when the connecting tube is of considerable length. The use of a small manometer has the advantage that when the mercury column rises, only a small amount of fluid is drawn from the dural sac, whereas the manometers of greater caliber and the ordinary water manometers have the disadvantage of withdrawing enough fluid from the dorsal sac to diminish the pressure materially. On the other hand, water manometers have the advantage of making larger excursions with low pressure. This, however, does not offset the above disadvantage.

As regards the dural pressure under physiologic and pathologic conditions, it

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may be said that in the dorsal position it is 60 to 100 mm. of water (5 to 7.3 mm. Hg.) under normal conditions, and 200 to 800 mm. of water (15 to 60 mm. Hg.) in pathologic conditions, such as meningitis and tumor of the brain.

After determining the pressure, a specimen of cerebrospinal fluid may be removed for the purpose of examination. It should be remembered that it is not justifiable in case of simple exploratory puncture to remove large quantities of the fluid, because of the dangers that supervene when the pressure is reduced below 60 to 80 mm. of water. These dangers have led certain authors to desist entirely from the therapeutic use of lumbar puncture.

The fluid is either withdrawn by aspiration or allowed to trickle out through the tubes of its own accord. This latter procedure has the advantage of withdrawing the fluid gradually, and of allowing one, by holding the tube at a certain level, to prevent the pressure approaching the danger line—*i. e.*, below 80 mm. This may readily be accomplished by holding the open end of the tube 80 mm. above the site of the puncture.

So far as the peculiarities of the fluid are concerned, it will be found that under normal conditions it is colorless, water-clear, and of a specific gravity only a little above 1000 (1003). Under such conditions it contains very little albumin and shows no distinct nucleo-albumin reaction (p. 711). An increase of the specific gravity will indicate meningitis, although a normal specific gravity may be present in this disease. Lenhartz claims that more than 0.25 per cent. of albumin also indicates meningitis; although the same author, in exceptional cases of cerebral tumor and apoplexy, has found albumin up to 1.5 to 2.25 per cent. The macroscopic appearance of the fluid is of considerable importance. A cloudy fluid indicates inflammation, as does also the presence of numerous white blood-corpuscles on microscopic examination. These are, as a rule, the cause of the cloudiness. On the other hand, the fluid may remain perfectly clear in cases of inflammation, especially in tuberculosis, and even in purulent cerebrospinal meningitis. Marked cloudiness produced by white blood-corpuscles is in favor, generally speaking, of purulent and against tuberculous meningitis. The degree of cloudiness may under certain circumstances serve to distinguish between brain abscess and cerebral meningitis, or to establish a diagnosis of a combination of the two, a point which may be of value in determining the advisability of operative interference. A clear fluid in these cases is against, and a cloudy fluid in favor of, purulent meningitis. In case of meningeal hemorrhage and pachymeningitis hemorrhagica, the fluid has been found blood-stained, a point which may be of differential diagnostic importance in connection with spontaneous cerebral hemorrhages and softening, although, of course, an intracerebral hemorrhage perforating the ventricle would be liable to cause a hemorrhagic cerebrospinal fluid. In tumors of the cord and meninges, one should observe whether the fluid contains tumor cells, although we have no positive information of their being found in such cases, and a case examined by the author with this in view furnished negative results. Still the possibility of finding cells analogous to those found in pleuritic and peritoneal exudates should be borne in mind.

It is of great importance for the purpose of diagnosis to examine a dry preparation of the cerebrospinal fluid for bacteria. In epidemic cerebrospinal meningitis Weichselbaum's Diplococcus intracellularis will be found. This was formerly confounded with Fränkel's pneumococcus, although there is little resemblance between them,[1] the former being more closely related morphologically, and so far as culture is concerned, to the ordinary staphylococci.

Furthermore, the frequent occurrence of polyarticular inflammation of the joints in cerebral meningitis is in favor of a closer relationship between the meningeal bacteria and staphylococci, for there can be no doubt that many cases of polyarticular inflammation are due to coccus infection. In tuberculous meningitis it is quite surprising that tubercle bacilli are found in the puncture fluid

[1] H. Jäger, *Zeits. f. Hyg.*, vol. xix., p. 351 This work contains photographs of the Diplococcus intracellularis.

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in over 50 per cent. of the cases. They are found most easily after allowing the fluid to settle for some time until a coagulum appears, from which a dry preparation is made (see p. 593). In cases where no coagulum forms the fluid must be centrifuged for some time, so that the bacteria will settle through the lower layers. Should the fluid contain nucleo-albumen, for instance (p. 711), it is well to precipitate this with acetic acid, then centrifuge, and hunt for the tubercle bacilli in the isolated precipitate. (See Examination of the Sputum according to Ilkewitsch, p. 596.)

HARPOONING.

For the purpose of obtaining small pieces of deep-seated tissue for the purpose of examination, harpoon-like instruments have been introduced through a cannula and then projected beyond the protecting sheath, to be subsequently drawn back into the cannula with a portion of the tissue desired. As a rule, harpooning is unnecessary, as it is usually possible to obtain sufficient tissue for an examination by using the ordinary aspirator in the manner explained on p. 208. At any rate, the harpoon will be used only in cases where the aspirate fails or where larger particles are desired for the purpose of making microscopic sections. These harpoons may be obtained from an instrument maker, or they may be improvised by cutting a notch in the stilet of an ordinary trocar. Care should be taken not to have the notch in the stilet so deep that there is danger of the bob breaking off. Harpooning is a much more severe procedure than the exploratory puncture, because for safety it is essential that the harpoon be of a certain thickness. Generally speaking, harpooning has been justly given up.

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RÖNTGEN-RAY EXAMINATIONS.

THE editors consider it advisable to insert a few illustrations which will show some of the more typical results of *x*-ray examination in cases where this method may aid in arriving at a diagnosis.

FIG. 274.—Gouty deposits in feet and hands. Note the symmetric distribution of the bony disorganization in the heads of the first and fifth metatarsal bones; the bony disorganization of the first phalanx of the index finger; also the light elliptic and circular spots in photograph, showing the destruction of bone and the deposit of urate of soda (Massachusetts General Hospital, service of Dr. Cutler).

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It is hardly within the scope of this book to do more than to explain the illustrations as briefly as possible. For all other information in regard

FIG. 275.—Normal x-ray of lumbar region. Plate on back. In order to detect a renal calculus the plate should show good definition in the lumbar vertebræ and well-defined transverse processes with detail in the soft parts. The patient's bowels should be thoroughly evacuated, and the diaphragm immobilized by the pressure cylinder or a swathe. In this plate the observer can make out the oblique lines marking the muscular masses. An absolute outline line of the kidneys can often be defined in a perfect plate. Here there is so much detail of soft parts it is almost impossible to see the kidney. This plate should be contrasted with Figs. 276 and 277 (Massachusetts General Hospital).

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to the technic, instruments, methods, and diagnostic value to be attached to the findings, we refer to text-books and to the periodicals devoted to

FIG. 276.—Calculus in right ureter. Before a diagnosis of renal or ureteral calculus is made, several plates should be taken and the findings should agree. Two plates taken on this case: calculus removed (Dr. John Elliott's case, Massachusetts General Hospital).

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the subject. In our opinion the method is useful only when its results confirm those of various other methods of examination. In a case of

FIG. 277.—Renal calculus (right kidney). The density of the shadow of this stone indicates that it is either phosphatic or oxalate. Uric acid stones cast a very faint shadow owing to their organic composition (Dr. S. J. Mixter's case, Massachusetts General Hospital).

aneurism of the arch of the aorta, or in a case of renal or bladder calculus, we may sometimes obtain evidence of as much or more value than that from any other source.

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FIG. 278.—Left pneumothorax, showing dislocation of the heart and compression of the lung. Black square is a lead marker at the level of the nipple: A, shadow of the cardiac apex; L, compressed lung (Dr. J. J. Minot, Massachusetts General Hospital).

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The orthodiagraph, a recently devised instrument, is worth mentioning. It promises to add valuable help in more accurately determining

FIG. 279.—Vesical calculus (marked by arrow).

the outlines of the heart and the arch of the aorta. The instrument has not been employed long enough to warrant criticism, but its utility seems assured.

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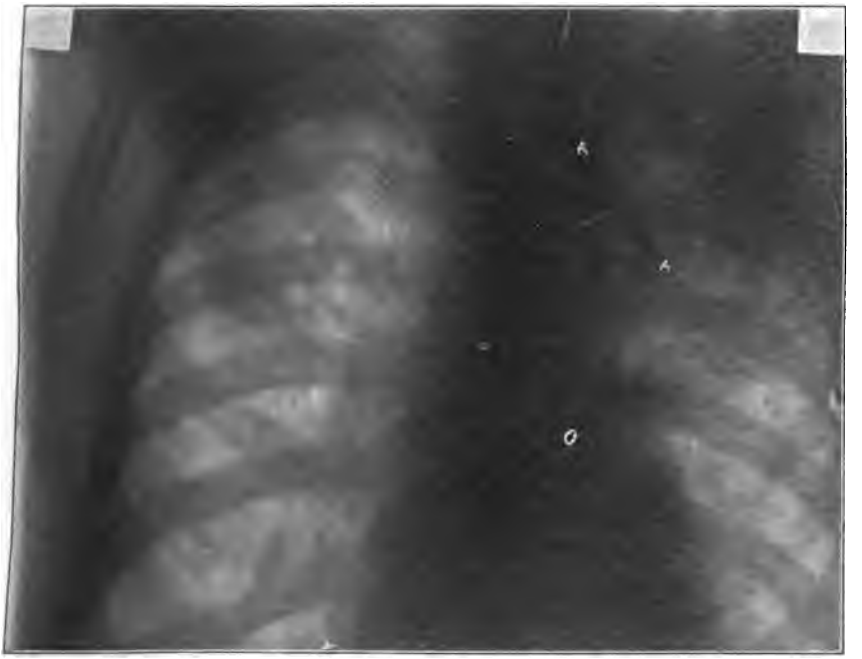

FIG. 280.—Chronic tuberculosis of both lungs: An abscess cavity in left upper lobe connecting with esophagus (A—A). The multiple foci of tuberculosis are well shown in this plate (Dr. E. G. Cutler's case, Massachusetts General Hospital).

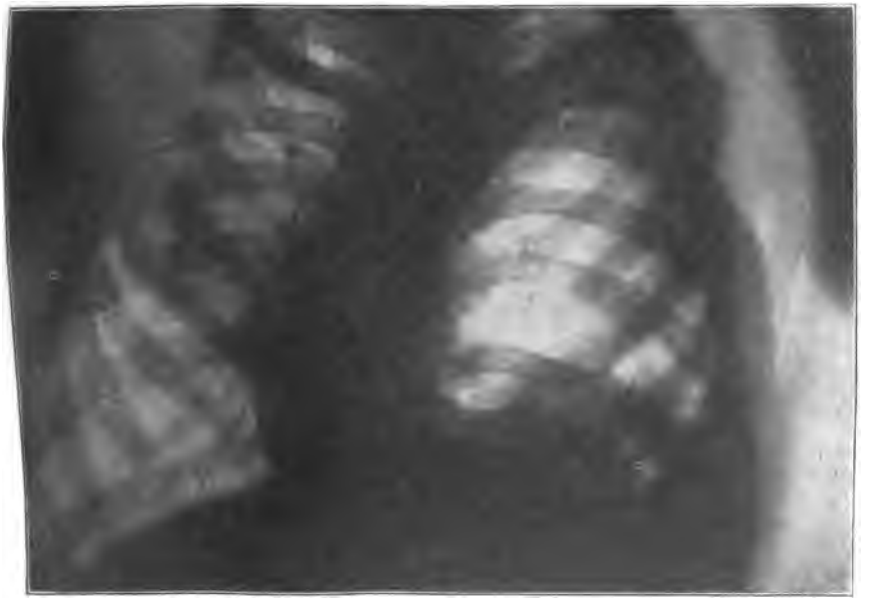

FIG. 281.—Hernia of diaphragm. Note line of diaphragm about third interspace of the right lung.

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FIG. 282.—Arthritis deformans. This plate should be contrasted with Fig. 274 (Goldthwaite, Massachusetts General Hospital).

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The illustrations have all been made by Mr. Walter Dodd, at the Massachusetts General Hospital in Boston, and we wish to take this

FIG. 283.—Large aneurism of arch of aorta (Massachusetts General Hospital)

opportunity of acknowledging our indebtedness to him, and to the courtesy of the various physicians whose cases he photographed.—ED.]

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EXAMINATION OF THE NERVOUS SYSTEM.

ALTHOUGH the technical aids to the examination of the nervous system are usually simple and require but brief notice, it is more important in this department of internal medicine than in any other to conduct the examination in accordance with a definite, logical plan. The complex functions of the nervous system make such a plan essential in order that no symptoms may be overlooked, and that the symptoms may be so grouped as to suggest the diagnosis. Such systematic procedure takes time; but it is quite simple and, in fact, often easier than in other forms of disease, since the examiner needs less technical skill. The reason why the beginner finds the diagnosis of nervous diseases so difficult is because he is not familiar with those anatomic and physiologic facts which are absolutely essential for the accurate interpretation of clinical cases.

GENERAL PART.

I. PSYCHICAL EXAMINATION.

The clinician now and then observes in his patient mental disturbances which belong exclusively to the domain of psychiatry. For a comprehensive method of examination in such a case, it is advisable to consult text-books upon that subject. In this volume we shall describe only psychical disturbances which occur so frequently in clinical medicine as to be typical. These include the various grades of depression or irritative disorders of consciousness, delirium, and disturbances of intelligence and memory.

DEPRESSED DISTURBANCES OF CONSCIOUSNESS.

The term *somnolence* (sleepiness or hebetude) is applied to the mildest grade of a depressive disturbance of consciousness. This merges imperceptibly into lethargy or stupor, and finally into *coma*, or absolute loss of consciousness.

These depressive disturbances of consciousness are observed not only in brain diseases, but also in all sorts of general affections: 1. In any lethal illness, a short time before death. 2. At the height of infectious febrile diseases, though such diseases rarely produce complete loss of consciousness. 3. In uremia, and then generally accompanied by convulsions. 4. In diabetic coma. The gradual onset is here very characteristic; the loss of consciousness usually becomes complete, and the breathing deep, slow, and labored (see p. 91). 5. In cases of poisoning, especially with alcohol, morphin, chloroform, chloral hydrate, coal gas. 6. In epileptic attacks. 7. In many hysteric attacks. 8. In focal lesions of the brain of sudden onset, hemorrhage, softening (shock, stroke), in traumatic cerebral lesions (laceration, concussion), in the different varieties of meningitis, and in the later stages of brain tumors.

The disturbance of consciousness noted in *hysteria* is distinctive. If complete, it is ordinarily called *lethargy*. It can be differentiated from the comatose con-

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ditions observed in severe brain diseases (apoplexy, uremia, etc.) because it simulates normal sleep so perfectly. Presumably it has the same psychologic genesis as that torpor of the cerebral cortex which we call sleep. The function of the cortex only is interrupted, so that those phenomena accompanying other varieties of coma and depending upon infracortical disturbances, such as stertorous or interrupted breathing, cyanosis, involuntary evacuations of feces and urine, etc., are lacking. The hysteric condition of lethargy differs from normal sleep in that the former arises and persists under conditions which would prevent or interrupt the latter. Quite a series of transitional forms may occur in the hysteric between lethargy (hysteric unconsciousness) and alert consciousness—*e. g.*, *somnambulism*, in which a person without any subsequent recollection may perform all kinds of complicated actions, influenced apparently by no ordinary motives, but by constraining ideas. During the somnambulistic condition, the memory frequently seems to be cognizant of what has occurred in previous attacks, whereas in the interval everything relating to the somnambulistic condition is effaced from consciousness (dual personality). These transitions between absent and present, or, better, between waking and sleeping consciousness, may be produced artificially in certain persons by hypnotic—*i. e.*, suggestive—influence, and are therefore called *hypnotic conditions*. Wundt regards them as limitations of consciousness; in other words, certain anatomic substrata in the brain are sleeping—*i. e.*, inhibited—whereas other areas functionate much more actively, and, in consequence of their isolation, oftentimes in peculiar fashion. A motor phenomenon frequently connected with hysteric disturbances of consciousness is the so-called *catalepsy* or *cataleptic muscular rigidity* described upon p. 751. Such hysteric (hypnotic) conditions associated with cataleptic rigidity are ordinarily designated as *catalepsy*, although the latter expression should really be limited to the condition of the motor system, and not include that of the consciousness.

IRRITATIVE DISTURBANCES OF CONSCIOUSNESS.

Delirium is practically the only one of these irritative disturbances which concerns the clinician. It means a dream-like, cloudy state of consciousness, generally associated with deranged ideas, in which the intellectual capacity is altered in a morbid, irritative way. It does not preclude a depressive condition in other provinces of consciousness, and so delirious patients may at the same time exhibit stupor. Delirium is frequently accompanied by *hallucinations* and *illusions*. We distinguish a *noisy* from a *quiet delirium*. The highest grade of the former is the so-called *maniacal delirium*. In *muttering delirium* the patient lies quietly in bed and continually mutters to himself. Delirium may be observed in any severe general condition; it is especially frequent in fever, and then it nearly always denotes a severe sickness. But we should remember that children become delirious more readily than adults, just as in fever their temperatures run higher. Many acute brain diseases manifest active delirium.

The irritative disturbances of consciousness depressive in nature which accompany hysteria and which occur especially in the so-called "hysteria major" are essentially a variety of delirium, and should be attributed partly to the influence of inhibition, as a result of depressive conditions of other districts of consciousness.

The characteristic *delirium of alcoholism* (*delirium tremens*) is for the most part a very noisy, maniacal form, and is almost always associated with hallucinations. The patient sees black forms, mice, bugs, snakes, wires running through the air, a policeman, etc. These appear-

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ances have been attributed to the presence of scotomata in the visual field; but if this explanation were correct, the condition would be one of illusions, or of errors in sight, not of real hallucinations. The typical tremor which almost always accompanies delirium tremens may decide the diagnosis.

Most cases of quiet delirium which are noticed in patients seriously ill and which are accompanied with the so-called *carphologia* or *floccillation* render the prognosis rather grave. The patient lies completely oblivious of his environment, continually picking at the bedclothes, or making motions with his fingers as if he would pick off flakes or scales. Carphologia, a symptom which generally precedes death only by a short time, probably depends upon hallucinations.

DISTURBANCES OF THE INTELLIGENCE.

These may be independent of any of the disturbances of consciousness, although they are frequently associated with them. The patient's history or a previous acquaintance with him is all there is to aid the

FIG. 284.—Cretinismus (Dr. F. C. Shattuck, Massachusetts General Hospital).

FIG. 285.—Cretinismus. Same case as Fig. 284 after treatment (Dr. F. C. Shattuck, Massachusetts General Hospital).

physician in appreciating the milder grade of disturbances of the intelligence. On the contrary, the severer grade (*stupidity*) and the highest grade (*imbecility*) are evident enough from the facial expression and deportment of the patient.

Any of the incurable maladies may be accompanied by the milder

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disturbances of the intelligence—*e. g.*, *heart disease, nephritis.* Severe disturbances—*i. e.*, marked *stupidity* or distinct mental disease—on the contrary, point with much probability to disease of the brain, and, if we except imbecility, they point to *brain tumor, paralytic dementia, multiple sclerosis,* or the after-effects of acute focal lesions of the brain, such as *hemorrhage and softening.* In other cases *imbecility* is the expression of a congenital or early acquired cerebral anomaly (*idiocy* or *cretinism*). The disturbance of intelligence which is observed in *myxedema,* spontaneous or operative (such as after removal of a goiter), deserves special mention. This condition is either very closely related to or identical with cretinism. Such patients exhibit sometimes mild mental disturbance, such as slowness of thought; sometimes however, a much more serious trouble bordering on real imbecility.

DISTURBANCES OF MEMORY.

Memory is frequently appreciably impaired in old but otherwise quite healthy persons. In other cases it may be affected under the same conditions as the intelligence. In fact, after acute lesions of the brain (hemorrhage and softening) a weakness or even sometimes a complete loss of the memory is very often noticed, and frequently it persists, although the intelligence is not necessarily affected. The so-called traumatic neuroses are very often accompanied by a failing memory.

II. EXAMINATION OF MOTILITY.

1. PARALYSIS.

To demonstrate paralysis the examiner requests the patient to perform voluntary motions either without or against resistance. If paralysis of the muscles exists, such a movement is either not performed at all (complete paralysis) or more slowly and less completely than normally (*incomplete paralysis, paresis, motor weakness*). We easily recognize severe paralysis or even motor weakness in this way. In many instances the posture of the body at rest is in itself so distinctive that we need no attempt at motion to appreciate the complete loss of power. For example, a paralysis of the eye muscles is often evident from the abnormal position of the eyeball; facial paralysis from the asymmetry of the face; paralysis of the arm from the dropping and dangling of the arm; peroneal paralysis from the drooping of the tip of the foot (equinovarus); a musculospinal paralysis from the *wrist-drop;* an ulnar paralysis from the characteristic *claw-like* position of the fingers (extension of the proximal phalanges and flexion of the terminal phalanges); a median-nerve paralysis from the abduction and extension of the thumb, simulating the ape hand. We must, however, always combine inspection with an attempt to secure voluntary movements, since to make a diagnosis it is necessary not only to determine a paralysis, but also exactly to localize it, and so we must test separately the functions of the individual muscles and muscle groups. The cranial nerve supply will be found upon pp. 826–883; the nerve supply of the muscles of the remainder

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of the body upon pp. 910–914. If the paralysis is unilateral and not very marked the examination is made easier by comparing the healthy with the affected side. In this way we can demonstrate in hemiplegia, for example, a weakness of the trapezius muscle, as well as of the respiratory and abdominal muscles of the paralyzed side, although otherwise it might easily escape the examiner's notice. The rapidity, the force, and the excursion of the movement should all be noted. An excellent method for detecting a very slight one-sided paresis of an arm or a leg is to have the patient raise both extremities as quickly as possible at the same time. The paretic arm or leg will then be plainly seen to remain behind the healthy member, and to fall more quickly from the raised position, even in cases where in testing the power by movements against

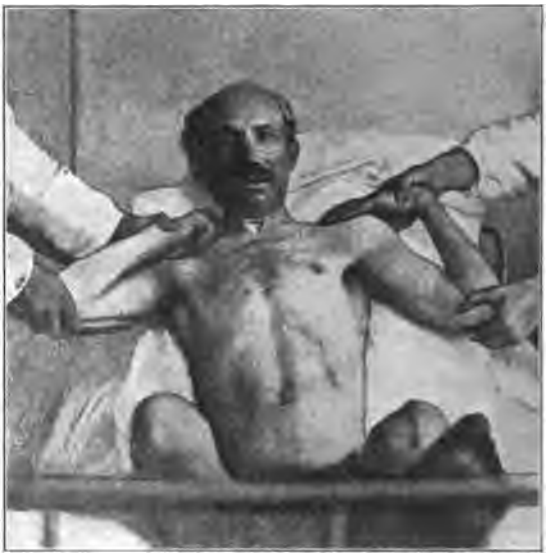

FIG. 286.—Right hemiplegia, organic; excessive flexion of the right forearm upon the arm.

opposition we can scarcely appreciate any abnormality. We can utilize the hand dynamometer to test the strength of the hand.

A mechanical limitation of movement in a joint, or pain incident to its movement, sometimes makes the movement weak, ineffectual, or almost lacking, and so is responsible for an erroneous diagnosis of motor paralysis or paresis. Therefore, before assuming the presence of a motor weakness we should particularly examine for such a condition of affairs. Of course, patients will have to be believed in regard to the influence of pain upon mobility, although they are often very unsatisfactory in their statements. They will acknowledge that they feel pain, but deny that it is responsible for the interference of motion. Frequently they are firmly convinced of the existence of a paralysis until the pain subsides under treatment and permits perfect motion. In such a case some unappreciated limitation of motion probably exists, depending upon pain. If the limitation of motility persists longer than the painful irritation, the so-called hysteric paralyses often result.

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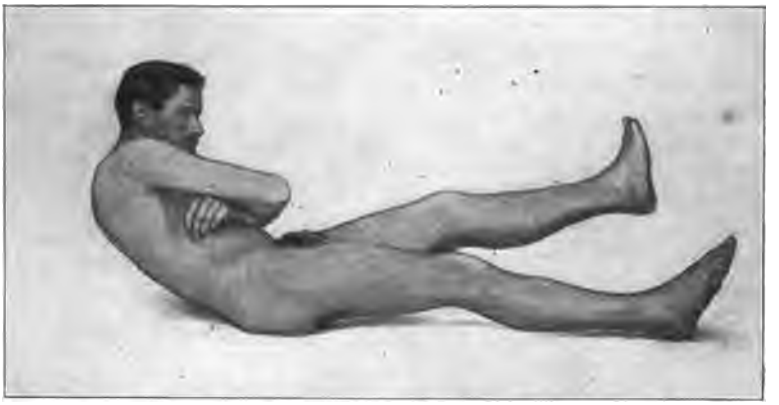

FIG. 287.—Left hemiplegia, organic (combined flexion of the left thigh and trunk); inability of the left leg to remain in contact with the floor while the patient attempts to sit up or lie down with folded arms. The legs should be slightly separated.

[Babinski has called attention to several aids in differentiating an organic from an hysteric hemiplegia. Three of these are well illustrated

FIG. 288.—Left hemiplegia, organic; sign of the platysma. The paralysis of the platysma upon the left side is evident.

in the following cuts (Figs. 286, 287 and 288) copied from his article.[1] —ED.]

[1] *Gaz. des Hôp.*, 1900, No. 52, p. 521.

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2. PHENOMENA OF MOTOR IRRITATION.

(*a*) Clonic Convulsions or Contractions; Clonic Spasms.

By these we mean involuntary muscular contractions repeated in shocks or series, generally with considerable force and rapidity, and contrasted with voluntary contractions badly co-ordinated. Thus, in spite of the great expense of power, there often results only very slight movement, because antagonistic muscles are working in opposition. An epileptic attack and the typical uremic and eclamptic spasms are types of the clonic convulsion.

Clonic convulsions are never occasioned by peripheral excitation of

FIG. 289.—Hysteria: blepharospasm. Contrast these two pictures with the picture of ptosis (Fig. 320) (Neurologic Department, Massachusetts General Hospital).

motor nerves. An irritative center whose action may be compared to that of a Leyden jar seems to be essential to set off the shock-like explosions. Clonic contractions are practically always occasioned either by a direct or by a reflex irritation of a motor center, whether it be the nucleus or the psychmotor center of the cortex.

(*b*) Tonic Convulsions; Tonic Spasms or Cramps.

By tonic convulsions are understood long-continued rigid convulsions of muscles which may change suddenly and lead to prolonged change of position or tension of the muscles implicated. Tonic convulsions may, under some circumstances, be associated with or even transformed into the *clonic* variety. Tetanus is the type of a tonic convulsion. Tonic convulsions originate in most cases from irritation of some center, and generally reflexly from a nucleus, as, for instance, in tetanus,

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strychnia tetanus, tetany, and the rigidity of the neck and back in meningitis. The well-known cramp of the gastrocnemius which is observed in health and in choleraic conditions, but whose genesis is still obscure, belongs to this group.

(c) Contractures.

When a joint becomes more or less permanently fixed from contraction of the muscles around it, so that active or passive movements are difficult or impossible, the condition is called *contracture*. The increased tension of the muscle may depend upon increased tonus—*i. e.*, an active (although reflex) contraction—so-called *active or irritative contracture;*

FIG. 290.—Same patient, showing an attempt to open left eye. Note the associated movements of the muscles supplied by other branches of the facial nerve (see pp. 750 and 863).

or it may depend merely upon a nutritive shortening of the muscle, *passive contracture.*

Active contractures are very closely related in their origin to tonic convulsions, and are to be distinguished from the latter only by being more permanent. In contrast with *passive contracture, active contracture* disappears during ether or chloroform narcosis; it ordinarily diminishes during a warm bath. Even without narcosis, *active contracture* may be overcome sometimes, though not always. Such contractures offer a somewhat elastic resistance to passive movements; they are practically always associated with increase in the corresponding tendon reflexes; every brusque attempt to overcome the contracture reflexly increases the tension; the muscles exhibit no signs of degeneration; and the contractures exhibit spontaneously a certain slow change or even a gradual transition to tonic convulsions.

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Passive contractures, on the contrary, are decidedly resistant to passive movements. They are influenced neither by narcosis nor by warm baths, and they are ordinarily associated with diminution of the tendon reflexes. If overcome by force, which is only possible by a tearing of tissue, they may disappear for a long time. This is a very rare occurrence with active contractures, except the hysteric. For the differentiation of active and passive contractures, Crocq recommends the application of the Esmarch bandage, which, like narcosis, has no effect upon passive contractures, but causes the active ones to disappear.

FIG. 291.—Tetany in an infant eleven months of age (Dr. Gaunett, Massachusetts General Hospital).

Active contractures may occur whenever the muscle tone is increased. Since the muscle tone is of reflex origin, this comes, on the one hand, from involvement of the reflex inhibitory fibers, situated perhaps in the pyramidal tracts, and, on the other hand, from increased irritability of the reflex centers. Curiously enough, active contractures fix the upper extremities in a position of flexion, and the lower extremities in extension. This agrees with the physiologic ascendancy of the muscles in question over their antagonists, and with the predominance of extensor paralysis in the upper extremities. To explain the varying relations of the flexor and extensor muscles in active contractures, Mann[1] assumes that the reflex

FIG. 292.—Tetany in an infant eleven months of age (Dr. Gaunett, Massachusetts General Hospital).

inhibitory fibers for the flexors run with the psychomotor fibers of the extensors, and the inhibitory fibers for the extensors with the motor fibers for the flexors, or, which the author thinks more prob-

[1] *Monats. f. Psychiatrie u. Neurologie*, vol. iv., 1898.

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Kernig's sign in a case of cerebrospinal meningitis. Note the contractures of ham-string muscles, the retraction of the head, the expression of suffering produced by the examiner's effort to extend the lower leg, and the pronounced emaciation (Harlem Hospital, Dr. R. G. Wiener).

Faulty method of determining Kernig's sign. The thighs should first be flexed upon the abdomen before the examiner attempts to extend the lower leg upon the thigh (see accompanying picture).

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able, are identical with them. This supposition coincides very well with the facts demonstrated by Sherrington and Herring (Physiologic Congress in Cambridge, 1892), that cortical stimulation of a muscle exerts an inhibitory influence upon its antagonists. In consequence of this, lesion of the pyramidal tract, causing a preponderance of the paralysis in one muscle group leads to the preponderance of the contracture in its antagonist. Still, a simple interruption of the reflex inhibitory tracts does not seem sufficient to explain the original of well-marked contractures. Muscle tonus and tendon reflexes, which pass along the same reflex paths, are generally increased in mere interference of spinal cord conduction with a lesion of the reflex inhibitory tracts (see p. 782); but an actual contracture appears only when a descending degeneration of the pyramidal tracts is superadded. For some unknown reason, only descending degeneration seems to be able to cause active contractures. Perhaps it may be explained by assuming that the lower end of the inhibitory paths —*i. e.*, the pyramidal tracts—even when separated from the source of the inhibition (the central apparatus), still succeeds in causing a certain inhibitory influence upon the muscle tone, an influence which disappears entirely only when the tract is degenerated down to its lowest termination in the motor ganglion cells of the anterior horns. When active contractures depend upon an anatomic lesion, and are not purely functional as in hysteria, they furnish an important sign for the diagnosis of *descending secondary degeneration of the pyramidal tracts, both in the brain and in the spinal cord* (*cerebral hemorrhage, softening, tumors, transverse myelitis, cervical hypertrophic pachymeningitis, syringomyelia, compression of the spinal cord, etc.*). The contractures may arise in a similar way from primary systemic degeneration of the pyramidal tracts (*spastic paraplegia, amyotrophic lateral sclerosis, etc.*). The mechanism of hysteric contractures is not yet fully understood, nor is the early (at times almost instantaneous) occurrence of contractures of the paralyzed extremities (contractura præcox).

Passive contractures occur wherever the insertions of a muscle are permanently approximated, so that the muscle is gradually shortened in a nutritive sense. This occurs *in mechanical fixation of joints* by bandages which have been applied for a long time in surgical affections, and in *localized atrophic paralyses—e. g., infantile paralyses.* In the latter unparalyzed antagonists dislocate the affected limb permanently in their own direction, and thereby shorten themselves, in the nutritive sense. There is still another type of passive contracture which arises from contracting connective tissue which gradually takes the place of a paralyzed and degenerating muscle and overcomes the tonus of the weaker antagonists. Here, of course, the contracture is not in the direction of the preserved, but in that of the affected, muscles.

The result of contractures is the fixation of parts which we see so frequently in certain paralyses. These deformities have been variously named according to the anomalous position of the limbs. For example, *equinovarus* in peroneal paralyses, *claw-hand* in ulnar paralysis (Fig. 293), the *ape hand* in median paralysis, the *preacher's hand* in pachymeningitis cervicalis hypertrophica. Special pathology should be consulted for more details.

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(*d*) Fibrillary Twitching (Better Characterized as "Fascicular").

These are brief chronic contractions, not of an entire muscular belly, but of mere individual groups of muscle fibers. Outside of the small muscles of the hand and face, these contractions do not produce an actual locomotion of the insertion of the muscle, but merely a peculiar trembling or wave-like rhythmic vibration of the muscular mass. The cause of fibrillary twitching is not yet explained. It appears in paretic and atrophied muscles, particularly when of nuclear or subnuclear origin (*in spinal neuritis, muscular atrophy, and bulbar paralysis*). It may also occur even without paresis or atrophy—*e. g.*, in the

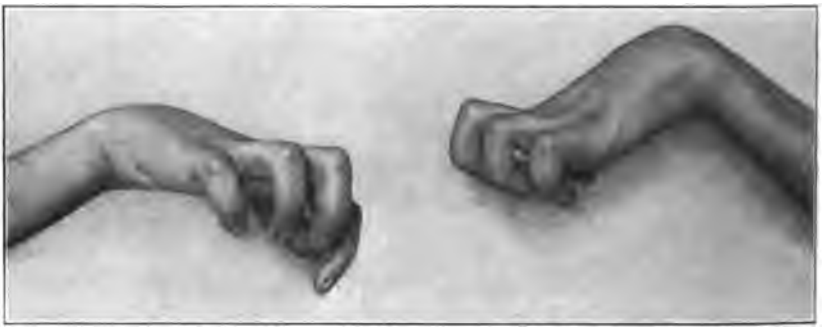

FIG. 293.—Claw-hand of ulnar paralysis.

traumatic neuroses. A certain diagnostic significance is often attributed to this phenomenon because it is an impossible symptom to simulate; but we must be very guarded in drawing conclusions, because the action of cold upon the naked body of many healthy individuals may cause fascicular contractions.

(*e*) Tremor (Trembling).

By tremor we mean rapid and rhythmic muscular contractions which are relatively but slightly pronounced. They produce movement of the insertions of the affected muscle or the skeletal parts, thus differing from fascicular contractions. There are transition forms between *clonic spasms* and *tremor*, as well as between *fibrillary contractions* and *tremor*. We distinguish between *intention tremor*, which arises only with purposeful movement, and *tremor*, which persists during rest. The intention tremor is typically observed in multiple sclerosis. It was formerly regarded as a paralytic tremor, since it was assumed that the disturbed conduction transformed the voluntary impulse into a succession of rhythmic impulses (from which, as physiology teaches, the voluntary tetanic contraction is constructed), and that the muscle lost the greater portion of the separate stimuli. From recent experience it would seem that this view can no longer be maintained. The author has observed that in cases of multiple sclerosis, where the increased tendon reflexes lead to clonic tendon reflexes (ankle clonus, knee clonus, wrist clonus, etc.), the intention tremor is often synchronous with the

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clonus. This would certainly indicate that the tremor of multiple sclerosis is nothing else than a clonic tendon or muscle reflex which is excited and maintained by the tension which the muscle experiences during the intended movement. The tremor of multiple sclerosis is consequently a spastic phenomenon, as is also true of the majority of tremors. This conception of the intention tremor of multiple sclerosis explains why it may gradually assume the character of those less regular arrhythmic contractions which appear during the course of voluntary movements and make them simulate ataxia. The so-called rest tremor is not dependent on voluntary movement, but occurs also during rest. In fact, it may be present only during rest, and may sometimes be voluntarily controlled. This variety of tremor is one of the characteristics of paralysis agitans. It is a spastic phenomenon, the cause of which is located above the circuit of the tendon reflexes, and has nothing to do with an increase of these reflexes.

To avoid overlooking tremor of any sort, we should observe patients both at rest and during purposeful movements, as well as in positions of the body which require muscular action—*e. g.*, horizontal projection of the forearm and hands with the fingers spread.

The characteristics of the well-known tremors are as follows:

1. The peculiarity of *intention tremor* in multiple sclerosis is that it ceases during repose and that it is very slight at the beginning of the movement, then gradually becomes more extensive and rapid until it seriously interferes with the accomplishment of the attempted movement. The excursions are frequently so large and the rhythm of the oscillations so irregular that the tremor resembles ataxia; but the attempted movement always keeps at least to its general direction. The tremor of multiple sclerosis is usually more pronounced in the upper extremities.

2. The tremor in *paralysis agitans* is relatively slow, persists during repose, is usually somewhat lessened by purposeful movements, and is inhibited, often for a considerable time, by a very strong voluntary impulse. The rate of the oscillations is usually 5 or 6 in the second. The tremor is generally noticed first in the hands, in which it is especially distinctive. It often resembles certain fine intention movements like the rolling of a pill between the thumb and forefinger. There is rarely any tremor of the head in paralysis agitans, but to this rule there are exceptions.

3. In *senile tremor* there are 4 to 6 oscillations in the second. In less marked cases it is an intention tremor; in more marked cases it also continues during rest. The head and frequently the lower jaw, as well as the upper extremities, are the seats of this variety of tremor.

4. The tremor of exophthalmic goiter and of other *neuroses* (hysteria) is usually fine and rapid (8 to 9 oscillations to the second). It can generally be recognized most easily in the hands when they are extended horizontally with the fingers apart. Purposeful movements of the hands sometimes increase it.

5. *Toxic tremor* may assume varied forms in alcoholics, morphin

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habitués, in those poisoned by *mercury* and *lead*, as well as in acute and chronic nicotin-poisoning. Alcoholic tremor is characterized by early involvement of the lips, as well as of the hands. It is most pronounced during purposeful movements, and ordinarily disappears after taking a large quantity of alcohol. The toxic tremors are, as a rule, rapid.

It should be borne in mind that in addition to these varieties tremor may under some circumstances occur in health—*e. g.*, after vigorous bodily exercise, from mental excitement, intense cold, etc. Diagnostic stress can therefore be attributed to the occurrence of a tremor only after these physiologic conditions are excluded.

(*f*) Choreic and Athetoid Movements.

Choreic movements, or *chorea*, are involuntary (often very extensive), muscular contractions, which are not like clonic convulsions, absolutely without co-ordination, despite their lack of purpose or rule in co-ordination. They can be differentiated from voluntary movements by their bizarre character and their aimlessness. In pronounced chorea, the entire body is thrown into constant motion, so that any attempt at purposeful movements is often entirely fruitless. Chorea of the facial muscles is exhibited by grimaces; chorea of the breathing, voice, and speech muscles by the choreic articulation, which may render speech quite incomprehensible. Choreic movements are most pronounced in the *neurosis chorea*, in which they constitute the principal symptom. Symptomatically, choreic movements occur in the form of hemichorea in some cases of hemiplegia, especially with lesions of the posterior part of the internal capsule, and the optic thalamus (chorea post- or prehemiplegia).

Athetosis may be regarded as a variety of chorea. When the involuntary movements occur slowly and very regularly, the condition is called athetosis. Its significance is not essentially distinct from that of ordinary symptomatic chorea. Posthemiplegic athetosis, involving the muscles of the hand and fingers, is the most frequent form.

The muscle unrest observed in *Friedreich's* or *hereditary ataxia* is diagnostically an important symptom. French authors speak of it not unfittingly as "instabilité choréiforme." It also belongs to the symptomatic conception of chorea.

(*g*) Associated Movements.

These are to a certain extent physiologic. They depend upon the fact that intense motor impulses which escape inhibition are switched off to neighboring or more distant muscle territories. Pathologic associated movements are noticed especially when, through some interruption of conduction, the impulse of the will cannot be transmitted in a physiologic direction. A weakening of the physiologic associated movements in the territory supplied by the facial nerve is sometimes of service in suggesting the existence of a paresis of this nerve quite early in its involvement.

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Strümpell has described a special form of associated movement as "tibial phenomenon"; it consists of a contraction of the tibialis anticus during adduction of the leg. This phenomenon is observed in spastic paralysis, and is regarded by Strümpell as indicative of an anatomic lesion in the pyramidal tract. According to Mann, this phenomenon may also be reversed, the leg being adducted when the tibialis anticus contracts. Other associated movements occurring with lesions in the pyramidal tract are, according to Strümpell, extension of the great toe during extension of the leg; the so-called radial phenomenon, which consists of extension at the wrist during flexion of the fingers; and a pronation of the hand which follows the elevation of the forearm from a dependent position, the palm having been directed anteriorly. As a symptom of a lesion in the pyramidal tract, Babinski describes an associated movement occurring particularly in hemiplegia, which consists of the elevation of the paralyzed thigh when the reclining patient sits up or simply attempts to assume a sitting posture.

In reference to the associated movements of the eyeball in facial paralysis, which have been described as Bell's phenomenon, the reader is referred to p. 863 *et seq.*

(*h*) Forced Movements.

These movements, which are sufficiently characterized by their name, are highly co-ordinated, and so constituted that they can be simulated.

They are especially typified by most of the hysteric spasms, laughing and crying spasms, "arc de cercle," "grands mouvements," and "attitudes passionelles," as well as by many phenomena of convulsive tic. The forced movements, lateral positions, rolling and manège movements of patients with lesions of the median cerebellar peduncle belong to the same category.

A peculiar form of forced movement is the so-called forced laughing which occurs as one of the characteristic symptoms of multiple sclerosis. The laughter of these patients, whether it be spontaneous or the result of external stimuli, is spasmodic and uncontrollable; it apparently arises in the ordinary psychologic way, and then, according to the statements of the patient, takes on all of the characteristics of a pure somatic spasm. The fact that the forced laughing of multiple sclerosis is associated with a spasmodic condition of the musculature and increased muscle and tendon reflexes would naturally lead us to suppose that the slight motor stimulation and tension of the laughing muscles always produced by the suggestion of laughter-exciting objects or ideas, together with the increased reflex activity of the laughing muscles, is sufficient to throw the laughing mechanism into increased and continued spasmodic action.

(*i*) Catalepsy; Cataleptic Rigidity (Flexibilitas Cerea).

By catalepsy is understood a peculiar state of the motor system not yet explained, in which the limbs seem to be fixed and to present to passive motion a moderate dough-like resistance, retaining for a longer or shorter period the position given them. Catalepsy can be differentiated from active contractures by the fact that the parts do not resume their former position after they have been forcibly changed. This cataleptic condition of the muscles occurs in the hysteric, in hypnosis (see p. 739), in the psychosis designated *katatonia*, and sometimes in brain tumors leading to stupor [especially those implicating the corpus callosum.—Ed.].

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(*k*) Myotonia.

This is a rare condition of the muscles which, as yet, has been observed only in Thomsen's disease (myotonia congenita). In this affection the muscles appear normal during repose, but when voluntary movements are attempted assume a characteristic tension which hinders the movements. The tension then gradually diminishes, enabling the movements to proceed in an approximately normal way. (See later, Myotonic Reaction, p. 815).

3. ATAXIA; DISTURBANCE OF CO-ORDINATION AND SO-CALLED CEREBELLAR ATAXIA.

To facilitate better comprehension of this section, it is advisable to read first the section upon Examination of Complicated Sensory Functions, p. 766 *et seq.*

Ataxia means absence or disturbance of the co-ordination necessary to the accomplishment of purposeful movements. Co-ordination is the effectual distribution of voluntary impulses to the groups of muscles which must act harmoniously in each intended movement. Ataxic movements are inco-ordinated or badly co-ordinated movements.

Ataxia shows itself clinically by movements which are not necessarily curtailed in power, but which accomplish a result different from the one expected. Ataxic movements produce an impression of insecurity; they either overshoot the mark, or, conversely, do not reach it or reach it in a roundabout way with loss of time. The movements of a child making his first attempt at walking present the best picture of ataxia; he is not yet "master of his muscles." Ataxia is to be demonstrated clinically by observing how a patient performs voluntary movements. If the ataxia is pronounced it may be evident in the simplest movements, because even these require co-ordination of various muscle groups. If less marked, it may be noticed only in attempts at more complicated and finer movements. Ataxia of the lower extremities is usually most noticeable in the patient's gait. A pronounced ataxia gait presents a peculiar clinging, swinging, frequently stamping character. Ataxia of the upper extremities may be tested by having the patient attempt to touch an object—*e. g.*, his nose with the index finger. If he is ataxic he will either be unable to do so, or he will succeed only after several trials. In a similar way the lower extremities may be tested for ataxia. Ataxia, both of the upper and lower extremities, is usually increased when the patient's eyes are closed and he is thus deprived of the aid furnished by the sense of sight in correcting his movements.

Ataxia appears typically, and by far the most frequently, in *tabes dorsalis*, in *hereditary tabes* or so-called *Friedreich's disease*, in *lesions of the motor cortex* (cortical ataxia), in *polyneuritis*, especially in *polyneuritis alcoholica*, and rarely in *multiple sclerosis*.

In each case the ataxia may arise from a different cause. It seems to be certain that in children co-ordination is learned essentially by the sensory control of the completed movements. A child congenitally

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anesthetic and blind would never learn to perform co-ordinate movements. Sensory control is necessary for the acquisition of co-ordination, and probably likewise for its preservation, although perhaps not in the fullest measure. We may therefore say that the disturbances of sensibility which occur contemporaneously with ataxia have a direct bearing upon the occurrence of the latter. This is especially plausible in tabes dorsalis, in which sensory disturbances are the rule. It is self-evident that the sensibility of the skin is of much less importance than that of the deeper parts—the joints and the muscles—which aid us so efficiently in determining changes of position of the extremities. (See Testing the Appreciation of Passive Movements, p. 767 *et seq.*). Therefore it follows that ataxia need not accompany a pronounced cutaneous anesthesia, provided the perception of passive movements is not affected.

In many examples of ataxia, however, we cannot determine any very extensive disturbances of the deep sensibility of the extremities, so that we must search elsewhere for the explanation. Many authors (especially v. Leyden and Goldscheider) assume correctly, as the author thinks, that the disturbances of sensibility, especially in tabes, are frequently overlooked, because sufficiently accurate methods of examination are not employed to disclose the slight degrees which are present. In fact, it is conceivable that very insignificant disturbances of sensibility of joints, tendons, and muscles are quite capable of upsetting that fine control which is regulated by unknown centripetal impulses and which persists during movement only for an instant, and yet are so slight that they might be entirely overlooked during a rough test. It should be borne in mind that, by means of the mechanism described upon p. 767, the sensation of innervation for the perception of passive movements of the extremities may also be utilized.

Even after excluding these cases with sensory disturbances which are rather insignificant and difficult to demonstrate, there still remain other cases in which such an explanation does not apply, and for which, therefore, we must work out some other of the explanations which follow deductively from the theory of ataxia.

Co-ordination, as we have seen, is accomplished to a certain extent after the manner of a reflex, using the word reflex in its broadest sense. Peripheral stimulation incites the central organs to transmit throughout the duration of the movement just the appropriate amount of motor stimulation to each muscular district concerned. Obviously, such a procedure may be damaged either in the sensory limb of the reflex arc, in the psychomotor center, in its immediate neighborhood where the centripetal stimulation is transferred to the center, or, finally, in the motor limb of the reflex arc. Injury of any one of these components will probably produce ataxia.

In the so-called *cortical or central ataxias* which are observed in focal lesions of the motor convolutions unaccompanied by sensory disturbances, we must assume that the cortical centers have lost their capacity of carrying out normally co-ordinated representations of movements and impulses. We cannot, however, exclude the possibility of

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such ataxias arising from an imperfect conduction of the centripetal sensory impulses to the motor center, for an imperfect conduction is quite possible, even if the sensory appreciation itself seems normal. Whenever an ataxia of cortical origin is also associated with sensory disturbances for the appreciation of passive movements, the latter difficulty alone is quite sufficient to account for the ataxia, just as it is in tabes. In such cases the only distinction from tabes would be that in the cortical variety the sensory path would be affected much nearer to the motor center than to the periphery. As a rule, these cortical ataxias are not very pronounced, at least in so far as they depend upon an extensive lesion of the motor cortex, because if the cortical lesion is very large the picture of the paralysis overshadows that of the ataxia. (Consult p. 763 in regard to the character and recognition of the ataxia due to disturbances of the representations of movement.)

A lesion in the motor limb of the "co-ordination arc" may also produce *ataxia*, as is well shown by the cases of motor polyneuritis without sensory disturbances. It is clear that a co-ordinated impulse can accomplish its proper action only when the conduction is perfect, even down to the muscles. If certain ramifications of the motor conduction are affected by serious obstructions, the co-ordinated impulse is naturally curtailed and ataxia must result. Under these conditions a certain degree of motor paralysis would, from the nature of things, be present, and many authors are inclined to exclude such an ataxia with paresis from the true ataxias, and to call it a *pseudo-ataxia*. The author does not consider this justifiable. The characteristics of these cases may be exactly the same as of those of other origin, and even the theoretic difference exists only in a different localization of the interruption of conduction within the aforesaid reflex arc. Moreover, we must assume an interruption of conduction also both in ataxias of sensory origin and in the cortical or central ataxias, independent of sensory disturbances. Besides, in cases of polyneuritic ataxia the paralysis is sometimes very much in the background or can hardly be observed at all. To be sure, such a paralysis could always be detected if we were able to test the motor power of each muscle individually; but this is not possible, because in general we can test muscles only as groups. Hence slight paresis of individual muscles may readily escape our notice, although it suffices to explain the ataxia. Ataxia may even be caused by mere diminution in the tone of certain groups of muscles, from peripheral disturbances of conduction which cause retarded action of these muscles.

The above explanation postulates co-ordination as a complicated reflex which is centered in the cortex. In opposition to this conception, Cyon has attempted to localize the co-ordinating reflex center in the spinal cord. His hypothesis not only fails to explain cortical ataxia, but is also opposed to our idea of the simplicity and elementary nature of the spinal-cord reflexes. Yet it must be acknowledged that, inasmuch as it determines muscle tonus, the spinal cord does play some part in co-ordination, because a muscular action takes place promptly

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and securely only when the muscle at the beginning of its contraction possesses a sufficient degree of tension.

Our hypothesis, nevertheless, does not admit of a special co-ordinating center in the brain distinct from the motor centers of the cortex, and from this a centrifugal co-ordinating tract descending through the cord. Many authors, however, have assumed this. The truth is that all our knowledge of the function of the cerebral cortex, especially our views upon motor aphasia and the results of stimulating the motor cortex in animal experiments, shows that the cortex is the motor organ of highest development, and that it sends out impulses which have been already co-ordinated. If, however, as is practically certain, the psychomotor or pyramidal tract transmits co-ordinated impulses as a direct radiation from the cortical motor center, it is quite incomprehensible what there is for special co-ordinating centers and co-ordinating tracts to do. The theory that the degeneration of the posterior column in tabes involves a special centrifugal co-ordinating tract, and so leads to ataxia, is thus disproved, even if we disregard wholly the fact that the posterior columns are nothing but prolongations of the sensory roots, a fact which has been proved both anatomically and experimentally. As a matter of fact, this theory, opposed to the attribution of ataxia uniformly to disturbances of sensibility, was advanced only to explain cases of tabes in which ataxia was found presumably without sensory disturbances. But even if we disregard the probability of having overlooked slight disturbances in sensibility of the deeper parts (especially emphasized by v. Leyden), these cases are easily explained by assuming that the ataxia depends either upon involvement of the representations of movement in the motor cortex or upon lesions of the peripheral motor tracts. Pathologic changes have been found both in the cortex (Jendrassik, see p. 764) and in the peripheral nerves of tabetic patients. As has been mentioned, ataxia may also depend upon a disturbance of muscle tonus. This latter, which is ordinarily very striking in tabes, can readily be explained by the lesions of the reflex collaterals and of the posterior columns, even without any sensory disturbance. We know that the fibers of the pyramidal tracts subdivide in the cord into "*collaterals*," just as the sensory fibers of the posterior columns do, and that these "*collaterals*" are first connected with the ganglion cells of the anterior horns by means of "terminal arborizations." Hence we must accept the fact that ataxia may ensue from partial lesions of the pyramidal tract in the neighborhood of the "collaterals," as the result of an imperfect distribution of the motor impulses.

So-called "*cerebellar ataxia*" requires special mention, as symptomatically it does not agree with the picture of ataxia described above, nor can it be explained in the same way. It may be described as a characteristic reeling, both in walking and standing, which is observed especially in patients with cerebellar tumors, particularly when the worm is involved. This staggering gait, resembling that of a drunken person, is quite different from the gait of the true tabetic. It results from the disturbance of equilibrium which occurs in cerebellar diseases,

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and is attributed to the relation of the cerebellum to the semicircular canals. We assume that this connection is established by the median acoustic roots crossing over in the vestibular nerve. *Cerebellar*, as contrasted with true, ataxia is associated with vertigo, and for the most part is noticed only while the patient is walking or standing; whereas, while he is lying in bed, the movements of the legs, as well as those of the arms, appear quite normal.

There is, however, sometimes a distinct uncertainty in the movements of the arms and legs even in bed, which is suggestive of tabes dorsalis. This will be understood if we remember that scarcely any extensive movement of the extremities is possible without some more or less vigorous participation of the sense of equilibrium on the part of the cerebellum in order to correct the altered relations of gravity. The changed muscle tonus which is almost always demonstrable in these cases may also play a part in tbis true ataxia of cerebellar disease. This association of change of muscle tonus with cerebellar affections has been explained by the work of Luciani, which shows that the muscle tonus is diminished by extirpation of the cerebellum. It has previously been pointed out that a diminished muscle tonus may produce ataxic phenomena. We consequently see that the so-called cerebellar ataxia is made up of two components—the disturbance of equilibrium and actual ataxia.

The gait is similarly affected when centripetal stimulations are not transmitted normally to the cerebellum, for these are essential to its rôle as the organ of equilibrium—*e. g.*, in *eye-muscle paralysis*, and in *affections of the organs of hearing* which lead to vertigo, especially in *affections of the semicircular canals* (Menière's disease). The same thing is seen in hereditary ataxia, in which it is probably due to the loss of the centripetal impulses conveyed to the cerebellum through the direct cerebellar tract and the tract of Gowers.

III. GENERAL DISCUSSION OF THE METHODS OF TESTING THE SENSIBILITY.

The method of testing the higher senses will be described in the part devoted to the special examination of the cranial nerves. Here we mention merely the testing of the common sensibility, especially of the trunk and extremities. The work of M. Burchardt, referred to upon p. 760, should be consulted in regard to the simulation of sensory paralysis, and also p. 760, concerning the impossibility of simulating certain results obtained by testing the pressure sense with v. Frey's "irritation hairs."

SENSORY PARALYSIS.

Complete abolition of the sensibility of a definite part of the body is called *anesthesia;* a mere diminution of sensibility, *hypesthesia.* In all examinations for sensory defect it is important to have the patient shut his eyes, in order to exclude any visual control of the results, intentional or otherwise.

The examination is complicated because it includes the determination of the relations of very different sensory functions.

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(a) Examination of Simple Sensory Functions.

Method of Testing Tactile and Pressure Sensibility.—Tactile and pressure sensibilities are in a way identical. The estimation of pressure sensibility is nothing more than a quantitative estimation of the sensation of touch. Conversely, the examination of tactile sensibility furnishes a merely qualitative estimation of the pressure sense. Among other things in favor of this conception is the circumstance that both senses are localized at identical points of the skin, the so-called pressure points (see below).

Tactile sensibility is tested by touching the patient's skin lightly with the tip of the finger, or, in finer tests, by means of a dry camel's-hair brush, and ascertaining whether he feels the touch or not; if so, what he feels and where he feels it. Anesthetic spots can then be marked upon the skin with a dermatographic pencil (p. 164), and the result afterward transferred to a diagram of the body. By means of this test we can sometimes determine *a complete loss of tactile sensibility* over a considerable area; in other cases we can gather from the patient's statements that he appreciates the touch, but less plainly than normal, showing only *a slight diminution of tactile sensibility*. The comparative examination of different symmetric parts of the body is here of the greatest importance. (In regard to the significance of so-called tactile *hyperesthesia* or, better, *hyperalgesia*, compare the section on Sensory Irritative Phenomena, p. 769 *et seq.*)

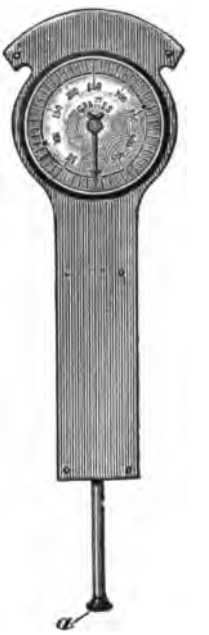

Fig. 294.—Eulenburg's baresthesiometer.

Pressure sensibility or perception—*i. e.*, the quantitative appreciation of the sense of touch—can be tested accurately only when the skin has a firm subcutaneous support—*e. g.*, over the ulna and radius. The part to be examined must itself be firmly supported. Otherwise, to appreciate the pressure, a patient would make use of his muscular power—*i. e.*, the innervation sense (see p. 762 *et seq.*)—and the results would no longer be correct. Gross disturbances may be recognized accurately by simply touching the patient's skin with the finger-tip, varying the amount of pressure and having him describe what he feels. For more exact quantitative determination, small objects of the same bulk but of different weight, or, still more accurately, the *baresthesiometer* (Eulenberg's, Fig. 294) may be employed. This is a simple instrument in which a pelot (*a*) working upon a spring is applied with varying degrees of pressure to the skin, and the grams of pressure employed can be read upon the scale. By means of this instrument we can determine, on the one hand, the minimal amount of pressure which can be appreciated as differing from a mere touch, and, on the other hand, the size of the pressure difference that can be felt—*i. e.*,

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how much the indicator moves before the patient can be sure that the pressure has been increased.

v. Frey[1] has found that the value of the pressure appreciation depends essentially upon the size of the area pressed upon and upon the rapidity of the pressure. The second factor can neither be exactly estimated nor regulated by simple clinical methods, hence the results of the above method are approximate only. In any case they are of value only if the deviations from the normal are quite striking. It is at all events desirable, in order to establish such deviations, that both the patient and a normal control individual be examined exactly in the same way —*i. e*, with corresponding areas of skin and equal rapidity of pressure.

In regard to the differences of the value of the pressure appreciation, Weber found that, normally, differences of weight of 1 : 30 can be distinguished by the palmar surface of the third phalanx of the index finger (excluding the balancing movement—*i. e.*, with the finger lying upon the table). This relation remains almost the same, according to Fechner's psychophysical law, if we alter the absolute size of the burden within wide limits. The rapidity of the pressure probably influences these conditions. Although neglected by Weber, it should be taken into account in making comparative tests. His estimates are based upon quick pressure. In consideration of the insufficient data upon the physiologically normal values, it is advisable for clinical purposes to make control tests upon the corresponding cutaneous areas of a healthy individual under absolutely identical conditions (the same pressure surface and, as nearly as possible, the same rapidity of touch). If the affection is unilateral, it is possible to test the pressure sense by a simple, unobjectionable method. Equal weights (of identical surface and material) are placed as slowly as possible upon symmetric parts of the skin, and the patient, with closed eyes, determines whether one of the weights seems heavier than the other. Unsymmetric cutaneous areas vary so decidedly, even physiologically, in the refinement of their pressure sense, that it is not safe to employ the above-mentioned method of comparison except with the greatest care.

So-called Pressure Points; v. Frey's Irritation Hairs.—The quantitative estimate of the pressure sense has been placed upon an apparently more secure basis, since we have known (Blix, Goldscheider) that the pressure sense is not scattered diffusely in the skin, but that it depends upon localized organs. The projections of the latter upon the surface of the skin are called "pressure points," and their anatomic substratum, according to v. Frey,[2] consists of nerve fibers arranged around the coronary hair follicles. The pressure points are distributed in widely differing numbers to the different parts of the skin. The enumeration of the hairs practically determines the number of pressure points, because a pressure point lies in the projection of each hair follicle, and about 95 per cent. of the skin surface is covered with hair. As a matter of fact, there are some pressure points between the hairs and upon hairless places—*e. g.*, they are specially numerous in the palm of the hand and in the sole of the foot. (On the palm of the hand, according to v. Frey, there are at least 100 pressure points to each ¼ sq. cm.) These isolated pressure points are the only receptors of tactile and pressure perceptions. A slight touch or pressure carefully localized between them either cannot be appreciated at all, or can be appreciated only when the skin is so deformed by the pressure that neighboring pressure points are affected. v. Frey claims that the existence of localized pressure points thus renders possible a strictly localized testing of the pressure perception, and his examination seems to furnish much more trustworthy results than superficial testing. In testing pressure points, we first determine the location of the points and then estimate their ability to discriminate pressure. v. Frey's irritation hairs accomplish both purposes most efficiently. They are short spears of hair of varying stiffness, glued at right angles into the end of a wooden handle. Human hairs are employed

[1] v. Frey, "Untersuchungen ueber die Sinnesfunctionen der menschlichen Haut." *Abhandlungen der mathem.-physischen Classe der königl. sächs. Akademie der Wissenschaften*, vol. xiii., No. 3, Leipzig, 1896.

[2] *Ibid.*

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for weak stimulation, and horses' hairs for strong stimulation. Hairs have two advantages for the mechanical irritation of the skin. In the first place, they act upon very small surfaces of the skin—*i. e*,, they are sharply localized—and, in the second place, the intensity of the irritation can be graded. To estimate the hair's irritation value, we first determine the amount of weight in one scale of a delicate chemical balance which the hair can lift by exerting pressure with its end upon the other scale. The power of the hair, a term which v. Frey applies to the weight raised by the hair, is a constant factor for each irritation hair, because a pressure sufficient to balance a heavier weight would bend the hair and consequently destroy its power. By means of such irritation hairs the pressure points can not only be mapped out, but also sharply localized and quantitatively stimulated. The irritability or pressure susceptibility of a certain pressure point is to be estimated by determining the weakest hair which can be felt at that point. With hairs of different thicknesses the cross-section as well as the power influences the variation in the irritation value, and v. Frey has found that the irritation value is proportional to the product of the power by half the cross-section. This product he calls the *tension value.* It also measures the pressure irritation value of the hair. v. Frey found the mean susceptibility of the pressure points he examined to equal a tension value of 1.44 grammillimeters.

The power of any hair will also depend upon its length, since a long hair will be bent by less force than a short one, and it is upon this principle that v. Frey's esthesiometer has been constructed. The instrument consists essentially of a strong irritation hair (horsehair), which can be pushed out of a sheath to a greater or less distance, and whose length can be read in millimeters from a scale on the sheath. The millimeter scale is gauged empirically by estimating the power of the projecting portion (by means of a delicate chemical balance) for every fifth or tenth division of the scale. The instrument is manufactured by Zimmerman, of Leipzig. The thickness of the hair must also be measured in order to determine accurately the pressure value of the instrument for a definite length of hair. Half its cross-section multiplied by the power gives the *tension value* of the hair, which, as mentioned above, is identical here with the irritation value. The examination of the pressure sense should be conducted as follows: The positions of the pressure points are first localized with stronger irritation, and then their susceptibility to pressure estimated with weaker irritations. As yet no clinical experience has been tabulated with this method, but it would seem to the writer to possess considerable utility, although less for examining anatomic lesions of the nervous system, wherein only the grosser disturbances are of diagnostic value, than for the examination of *neurasthenic* and *hysteric symptoms*, for the investigation of the action of drugs upon the sensory nervous system, and the like.

v. Frey believes that by means of this method we can demonstrate the oscillating character of the pressure sense—*i. e.*, the periodical increase and decrease of the perception, and even its fatigue. The method may therefore perhaps obtain a similar significance as the demonstration of the fatigue of the visual field in neurasthenia and hysteria. As contrasted with the latter test (visual field), the results obtained by means of the irritation hairs can in no way be simulated, because the patient is unable to orient himself concerning the pressure points that are being stimulated. v. Frey states that the mean irritability of the pressure points over the entire body is about equal. This is interesting and important for the clinical utility of the method. The local variability of the acuteness of the pressure sense seems to depend more upon the number of pressure points. The rapidity of applying the irritation hairs has but little influence, yet it is better to do it as slowly as possible.

Method of Testing the Cutaneous Sensibility to Pain.—By pricking a patient with a pin, we determine whether the prick as well as the touch is felt and properly localized. The anesthetic or hyperesthetic spots are then marked upon the body and later transferred to a diagram. In this test special attention should be paid to deter-

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mining any delay in transmission of the impression, such as is so common in tabes dorsalis and peripheral neuritis. A good plan is to have the patient call out the word "now" the instant he appreciates the touch, and the word "prick" the instant he appreciates the prick. A distinct interval often elapses between the two. v. Frey's[1] researches attribute this difference to the increase of a physiologic peculiarity of pain perception, which, in so far as it is mechanically discharged, always possesses a distinct latent stage, which does not exist in tactile perception. It is a well-known fact that the pain caused by stubbing the naked toe becomes more severe after the lapse of an instant. The accentuation of the pain sense, the painful after-sensation, and the repetition of a painful impression (successive polyesthesia) dependent upon a slight prick are all related to this delay and occur under similar conditions. Simultaneous polyesthesia is a different phenomenon, and probably depends upon an irradiation or a reflex perception (see p. 774 *et seq.*). In this variety several pin pricks are simultaneously perceived at neighboring spots instead of one single pin prick. The examination with the pin prick can be made easier by comparing different parts of the body.

Testing the Sense of Pain by Means of Irritation Hairs; Pain Points.—v. Frey[2] has recently demonstrated that the appreciation of pain, like that of touch, is not diffused over the skin, but is localized at circumscribed points, the so-called *pain points*. The territory between these points is insensitive to pain. Ordinary experience with pin pricks at first makes this seem paradoxical, until we recall that any sharp mechanical irritation of the skin sufficient to excite pain causes a deformity upon every side. To demonstrate the pain points, v. Frey has employed the irritation hairs described upon p. 758. The pain points are very thickly distributed, so that very fine hairs are selected, in order to avoid the skin deformity which would otherwise prevent an isolated irritation; they must, however, possess relatively considerable strength (see p. 758), so v. Frey sharpened the horsehairs by means of a scalpel under a lens. Experience shows that for demonstrating pain points they are superior to needles, because they do not injure the skin. Such pointed irritation hairs, if possessed of sufficient "power," cause pain, which proves that the painful irritation of the skin from needle pricks does not depend upon the injury as such. If we sharpen the hair we can employ the esthesiometer (described on p. 759) to test the pain points. Only by employing these irritation hairs can we be positive that a painful impression is produced immediately at a definite spot, without any trace of a preceding sensation of touch. v. Frey has further found that the *irritation value* of a hair for testing the sense of pain is equal to the product of its power (see p. 759) and the square of its touching surface—*i. e.*, its point. He calls this varying power of irritation which decides the degree of pain the *pressure value* of the irritation hair, in contrast with the *tension value*, which, according to p. 759, measures the sense of touch. He has not yet estimated the median pressure value of the pain points, but it varies between 20 and 150 quadrimillimeter grams. The number of the pain points varies in different parts of the body between 100 and 200 to each square centimeter. With this great profusion of pain points it is easy to understand why we have only learned recently that pain perception is localized in points. The intra-epithelial free nerve terminations of the skin are thought to be situated in the anatomic substrata of the pain points; they are therefore located more superficially than the end organs of the sense of touch.

v. Frey's researches, as we have seen, prove that actual pain-percipient organs

[1] *Loc. cit.*, p. 242. [2] *Loc. cit.*

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exist in the skin. They are interesting theoretically because they show that pain, a phenomenon which otherwise we should have considered purely pathologic, has been to a certain extent anticipated by Nature and has been endowed with physiologic organs. Its importance can be easily understood, because the perception of pain in the skin protects the organism from impending dangers (transmitted pressure or actual injury) just as does the pressure sense. Whether the deeper organs also possess special pain nerves is not yet known. At all events, according to the statements made above, the very remarkable richness of the cutaneous pain nerves and the great distress that follows cutaneous injuries seem quite natural from a teleologic point of view.

Method of Testing the Cutaneous Thermal Sensibility.—A sufficiently accurate and very convenient method of testing the sense of heat and cold consists in breathing upon the patient's skin with the mouth wide open and again with it nearly closed. In the former case a sense of warmth, in the latter, of cold, will be appreciated distinctly and so characterized by healthy individuals. If any abnormality exists, its location can be represented upon a chart of the body. We can estimate the degree of disturbance more accurately by touching the skin alternately with test tubes filled with cold and warm water, and determining at what temperatures the water can still be discriminated as cold and warm. By this method we can estimate at the same time the so-called indifferent zone of the temperature sense—*i. e.*, the areas in which the tubes produce an impression neither of warmth nor of cold. These results are valuable only when symmetric parts of the body are compared, or when the results are compared with those from a healthy individual over corresponding cutaneous areas. This method is more useful than that of estimating the minimum difference of temperature which can be appreciated, because it furnishes conclusions about the relations of the nerves for warmth as distinguished from those for cold. Such a discrimination is necessary, because we know that the organs of warmth and cold sensation are anatomically distinct (warmth and cold points), and that warmth and cold perception may be affected independently of each other. Statements about the minimum temperature difference are of little value, because this difference may vary considerably according to the degree of the temperature used. Fechner's psychophysical law (see p. 758) is of no use, because the ordinary thermometer scales are divided only arbitrarily, and not according to the absolute temperatures. Especial instruments for testing the temperature sense may be desirable for physiologic purposes, but they are superfluous for clinical needs, especially if the principles of examination described here are followed.

An examination of the temperature sense is much simplified and entirely accurate if the disturbances are unilateral. Objects identical in size, shape, and substance, and of the same temperature are gently and slowly placed upon symmetric areas of the skin, and the patient, with eyes closed, tells the examiner upon which side the object feels warmer or colder. This method is of less value for differentiating disturbances which are not unilateral, because non-symmetric areas do not have the same temperature sensibility even physiologically.

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We are indebted to the studies of Blitz and Goldscheider for the demonstration that the temperature sense depends upon the existence of localized end organs in the skin which are specialized for detecting heat and cold. [Only a few physiologists and experimental psychologists accept these conclusions of Blitz and Goldscheider.—ED.] The warmth and cold points coincide neither with the pressure points nor with the pain points. Their relations have thus far been of no clinical significance. Should it be necessary in any case to determine the warmth and cold points, Goldscheider's method is the simplest. It consists in touching the skin with the slightly pointed end of a metallic cylinder varyingly heated. The end organs of the temperature sense—*i. e.*, the anatomic substrata of the warmth and cold points—are not yet known.

Method of Testing the Sensation of Innervation, or the So-called Sense of Strength; Judgment of the Conception of Movement.—By innervation sensibility is meant the ability to estimate the measure of voluntary movement impulses. One speaks of this capacity as the sense of strength in so far as by means of muscular power it serves to distinguish differences in weight. The designation *power sense* or sense of strength is really incorrect, because neither a separate sense nor special sensory nerves take part in this function. It is much more plausible to assume that the innervation sense differs from the actual sensory function by having its origin in the motor centers and not in the periphery. Probably the representation of movement becoming a reality—*i. e.*, by carrying out the activity of the will—is identical with the innervation sense. The expression *strength sense* or "power" sense can be justified only by its brevity and comprehensiveness. In reality the examination of this function might be considered quite as well under motility as under sensibility.

An entirely isolated test of the sense of strength is difficult, strictly speaking impossible, because the tactile sense simultaneously stimulated by the lifted weight is bound to influence the result. For practical purposes the test is performed in this way: Different weights are hung in a cloth sling looped about the hand, forearm, or foot, lifted by the patient, and then discriminated. At the same time the normal relations are best preserved by practising the same experiment upon control persons with healthy nerves—*e. g.*, upon the examiner. By this method we are able to determine not only the absolute estimation of weight, but also the estimation of differences in weight which in pathologic cases are thought to be either too small or too large. The results are of diagnostic value only when they are very striking, because people's ability to differentiate weights by the innervation sense varies, and is to a great extent influenced by practice.

In this examination two devices may be employed to aid in excluding the pressure sense. One is to make the sling holding the weight very broad, so that it will extend over as large an area of skin as possible, and so reduce the pressure sensation below the value for which Fechner's psychophysical law holds. A slight increase then in the weight will not influence the pressure sense. The other device, supposing we wish to estimate the strength sense of the arm, is to hold the sling with the strongest possible pressure with the hand. Here, conversely, the strong hand pressure has the effect of making the sensation of pressure so

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strong that, according to Fechner's law, a slight addition of weight will not appreciably increase the sense of pressure, but only the fatigue of the muscles. E. H. Weber has made use of the latter device for testing the strength sense. He found that the strength sense of the arm could appreciate differences of weight of 1 : 40, whereas the pressure sense alone of the fingers appreciated differences of 1 : 30 (see p. 758). His researches also showed that a combination of the pressure and the strength sense furnishes no more accurate results than the strength sense alone. The principle of this test can also be applied to the lower extremities by fastening the sling which carries the weight to the thigh by means of a strongly tied knot.

The strength sense can also be tested by means of Eulenburg's baresthesiometer (see p. 757, Fig. 294). As in testing the pressure sense, the part to be examined—*e. g.*, the terminal phalanx of the fingers or toes—must be supported artificially and not by muscular action.

Even excluding the influence of the pressure sense, the results of these tests do not always furnish direct conclusions upon the "innervation" or "strength sense." The discrimination of weight may be affected by disturbances of innervation sense which are localized in the psychomotor centers and by motor pareses, because in such cases an especially vigorous impulse of the will is required to lift a weight. Whether to credit a disturbance in the discrimination of weight to an actual involvement of the "innervation sense" or to an error in judgment due to motor paresis must be determined from the results of an examination such as we have described above, and especially from the condition of the motility. Actual affections of the innervation sense must be attributed to functional or anatomic lesions in the neighborhood of the motor cortex.

The innervation sense, in so far as it is tested as a sense of strength, always refers to the entire group of muscles which together exercise the necessary co-ordination for lifting a weight, and any disturbance detected must affect equally the entire group of muscles in question. Now, in lesions of the psychomotor center and in lesions of the peripheral motor fibers a case is conceivable in which the disturbance—*i. e.*, the disproportion between the conductor of the motor impulses and the size of the innervation, affects only individual muscles of the group ordinarily acting together. In such cases the trouble should be considered less a disturbance of the sense of strength than one of co-ordination, in that one muscle receives too much innervation for the accomplishment of purposeful movement, the others too little. On reflection it will be seen that here we are in reality only considering the two cases of ataxia alluded to on p. 753 *et seq.*—namely, cortical ataxia from disturbances of the co-ordination impulses or of the representations of movement, and an ataxia from partial motor paralysis (so-called pseudo-ataxia). In the *cortical form* the ataxia depends upon the actual disturbance of the innervation sense, and with it also of the representation of movements; in *partial motor paralysis*, on the contrary, while the innervation sense, as such, is correct, the impulses transmitted to the muscle are quantitatively incorrect, and therefore the resulting ataxia simulates a falsification of the innervation sense. Before attributing the ataxia in any given case to perversion of the innervation sense or

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of the representation of movement, every other possible cause of ataxia must be excluded—*e. g.*, other disturbances of motility, disturbances of the sensibility of passive movements, and anomalies of the muscular tone. If the examination discloses abnormalities of the muscular sense, the diagnosis will be strengthened. Yet this is not necessary. Just this kind of cortical ataxia has sometimes been noted in focal lesion of the motor cortex. But even in tabes dorsalis, should an actual ataxia appear without any disturbance of sensibility, this cortical ataxia may possibly play a part, because, according to Jendrassik's researches, this disease presents in common with general paresis a degeneration of the tangential fibers of the cerebral cortex.

The So-called Sense of Location, Better Described as the Ability to Localize Sensations.—There is no special sense of location, for every sensory representation has its own place—*i. e.*, it is localized or referred to a certain part of the body. Physiologic psychology claims that each conscious impression produces a so-called local sign, depending upon the fiber from which it is discharged. These local signs become indistinct if the intensity or distinctness of the impression itself has suffered. However, we scarcely notice any disturbed localization provided the impressions themselves are perfectly intact. On the other hand, indistinct impressions and inexact localizations go hand in hand. The so-called associated or reflex impressions (see p. 774), together with the indistinctness of the local signs, are much more apt to be the cause of inexact localization in diminished sensibility. Such reflex impressions arise quite readily in interruption of sensory conduction from the lateral dislocation of the impulses through the collaterals of the sensory tract.

Slight disturbances of the location of sensation are sometimes observed in parts in which the paralysis is purely motor. They are due to the absence of the daily exercise of localization and of the continued refreshing of localization by movement, and are explained by the fact that the actual conception of space and the sensation of localization are acquired ontogenetically through motility only, and consequently require motility for their intact preservation.

The power of localizing sensation is best investigated by applying the touch, pain, and temperature tests to the area to be investigated, the patient having his eyes closed, and then requiring him to open his eyes and locate with his finger the spot at which he experienced the particular sensation; or, if this be impossible on account of motor disturbances, to describe its location. The distance between the point irritated and the point indicated by the patient, expressed in centimeters, indicates the degree of the error in localization. Absolute values for the normal error in localization cannot be given, since they are subject to great individual variations and are markedly influenced by practice. It is consequently best to compare the investigated area with the opposite healthy one, or, if this be impossible, with the same region in another person of similar physical and mental characteristics. In view of the influence of practice, only marked errors in localization are of diagnostic importance.

The So-called Muscle Sense or Muscular Sensibility.—What we have described above as the innervation sense is often confused, under the common title of *Muscle Sense*, with the appreciation of active and passive movements, to be described later. Such a special

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muscle sense does not really exist, and the obscure term leading to a misconception should therefore, once for all, be dropped.

Method of Testing Bone Sensibility; the So-called Sensation of Vibration.—M. Egger[1] was the first to demonstrate the feasibility of testing the sensibility of the bones by applying a vibrating tuning-fork to them. The examination was suggested by the extreme sensitiveness of periosteal lesions. If we set a C tuning-fork of 132 vibrations or an ordinary A tuning-fork of 440 vibrations upon the surface of a bone, the person examined normally perceives a characteristic whizzing or trembling sensation. Egger has demonstrated that this perception in pathologic cases is entirely independent of the absence or presence of cutaneous sensitiveness, and has proved that it is actually peculiar to the osseous system. It may be retained with a diminished cutaneous sensibility or lost when the latter is normal, despite the fact that the vibrations are naturally transmitted to a considerable distance. Egger found that this sensation of whizzing was strictly localized, so that under pathologic conditions the tuning-fork was appreciated at one spot of a bone and not at a neighboring spot. He also demonstrated a hyperesthesia of the bones, for they could appreciate the vibration of a high tuning-fork (vibrating 2048 times), although this normally is not perceptible. Bone anesthesia occurs especially frequently in the ataxic stage of tabes. The occurrence of disturbances in the appreciation of active and passive movements is, however, independent of the bone sensibility. In many cases of tabes, and especially in the initial stage, tuning-fork vibrations produce a sensation of burning as well as of whirring (hyperalgesia of the bones). In the trophic changes of the joints and bones in tabes, the disturbances in bone sensibility are mostly localized in the vicinity of the affected parts. Disturbances in bone sensibility also occur in *syringomyelia*, and mostly in the diffuse areas of anesthesia so characteristic of this disease. In *Brown-Sequard's unilateral paralysis*, the osseous sensibility, like that for passive movements, and in contrast to the cutaneous sensibility, suffers upon the same side as the motor disturbance. In *cerebral hemi-anesthesia*, bone anesthesia is localized upon the paralyzed side, although it is ordinarily incomplete upon the head. Bone sensibility varies in *hysteric anesthesias*. Frequently, though not constantly, the bones share in the anesthesia. In hysteric conditions, the bone, as well as the cutaneous, sensibility not uncommonly suddenly disappears under the influence of the tuning-fork vibrations. Bone anesthesias sometimes of equal, sometimes of less, extent than the sensory disturbance of the skin, are also found in *transverse lesions of the spinal cord*. In the latter case, the bones of the lower end of the leg are ordinarily most affected. From the pathologic findings Egger believes that the tracts for bone sensibility take an uncrossed course in the gray matter.

Egger's supposition that the vibrations of the tuning-fork are perceived only through the bones has recently been shown by Goldscheider[2]

[1] *Jour. de Physiol. et de Pathol. gén.*, vol. i., No. 3, 1899.
[2] *Berlin. klin. Woch.*, 1904, No. 14.

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to be erroneous. The soft parts are also susceptible to these vibrations, but not to so great a degree, owing to their less ability to vibrate synchronously. Goldscheider found, in contrast to Egger, that the perception of tuning-fork vibrations is partly dependent also upon the condition of cutaneous sensibility. One way in which he proved this was by anesthetizing the skin with cocain. The cutaneous sensibility and the osseous sensibility to tuning-fork vibrations may be separately tested by pressing the tuning-fork softly against the part for the former, and firmly for the latter. In the first instance the vibrations remain upon the surface, while in the second they are conducted to the deeper parts through the compressed skin. Goldscheider calls the perception of the vibrations of the tuning-fork the sensation of vibration, and regards it, not as a specific sensation, but as an expression of the sensation of rhythmic interrupted irritation of the nerves that are responsible for the sensations of contact or pressure. In spite of his objections to the conception of Egger, Goldscheider regards the test with the firmly applied tuning-fork as the best procedure for the estimation of osseous sensibility.

(*b*) Examination of Complicated Sensory Functions.

Method of Testing the Perception and the Judgment of Active Movements of the Extremities.—Even with closed eyes a healthy person has a very exact knowledge of every change of posture of his extremities, and graduates his motor impulses accordingly. These perceptions of movement depend primarily upon the innervation sense (see p. 763 *et seq.*)—*i. e.*, upon the judgment of the impulses of contraction which the individual muscles receive in a certain position, and, secondarily, upon the sensibility of the deeper portions of the extremities—*i. e.*, of the muscles, joints, tendon sheaths—and even of the skin. The skin is, of course, compressed and stretched differently in every change of posture. So this performance does not depend merely upon a single sensory function, but upon the cerebral reception of several sensory impressions aided by the innervation sense, which belongs in reality to motility. Affections of the appreciation and judgment of active movements are therefore encountered as often in sensibility disturbances from peripheral interruption of conduction as in lesions of the psychomotor centers or tracts, causing an impaired judgment of the effect of the will impulses. Yet, when the cause of the affection lies upon the motor side, the sensibility of the deeper parts can to a certain extent perform vicariously the function of the impaired judgment of the condition of muscular contraction; and, conversely, when the defect lies upon the sensory side, the innervation sense can equalize a certain part of it. It appears that this vicarious performance of one function by another is possible, and varies somewhat with the individual.

To test the appreciation of active movement, we direct the patient either to describe as accurately as possible a position of his extremities, which he voluntarily assumes and alters while his eyes are closed,

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or to touch with an extremity an object whose position he has noted before he closed his eyes, employing the shortest and most direct movement possible. In the latter experiment any disturbance of the function in question is manifested by the ataxic character of the movement and by its lack of certainty. This test, then, is like that for ataxia. But although absence of ataxia proves that the patient judges his voluntary movements correctly, nevertheless, if ataxia is demonstrated, we require further evidence to decide that it is accompanied by a failure in the appreciation or judgment of active movements. The latter function may be absolutely preserved and the patient may be perfectly cognizant of the ataxic peculiarity of his movements, yet be unable to execute them co-ordinately if the motor elaboration of his movement impulses is defective. We need the most accurate analysis possible, and a consideration of all the conceivable causes of ataxia, in order to decide whether the ataxia proves faulty judgment of the patient's own movements. Should the muscular tone (p. 754) be normal and no sign of motor weakness be apparent (p. 753) we must attribute the ataxia to a defective judgment of active movements. Then we must decide whether such an affected judgment depends upon an actual disturbance of sensibility or of the innervation sense—*i. e.*, a faulty representation of movement (p. 762). If the former, the appreciation of passive movements will also be affected. As we have already mentioned, this is the commonest cause of ataxia. It is also worth noting that an intelligent patient, simply by his own observation, can often decide correctly whether his ataxia should be attributed to faulty judgment of movements or to a faulty motor conduction. If, however, he attributes the difficulty to the former cause, ordinarily he cannot determine whether an actual disturbance of sensibility or of the innervation sense is at fault. Upon this point the results obtained from testing the appreciation of passive movements must decide. Actual disturbances of sensibility are the usual causes of the ataxia in tabes dorsalis, but a disturbance in the innervation sense or in the representation of movements may cause ataxia in cortical affections, without any true disturbance of sensibility.

Method of Testing the Appreciation of the Position and of Passive Movements of the Extremities (in Brief, Postural Perception).—In estimating a passive change in position of the limbs, the innervation sense is of no assistance. We judge of it purely in a sensory way by employing the sensibility of the deeper parts, muscles, fasciæ, joints, etc. Theoretically considered, the appreciation and judgment of active movements must be easier than that of passive movements, because the former utilizes more aids. Therefore, even where the appreciation of active movement is intact, that of passive movements must still be tested for. It is done in this way: The patient, with closed eyes, is asked either to describe passive changes of position in his extremities, or to imitate them with another extremity whose innervation has not been involved. This will scarcely be possible if the appreciation of passive movements is affected. Yet it is to

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be noted that under some circumstances the patient can compensate moderate disturbances by informing himself of the position of his extremities through the appreciation of muscular contractions by means of the innervation sense. In this way many observers explain the fact that the estimation of passive alterations of posture is comparatively well preserved in moderate sensory disturbances of the extremities, although this gives us no right to assume that the sensibility of the deeper parts has escaped. To obtain clear results in such cases we must demand of the patient a practically complete muscular relaxation. In most cases disturbances in the appreciation of posture show themselves without anything further by an ataxia, because of the failure of the essential sensory control of the movements. Hysteric disturbances of sensibility present an exception to this rule; in them the appreciation of posture can be completely lost without the appearance of ataxia. This can be explained by the nature of hysteria; because here the disturbance lies in the most central organs of consciousness, so that the intelligent appreciation of passive changes of position is annulled, but not the control of movements, which is supplied by the sensory impulses further down.

Disturbances in the appreciation of passive movements are observed principally in tabes dorsalis, where, as mentioned repeatedly above, they furnish a sufficient explanation of the ataxia. It is not rare to meet such disturbances in diseases of the motor area of the cortex, apparently because, for the purpose of co-ordination, the sensory fibers which subserve the appreciation of posture are anatomically related to the psychomotor centers. The above-mentioned disturbances in the perception of posture which occur in hysteria belong, in a broader sense of the word, as most hysteric symptoms do, to cortical implication.

Method of Testing the Touch Perception (the Stereognostic Sense).—Touch perception—*i. e.*, the recognition of the form of objects by their surface—is by no means, as is sometimes supposed, merely a function of the tactile and pressure sensation. Its popular title, feeling sensation or feeling sense, is therefore, in the strict sense of the word, incorrect. In handling an object we first employ the touch and pressure sense, then the appreciation of the active movements essential to feeling the object, further the perception of the position of the fingers encircling it, and finally the sensation of temperature, to recognize the material of which it consists (metal, wood, etc.). Here again it is a question of very complicated perception worked out in the brain with various aids, but in no way the product of a single specific sensibility. This conception makes it clear how, even when the examination of the simple sensory functions (touch, perception, etc.) shows no disturbance at all, the stereognostic recognition of objects may be disordered in cerebral disease—*i. e.*, in lesions of the motor cortex, whose relation to the innervation sense and to the judgment of active and passive movements and of the position of the extremities has already been mentioned—as well as in peripheral motor paralysis, which prevents a correct appreciation of the innervation sense.

To test the stereognostic sense we ask the patient, with closed eyes,

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to name various small objects placed in his hand. This function is only slightly developed in the feet, yet a healthy individual can recognize larger objects even with them. Objects placed upon the back can be recognized only when they are very large and characteristic. This clearly demonstrates that the stereognostic sense is not essentially aided by the cutaneous sensibility, as the old name, "feeling sense," would lead us to believe. As is well known, the stereognostic sense is very acutely developed in the mouth, where it depends considerably upon the perception of the active movements and of the position of the tongue.

(*c*) **Observations upon the Technique of Testing Sensibility.**

In the foregoing description the author has refrained intentionally from describing the instrumental methods which are claimed to possess special accuracy in testing sensibility. Perhaps some readers may miss them, as, for instance, the examination for localizing the sensation of touch, or for testing the feeling crises with Weber's touch compass, or with Sieveking's esthesiometer, the examination of the temperature sense with the various peculiarly constructed thermo-esthesiometers, etc. He is convinced that none of these instruments aids clinical observation to any extent, because, in the first place, our diagnosis must depend upon the grosser changes which can be demonstrated by the simple methods described above, and because, in the second place, we either do not know the physiologic normal for such instruments, or else the normal varies so much according to the individuality that even the apparent exactness of the results may lead to marked diagnostic fallacies. Their apparent exactness is really fancied, because each method has so many sources of error that even the very great skill which is attainable by but few physicians will not obviate them. Everyone who has had any experience with the Weber's compass will agree with the author. Neither has he mentioned the electrical methods of testing sensibility, because in the present state of knowledge these will hardly be of much service to the diagnosis. As a matter of fact, we do not really know what we are testing for by this method, as it has not been studied enough physiologically. v. Frey's method of testing with irritation hairs should, however, be of real service in the future.

2. PHENOMENA OF SENSORY IRRITATION.

Paresthesia.—By paresthesias are understood subjective sensations—*i. e.*, sensations corresponding to no correlation in the external world—which are not actually painful, but which without any sharp boundary often merge into pain. They are sufficiently well characterized by such names as "*furry*," "*tickling*," "*crawling of ants*," and "*falling asleep*." The buzzing character and the complete dissociation of these sensations (the latter especially characterizes the sensation of fur or crawling of ants) depend, according to the research of v. Frey, upon the oscillatory character of the nerve stimulation in pressure sensation. This can very easily be demonstrated by exciting the pressure

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points by irritating hairs (see p. 759). Subjective feelings of warmth and cold correspond to similar paresthesias in the province of the thermal nerves; those of smell, sight, hearing, and taste, in the territory of the higher senses. Paresthesias may arise from an irritation of the sensory tracts at any point of their entire course, but they are most frequently observed in lesions of the sensory roots downward and are therefore localized in the peripheral nerves. The paresthesias which occur in spinal cord affections, and which are localized at the intercostal nerves or, rather, their sensory roots, are spoken of as "girdle sensation." The girdle sensation frequently becomes a "girdle pain."

Spontaneous Pains.—In a general way pain may be subdivided into parenchymatous and neuralgic pains. In *parenchymatous pains* the sensory fibers are irritated at their terminal ramifications, in *neuralgic pains*, at the trunks of sensory or mixed nerves, in the sensory roots or in the sensory centers. In the *former* the terminations of the sensory fibers are irritated quite independently of their origin, and therefore the pains overlap the boundaries of peripheral sensory areas, apparently at will. *Neuralgic pains*, on the contrary, according to the law of eccentric projection, are localized in areas that correspond exactly to the peripheral distribution of the nerve trunk or nerve involved. Pain may, however, be felt in neighboring nerve territories from irradiation of the pain from the involved nerve into them. (See p. 774 *et seq.*, Sympathetic Sensations.) A further difference between these pains is to be found in their severity. *Neuralgic pains* are generally much more severe than *parenchymatous pains*, for the reason that in the former a much larger number of fibers are painfully irritated, and ordinarily at the same moment. Probably for the same reason remissions in a severe pain are more decided in *neuralgic* than in *parenchymatous pains*. Some of these remissions can readily be attributed to a fatigue of a central pain-percipient apparatus, a fatigue which naturally occurs sooner and more pronouncedly after intense irritation than after weaker irritation. Another distinction is that generally with parenchymatous pains the entire painful area is sensitive to pressure. This is sometimes the case with neuralgic pains; but, as a rule, only that portion of the nerve trunk is sensitive to pressure which lies superficially or upon a hard foundation (neuralgic pressure points).

The best-known example of neuralgic pains are the *true neuralgias*, which occur in some instances in otherwise healthy individuals, but which in other instances are indirectly due to disease (*joint rheumatism, syphilis, diabetes*, etc.), and the so-called *lancinating pains* in spinal cord diseases, especially in the initial stage of *tabes dorsalis*.

To the parenchymatous pains belong most of those *pains* which occur in *organic disease of various viscera;* many of the *diffuse headaches* of *meningitis* and *intracranial pressure; toxic, febrile, dyspeptic,* and *anemic headaches*, as well as *migraine* and most forms of *neurasthenic headaches.*

In most cases the sensation of pain, whether it is of a neuralgic or parenchymatous character, is of peripheral origin—*i. e.*, depends upon an

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irritation of the peripheral sensory neurons (of the peripheral nerves or sensory roots). It still seems doubtful whether pains may be caused from involvement of the conduction tracts above the sensory roots in the spinal cord. This part of the conduction pathway has usually been considered to be purely esthesodic—*i. e.*, conductory but not irritable. On the contrary, eccentrically projected pains can doubtless be produced by lesions of the sensory tracts in the brain, especially of the posterior part of the internal capsules.[1] It is also certain that pains may take their origin from the most central organs of perception. It is to this category that the *suggested and auto-suggested pains and also many hysteric pains* belong. Naturally these pains have in their radiation the character of parenchymatous pains and not neuralgic, because the arrangement of the elements in the central parts—*i. e.*, the brain—does not correspond to the nerve trunks, but to the limits of the organ. For instance, hysteric joint pains are often improperly spoken of as articulatory neuralgia.

A peculiar combination of symptoms, so-called *anesthesia dolorosa*, should be mentioned here. It consists of the occurrence of spontaneous pains in a portion of the body which is anesthetic to external stimulation. Such conditions occur when a focus of disease, most often of the nerves or nerve roots, interrupts the conductivity of peripheral stimuli and at the same time causes irritation of the sensory fibers.

Hyperalgesia (Hyperesthesia); Sensitiveness to Pain.—By the term hyperesthesia or, better, hyperalgesia, we understand a condition of the sensory mechanism in which stimulation produces the sensation of pain in a certain area (especially of the skin), although normally such stimulation would not be painful. The slightest touch of the skin, moving the part, or the mildest sort of thermic influence may under such circumstances produce pain. We now recognize that pain perception (at least cutaneous pain perception) is a specific performance of a definite kind of nerve fibers (see p. 760 *et seq.*); hence, we may regard hyperalgesia merely as a supersensitiveness of the pain nerves. We have as yet no convincing demonstration that a vigorous stimulation of other sensory terminations—*e. g.*, of touch, warmth or cold—can produce pain, because we cannot exclude a coincident implication of the pain nerves in every such vigorous stimulation. Therefore we should substitute the expression hyperalgesia for hyperesthesia. We do not yet know what defects of the pain-conducting or the pain-appreciating parts of the nervous system can give rise to hyperalgesia; but it is certain that peripheral as well as central parts may assist in this hyperalgesia from an associated involvement of the sensory fibers or cells. Such involvement produces slight injuries which are insufficient to produce anesthesia, but sufficient to cause irritation. The best-known examples are the *hyperalgesias in the domain of neuralgic nerves during the early stages of neuritis*, the *zone-like hyperalgesias of the upper borders of sensory disturbances in cross-lesions of the spinal cord*, and the *general hyperalgesia of hysteric or neurasthenic patients.*

[1] See the meager literature upon this point in the article by Alfred A. Reichenberg, *Zeits. f. Nervenheilk.*, vol. xi., pts. 5 and 6, p. 349.

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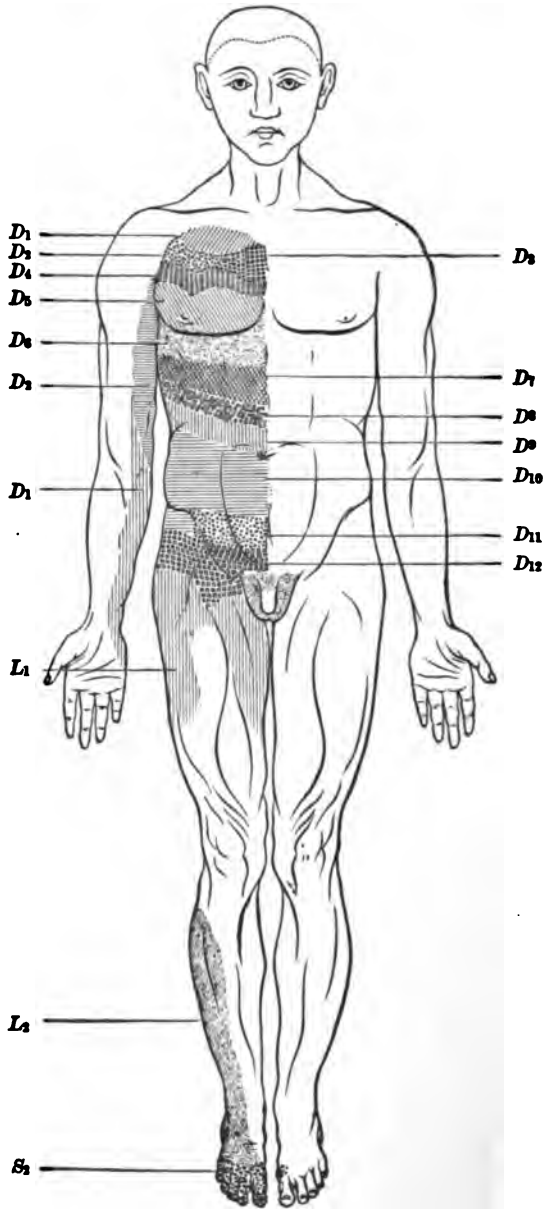

FIG. 295.—For description see next page.

The so-called *pressure sensitiveness* (better described as increased sensitiveness to pressure) is in reality nothing more than a special form of hyperalgesia. In the examination of the nervous system, the pain sensitiveness of the nerve trunk to pressure which occurs principally in

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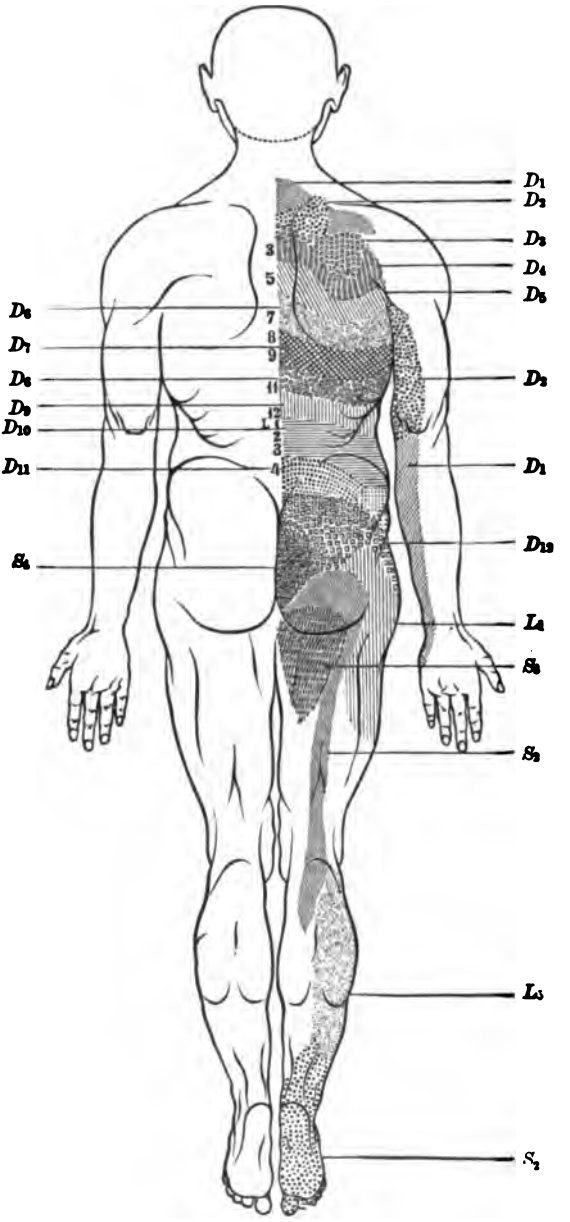

FIG. 296.

FIGS. 295 and 296.—Hyperalgesic and radiation zones of the skin in diseases of deeply situated organs. Zones on the trunk and extremities: Diseases of the heart, pain and hypersthesia in zones, C_3, D_1, D_4; tuberculosis of the lungs, D_1–D_7, particularly D_2, D_4. D_5; diseases of the esophagus, particularly D_5, D_6, D_8; diseases of the breast, D_4, D_5; diseases of the stomach, D_7, D_8, D_9; diseases of intestine (pylorus, colon), D_{10}, D_{11}, D_{12}; diseases of liver, D_7, D_8, D_9, D_{10}; diseases of the kidney and ureters, D_{10}, D_{11}, L_1; diseases of the bladder, S_2, S_3, S_4; diseases of the ovaries and testicles, D_{10}; diseases of the uterus, D_{10}, D_{11}, D_{12}, L_1; diseases of the cervix, S_1, S_2, S_3, S_4 (after H. Head).

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peripheral neuralgic and neuritic affections is of special interest. It is under some circumstances essential to examine into this appearance, even where no spontaneous pain is present.

The hyperalgesic zones of the skin in diseases of the viscera are mentioned in the following section.

Sympathetic or Reflex Sensation; Irradiation of Pain; Tickling; Hyperalgesic Zones of the Skin in Diseases of the Deeper Organs.—So-called sympathetic or reflex sensation is nearly related to hyperalgesia.

The best-known form of this is pain irradiation, in which the pain is perceived far beyond the limits of the painfully irritated peripheral part (pain in the entire trigeminal distribution, occasioned by a single carious tooth). This phenomenon can be explained only by assuming that the painful stimulation in the central organs (spinal ganglia, gray substance of the spinal cord or of the brain) overlaps or irradiates to neighboring tracts by means of dendrites and collaterals, and that, in accordance with the law of eccentric projection, confusion as to the origin of the perception results. [This entire subject has been put upon a satisfactory foundation at last by the studies of Head.—ED.]

The pain sense is not always concerned in reflexes either of primary or of secondary nature; in fact, touch and temperature sense or the higher senses can produce them, and such sympathetic sensations are not necessarily painful.

As an example of such associate sensation *tickling* may be cited. This is an irradiating or sympathetic sensation of an oscillating character diffused over a considerable surface of the skin, caused by a circumscribed skin stimulation.

Quincke[1] has made quite a complete collection of the practically important sympathetic sensations which have been thus far determined; but only a few of the most important will be mentioned: trigeminal neuralgia in affections of the frontal sinuses; parietal pain in affections of the middle ear and of the mastoid process; a tendency to cough from irritation of the posterior wall of the auditory canal (irradiation from the auricular branch of the vagus to the other vagus branches); a marked sensation of tickling and shuddering in biting upon sand; painful sensation in the back in swallowing; laryngeal pain in percussing a pulmonary abscess (Quincke); pain in the left arm (rarely in the right) in angina pectoris; pain in the back in stomach diseases; feeling of tickling in the nose from intestinal worms; pains in the shoulder in liver affections; pains in the left shoulder in affections of the spleen; pain in the lumbar region and genitalia in bladder affections; pains in the epigastrium and the stomach region in endometritis and during menstruation; pain in the knees in coxitis; simultaneous polyesthesia (p. 760) in spinal cord affections.

A phenomenon related to and frequently associated with these sympathetic sensations—a circumscribed cutaneous hyperalgesia depending upon diseases of the deeper organs—is of more diagnostic significance. The best-known example is the sensitiveness to pressure of the skin of the precordia in heart disease. This hyperalgesia is perhaps most satisfactorily explained thus: The centripetal irritations proceeding from the diseased organs which escape direct perception stimulate by irradiation the neighboring sensory parts of central organs. The latter are passageways for the sensory fibers coming from the areas of the skin in

[1] *Zeits. f. klin. Med.*, vol. xvii.,1890.

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question. In contrast to actual sympathy or irradiation, this irradiated stimulation is not strong enough to be appreciated by the sensorium as pain, but is

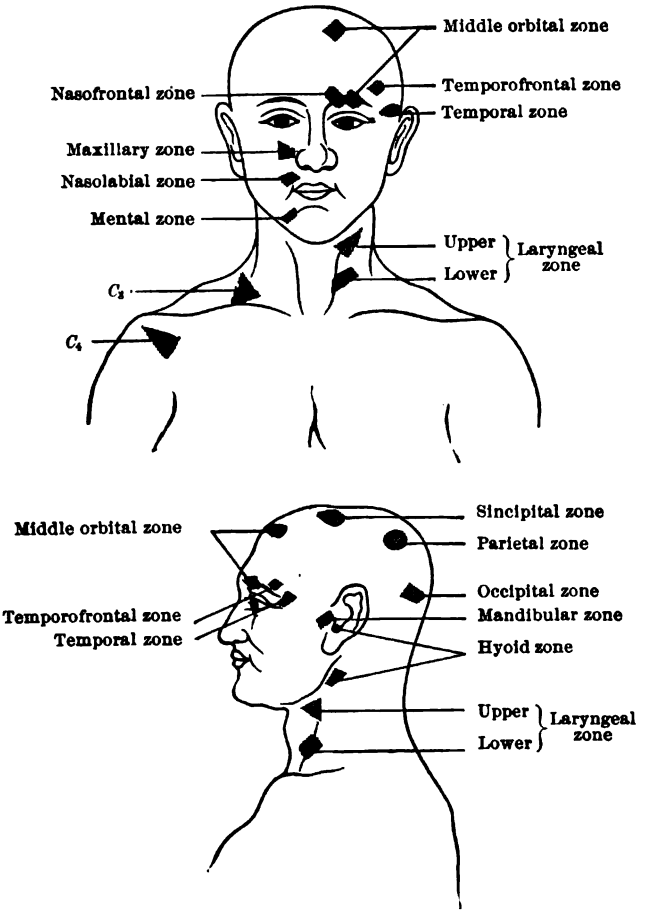

FIG. 297.—Hyperalgesic and radiation zones of the skin in diseases of deeply situated organs. Zones on the head and neck: Nasofrontal zone, diseases of the eyes, nose, and upper incisors; middle orbital zone, in hypermetropy; temperofrontal zone, diseases of the ears and heart; temporal zone, in glaucoma (after H. Head).

Sincipital zone: Diseases of the middle ear.
Parietal zone: Diseases of the ear and stomach.
Occipital zone: Diseases of the posterior half of the larynx and certain abdominal viscera.
Maxillary zone: Diseases of the iris and vitreous body.
Mandibular zone: Diseases of the upper molars.
Nasolabial zone: Diseases of the nose and dental pulp.
Mental zone: Diseases of the incisors and canines.
Hyoid zone: Diseases of the tonsils, tongue, and lower molars.
Upper laryngeal zone: Diseases of the dorsal surface of the tongue and the wisdom teeth.
Lower laryngeal zone: Diseases of larynx.

merely sufficient to cause a superior stimulation at the junctions of the sensory conductions coming from the skin.

Fig. 298, explains the process diagrammatically: *a* Represents the diseased organ with a centripetal stimulation proceeding to a sensory station, (*b*) for example, in the spinal cord; from there the stimulation can come to the senso-

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rium (*c*), either directly (*b–c*) or indirectly (*b–d–f*), *c*, jumping to a neighboring sensory tract (*e, f*). If this process of irradiation produces a painful stimulation at *d*, a painful sympathetic sensation will be referred to the cutaneous area (*e*); whereas if it produces merely a hyperirritable condition at *d*, the cutaneous area (*e*) will merely present a hyperalgesia to pressure and to other stimulations not causing pain. Thus, circumscribed cutaneous hyperalgesias, if anatomically established, may under some circumstances possess the same diagnostic value as actual sympathetic sensations.

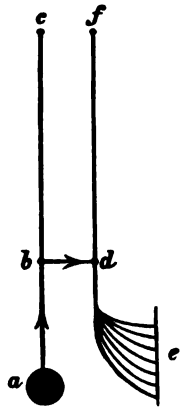

FIG. 298.—Diagram to illustrate skin hyperalgesias and the radiation of pain in disease of deeply situated organs.

Head, of London, has taken pains to investigate these zones of cutaneous hyperalgesia in a great number of pathologic conditions in order to establish some diagnostic relations to deep-lying diseases. Corresponding to the above explanation, he found that the hyperalgesic zones were identical with the zones to which the irradiation pains were projected in the diseased organs in question. He tabulated his observations in the accompanying plates[1] (Figs. 295, 296, and 297). The separate zones are shaded differently. The numbers and letters refer to the segments of the spinal cord (designated according to the corresponding spinal nerves) whose stimulation is the cause of the cutaneous hyperalgesia (based upon our knowledge of the spinal sensibility topography of the skin (see p. 922 *et seq.*). Probably the affected organs are also supplied by the same nerves. Thus far no satisfactory anatomic explanation has been able to correlate the localizations of hyperalgesic zones of the head to definite diseases.

Though interesting theoretically, Head's statements need further confirmation and perhaps modifications. Such relations of the deeper organs to the surface of the skin make one appreciate the therapeutic action of counterirritation to the skin, especially upon deep-seated pains. It is only fair to suppose that the same anatomic tracts which conduct the cutaneous hyperesthesias in diseases of the deeper organs can be utilized to transmit inhibition of pain by vigorous stimulation of the skin.

IV. EXAMINATION OF THE REFLEXES.

In testing reflexes it is advisable to distract the patient's attention as much as possible from the parts under examination, otherwise an involuntary inhibition may alter the reflex. The simplest device is to direct him to close his eyes. The fatigue of a reflex is sometimes responsible for mistakes in diagnosis. The response to the first tap should be observed attentively, because it may disappear after one or two repetitions. If the first response had not been noticed, the examiner would then incorrectly consider the reflex absent. It is therefore a safe rule to observe each reflex quickly and accurately, and to utilize repeated, careful examinations in order to discriminate in any doubtful case, for the reflexes, like other nervous functions, often vary at different times. These precautions are especially valuable in testing the patellar reflex, which is so important in diagnosis.

[1] The reflexes belonging to the cranial nerve territory will be described more fully in the special part devoted to examination of the separate cranial nerves. (Concerning the bladder and rectal reflexes, see the section upon Examination of the Bladder and Rectal Functions.)

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Ordinarily the reflexes are local in character—*i. e.*, they take place in the region of the body that is irritated. But with an increase in the reflex irritability, which may be partly within the normal physiologic limits and depend partly upon reflex stasis (described upon p. 783), the reflexes may be diffused in cross and longitudinal directions to other muscle areas and to other extremities. This corresponds to Pflüger's laws of reflex dispersion.

Increase of the reflexes, as well as decrease or absence and qualitative abnormalities (the so-called pathologic reflexes), are of considerable importance for diagnosis.

NORMAL CUTANEOUS REFLEXES.

The cutaneous reflexes of the upper extremities and of the face are very inconstant. They have, therefore, no diagnostic significance except when decidedly increased. The following cutaneous reflexes are clinically the most important:

Plantar Reflex.—Tickling or pricking the sole of a healthy person's foot produces a plantar flexion of the toes (see Fig. 299). A stronger irritation produces a dorsal flexion of the toes, combined with a dorsal flexion of the foot and flexion of the knee and hip joints.

The **cremaster reflex** is elicited when the inner surface of the thigh is irritated by scratching or pricking, or by stroking it quickly with the handle of a percussion hammer or some similar object. It consists of a sudden contraction of the cremaster muscle which draws up the testicle. This reflex should not be confused with the slow, worm-like contraction of the tunica dartos which frequently follows uncovering the patient, as a result of cooling.

The **inguinal reflex** (oblique reflex) (K. Geigel[2]) can be elicited in both sexes by an irritation similar to that described for the elicitation of the cremaster. It consists of a contraction of the lower fibers of the internal oblique muscle above and along Poupart's ligament. Since the cremaster muscle is nothing but a bundle of the internal oblique passing with the testicle through the inguinal canal, the cremaster reflex in reality belongs to the inguinal reflex. Examination of the latter in the female sex takes the place of that of the cremaster reflex.

The **abdominal reflex** is produced by tickling, scratching, or pricking the skin of the abdomen. It consists in a simultaneous contraction of the transverse oblique and recti muscles of the abdomen, which produces a depression of the abdomen and a pulling of the navel toward the side stimulated.

Several abdominal reflexes should be differentiated upon each side of the abdomen—a superior, median, and inferior. Stroking the abdominal wall in a horizontal direction in the region of the epigastrium or hypogastrium causes reflex contractions of the abdominal muscles, which remain localized at approximately the height of the stimulation. If, on the contrary, the entire length of the abdomen is stroked in a vertical direction, the whole half of the abdomen is contracted, and the

[2] *Deutsch. med. Woch.*, 1892, vol. viii., p. 166.

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maximum of the excursion is found at the height of the navel. This is what we ordinarily call the abdominal reflex (a poor term). With more vigorous irritation, horizontal stroking of the abdomen can produce the general abdominal reflex.

The **interscapular reflex,** which is elicited by stroking the inner edge of the scapula, consists in an adduction of the shoulder-blade. It is frequently absent.

The **gluteal reflex,** a contraction of the gluteal muscles produced by an irritation of the skin about the gluteal region, is also inconstant.

Anal Reflex.—Irritation of the skin of the anus, as by a pin prick, elicits a contraction of the sphincter and externus. It is often absent. (Consult p. 784 *et seq.* and p. 927 *et seq.* in regard to the diagnostic significance of abnormalities of the cutaneous reflexes and their localization in the spinal cord segments.)

TENDON, PERIOSTEAL, AND JOINT REFLEXES.

The **patellar reflex,** or the "knee phenomenon," consists in a contraction of the quadriceps muscle excited by a blow upon the patellar tendon. We may employ the ulnar edge of the hand or, better, the edge of a firm, not too light object—*e. g.*, a percussion hammer.

The **Achilles-tendon reflex** consists in a contraction of the calf muscles excited by a blow upon the Achilles tendon, producing a flexion of the foot. Another method of exciting this reflex is to increase suddenly the tension of these muscles by a passive dorsal flexion of the foot. If the reflex is increased, the latter method causes a series of rapidly succeeding plantar flexions of the foot, which often persist as long as dorsally directed pressure is exerted upon the ball of the foot. The repetition of the flexions apparently depends upon the fact that each contraction of the calf muscles increases the pressure upon the plantar surface, so that the pressure acts later as a renewed blow. We call such an increase of the Achilles-tendon reflex "the foot phenomenon," "foot-clonus," or "ankle-clonus."

The **tendon reflexes** of the upper extremities are extremely inconstant. In healthy individuals a flexion of the hand can sometimes be obtained by striking the flexor tendons at the wrist-joint, a bending of the forearm from the biceps tendons, or an extension from the triceps tendons. If the reflexes are increased a similar result can be obtained, even in the upper extremities, by striking almost any of the tendons. Sometimes an actual clonus can be produced by vigorously flexing the hand dorsally, "hand-clonus" (analogous to ankle-clonus).

Periosteal and **joint reflexes** are produced by striking various bony prominences and joints. They are inconstant in health. The best-known periosteal reflexes are those of the crest of the tibia, and of the ulna and the radius at the wrist-joint.

In testing tendon and periosteal reflexes it is especially important to observe the caution enjoined above—that is, to distract the attention of the patient from the part of the body to be examined, since otherwise the reflex may be inhibited. A very good plan is to interest the patient

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in some subject, and to engage him in conversation while you are testing. Jendrassik's device for reinforcement is often very successful in testing the tendon reflexes of the lower extremities. The patient is directed to lock his fingers and pull strongly, as if tearing them apart, but without separating them. Not infrequently, however, this reinforcement does the opposite to what it usually accomplishes—*i. e.*, it weakens or inhibits the patellar reflex—probably because, instead of actually concentrating his entire attention and effort upon the fingers, the patient renders the muscles of his lower extremities tense by associated movement, and so inhibits the patellar reflex. Another precaution that should always be taken is to be sure that the muscles concerned in the reflex are thoroughly relaxed. This is especially important in testing the patellar reflex. For the latter the sitting posture is most desirable, with the leg to be examined crossed and hanging limply over the other. In bedridden patients the leg is supported below the knee by the hand of the examiner in semiflexion.

CONSTANCY—*i. e.*, FREQUENCY—OF OCCURRENCE OF THE NORMAL SPINAL REFLEXES.

Of the above-described reflexes there are only a few which are really constant, and even these not absolutely. Many are even present only in a minority of cases. According to the researches of Pflästerer, the reflexes are found as follows:

MALES.

Epigastric reflex (upper abdominal wall reflex),	present in 62 per cent.[1]
Abdominal reflex (middle abdominal wall reflex),	" " 99 "
Cremaster reflex,	" " 66 "
Plantar reflex,	" " 98 "
Interscapular reflex,	" " 15 "
Gluteal reflex,	" " 28 "
Periosteal reflex of the anterior tibial edge,	" " 5 "
Periosteal reflex of the lower end of the bones of the forearm,	" " 29 "
Patellar reflex,	" " 98 "
Achilles tendon reflex,	" " 57 "
Biceps tendon reflex,	" " 47 "
Triceps tendon reflex,	" " 48 "

FEMALES.

	Present.	Absent.	Unilateral.	Doubtful.
Plantar reflex	88	11	1	..
Abdominal reflex	92	7	..	1
Interscapular reflex	13	86	1	..
Gluteal reflex	11	89	..	..

RECENT VIEWS CONCERNING THE ORIGIN OF THE REFLEXES.

Formerly it was believed that the spinal cord was the center of all reflexes. This view was based upon the results of animal experiments and upon the observation that most transverse lesions of the cord were associated with increase of the reflexes. Modern neuropathologists, however, following the teaching of Bastian, endeavor to dethrone the cord from its position as a reflex organ. Bastian and others have shown that all the reflexes were found to be completely abolished in some cases in which there is complete transverse lesion of the spinal cord. It was

[1] Cited by K. Geigel, *Deutsch. med. Woch.*, 1892, No. 8, p. 166.

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therefore argued that no transverse lesion could be complete if the reflexes in the region innervated below the injury either persisted or were increased. A few such observations, however, prove nothing, because the part of the spinal cord situated below the obstruction might be affected in such cases by inhibition or by a diminished blood-supply conditioned by lesion of the spinal arteries.[1] In order to determine whether the reflexes of clinical importance have their centers in the spinal cord, it is much more essential to inquire whether there have been cases with persistent reflexes of the lower extremities in which a careful anatomic examination has shown postmortem complete cross-section of the spinal cord. In recent years such cases, absolutely free from question, have been described,[2] and show beyond question that the tendon reflexes, at least, are independent of nervous mechanism beyond the spinal cord. Similar results have been proved by the persistence of the tendon reflexes after decapitation (Laborde).[3] Hence it is plainly incorrect to maintain that all reflexes require the aid of the brain.[4] The discussion has been valuable in more accurately determining the merits of the earlier conception, which argued that all reflexes took place in the spinal cord, and homologous portions of the brain stem. In this connection Jendrassik has formulated a theory of the reflexes which is based upon clinical evidence and which is worthy of careful study. [See also upon this subject, Collins and Fränkel, "Muscle Tonus and Tendon Phenomena," *Medical Record*, Dec. 12, 1903.—ED.]

According to Jendrassik there are spinal and cerebral reflexes, as well as a combination of the two—*i. e.*, reflexes requiring both cerebral and spinal centers for their normal performances.

I. **Spinal Reflexes.**—This division includes *tendon*, *periosteal*, and *joint reflexes*. Their characteristics are as follows: 1. They are generally discharged from parts which possess little sensation. 2. The reflex is associated with no particular feeling. 3. The discharge takes place by means of a simple mechanical irritation, snch as a blow, etc. 4. The intensity of the reflex depends upon the intensity of the irritation, not upon its duration. 5. The reflexes are quite as easily excited in ourselves as in others. 6. The latent time of the reflex, corresponding to its origin in the spinal cord, is the shortest. 7. The ensuing movement is a very simple one and serves a recognizable purpose. 8. Making other muscles tense increases the reflex (reinforcement method of Jendrassik ; see description on p. 779). 9. Slowing of these reflexes never occurs pathologically. 10. Psychical inferences have no effect upon these reflexes aside from distraction of attention, which increases them.

II. **Cerebral Reflexes.**—These are to a large extent the cutaneous reflexes. The scapular, abdominal, cremaster, gluteal, plantar, eyelid, palatal, conjunctival, and anal reflexes belong to this group. Their characteristics are as follows: 1. They are discharged from sensitive spots which are not ordinarily accustomed to a light touch (tickling). 2. The liberation is associated with a specific sensation (prickings, cold, tickling, etc.). 3. Brief stimulation is efficacious for their liberation, just as it is for the spinal reflexes described in the previous paragraph. 4. A light touch has often a more vigorous action than a stronger one; individu-

[1] Gerhardt, "Über das Verhalten der Reflexe bei Querdurchtrennung des Rückenmarkes," *Deutsch. Zeits. f. Nervenheilkunde*, 1895, vol. vi., p. 127, and Jendrassik, "Über die allgemeine Localisation der Reflexe," *Deutsch. Arch. f. klin. Med.*, 1894, vol. lii.

[2] *Ibid.*

[3] Quoted by Jendrassik.

[4] According to Bastian's and Jackson's view, especially of the cerebellum.

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ality has a decided influence. 5. These reflexes can scarcely ever be liberated by the person himself, and then only very slightly. 6. The latent time is longer and not as constant as with the spinal reflexes. It is quite dependent upon the sensation time and corresponds to the reaction time—*i. e.*, the time which the voluntary reaction demands of a sensory stimulation. 7. The resulting movement is simple, and its principal characteristic is that it shows an effort to escape from the irritation. 8. Increased activity of other muscles never increases the reflex, but may even diminish it. 9. These reflexes are diminished on the paralyzed side in cerebral hemiplegia. 10. They are delayed in cases of delayed sensation. 11. Psychical influences can either diminish or even increase these reflexes; distraction of the attention impairs them.

III. **Complex Reflexes.**—To this group belong reflexes which have complicated "centers," within which the reflex occurs, not as a single movement, but as a series of such—*e. g.*, sneezing, vomiting, swallowing, coughing, urinating, defecating, genital reflex (ejaculation). The characteristics in common are as follows: 1. They are liberated from sensitive places. 2. The liberation takes place with a specific sensation, which plays even a greater rôle in the origin of the reflex than in those of the cerebral group. 3. The liberation requires protracted stimulation. 4. Individuality has a great influence upon the occurrence of the reflexes. 5. The stimulation which produces these reflexes is a specific and complicated one. 6. The latent time is longer than for any of the other reflexes. Their liberation seems to require a sort of summation of stimuli. 7. The resulting movement is very complicated and bilateral; several muscle groups take part, and in some of them the reflexes act antagonistically. 8. Muscular activity produces a certain enfeebling in their action. 9. Psychical influences produce a great effect. 10. Reflexes of this group belong to the vegetative functions.

The distinction between Groups III. and II. is essentially this: in the latter the sensation is transposed directly into simple reflex movement; whereas in the former the sensation—*i. e.*, the cortical stimulation—first of all excites a complicated reflex center to activity. This center is composed of different separate centers, and within the main center the reflex process then takes an independent course. Should this subdivision be accepted, the writer would call attention to the fact that the peculiarities stated by Jendrassik as characteristic of the individual groups do not always materialize. It would lead us too far, however, to discuss these points in detail. For the last group of Jendrassik the writer would suggest the name of corticonuclear reflexes. Those which also involve spinal areas of innervation might with equal propriety be designated as cerebrospinal reflexes. In the individual cerebronuclear reflexes the cerebral factor plays a varying but important part, as is shown by the fact that completely unconscious individuals never cough nor sneeze, but, on the other hand, they can under some circumstances still swallow and normally urinate and defecate, even if unwittingly. Various examples might be added to show how dependent the reflexes of Groups II. and III. are upon the cerebrum; but the writer will allude only to the following: the diminution of the cutaneous reflexes over the anesthetic area of the hysteric; the purely cerebral origin of the plantar reflex in ticklish persons, in whom it may be elicited by threatening to tickle them, even without touching them; and the occurrence of vomiting evoked by some disgusting conception.

Recognizing the above theories of the origin of reflexes, we should now attempt to explain their clinical manifestations under pathologic conditions, especially in the case of an interrupting focal lesion of the brain and spinal cord.

Cerebral Hemiplegias.—The tendon reflexes are here, as a rule, preserved, because they come under Group I. (the spinal). In the beginning they may be lost, probably on account of the inhibiting action of the lesion, but later they are usually increased, because of the complete abolition of cerebral inhibition. (See p. 746 *et seq.*, Active Contractures.) The behavior of the cutaneous reflexes (Group II.) in cerebral hemiplegia is explained by the supposition that, for the most part, the voluntary tracts (pyramidal tracts) are either identical with, or, at least, run very near, the motor limb of the cortical reflex arc. We therefore

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generally find upon the paralyzed side diminution or disappearance of the cutaneous reflexes. In hemiplegia of indirect origin—*i. e.*, when the lesion is not immediately of the pyramidal tract—the cutaneous reflexes may be retained, although they, too, are in the beginning usually diminished. This is explained by the supposition that the lesion here does not permit the passage of the voluntary impulse, while it offers no obstacle to the reflex impulse. The persistence of the cutaneous reflexes upon the hemiplegic side can be regarded as a favorable prognostic sign, because it argues for an incomplete interruption of the motor tract. The condition of the combined reflexes (Group III.) in cerebral hemiplegia depends upon the varying degree of influence which the cerebral factor has upon their origin. As a general rule they are not affected, because bilaterally innervated. The more important of these reflexes, the bladder and rectal, will be discussed later.

Transverse Lesions of the Spinal Cord.—If Jendrassik's conception is correct, the tendon reflexes must in general be considered pure spinal reflexes, and the cutaneous, on the other hand, cerebral reflexes. Clinical experience does not seem to coincide perfectly with this conception, for we ordinarily find both the cutaneous and the tendon reflexes increased in transverse spinal cord lesions, such as those caused by myelitis. How is this to be explained? The persistence of the tendon reflexes is clear enough, and their increase is easily explained by supposing that the inhibitory fibers running in the lateral tracts have been cut off; but how can we explain the increase of the cutaneous reflexes, if, as Jendrassik assumes, they really have their reflex arc in the brain? Jendrassik considers that these reflexes, which in transverse cord lesions are ordinarily regarded merely as accentuated cutaneous reflexes, are in reality pathologic cutaneous reflexes, which normally do not occur in this shape at all, but in transverse lesions take the place of the normal cutaneous reflexes which have been destroyed. He offers the following arguments in support of this theory; The normal cutaneous reflexes of the lower extremities can be elicited only from the sole of the foot, no matter how vigorous the stimulation may be; while the pathologic cutaneous reflexes in cross-lesions of the cord can be elicited by irritating any part of the lower extremities. The normal plantar reflex is associated with a sensation of pain and tickling, and the time of its appearance depends upon the occurrence of such sensations (best recognized in the delayed transmission of pain in tabes dorsalis); the pathologic reflexes, on the contrary, are not dependent upon this sensation nor are they associated with it. The physiologic reflex even exhausts after repeated testing even though it is very vigorous; whereas the pathologic reflex never exhausts; it can be elicited again and again. A light touch will very easily stimulate the physiologic cutaneous reflex; whereas the pathologic reflex is in direct ratio to the intensity of the irritation. The pathologic cutaneous reflex always consists of a maximum flexion of the thigh, outward rotation of the knee, and dorsal flexion of the foot (in rare instances the reverse—*i. e.*, an extension of the thigh and plantar flexion of the foot). Both of these types are very different from the physiologic reflex, which is essentially a movement of escape, and consists of a dorsal flexion of the foot, accompanied by a very slight movement of the muscles of the thigh and pelvis. Jendrassik explains the appearance of these vigorous abnormal reflexes of transverse cord lesions in the place of the normal by assuming that the sensory impulses which are cut off at the point of the lesion make for themselves a sort of lateral path which is not normally in use. There are cases of acute transverse lesions of the cord in which the cutaneous reflexes are diminished instead of increased. In them he assumes that the lower part of the spinal cord is also affected, so that these pathologic reflexes cannot take place perhaps because of an anatomic lesion (deficient blood-supply), or perhaps on account of an inhibitory influence by the lesion. A similar explanation will account for the loss of the tendon reflexes which we see so often in acute traumatic transverse division of the cord. The increase of the tendon reflexes, and the appearance of the pathologic cutaneous reflexes which is observed later in the course of these cases, are sufficient evidence to support such an explanation.

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Jendrassik's theory of the genesis of the reflexes has many arguments in its favor, but in the writer's opinion is very weak in one point—the supposition that in cross-lesions of the cord the reflexes which belong to parts supplied by the lower portion of the cord are not the ordinary retained reflexes, but newly formed pathologic reflexes. This assumption seems permissible in the case of the vigorous and altered reflexes of the lower extremities, as they were described above. But it compels us to assume, should the cremaster and abdominal reflexes, etc., be preserved in a myelitis of the upper dorsal cord with complete motor and sensory paralysis, that such circumscribed reflexes must arise by another path than the physiologic, and that in such complete paralysis the physiologic cerebral reflex is injured. The persistence of these reflexes, whether weakened, normal, or increased (any one of these conditions is possible in transverse lesions), is most readily explained by assuming that their center or, better expressed, their reflex area lies below the transverse lesion, and is thus preserved. On the other hand, the absence of these reflexes in cerebral hemiplegias seems difficult to reconcile with such a supposition. The following theory will, the writer believes, clear up the difficulty: The cutaneous reflexes which Jendrassik considers to be purely cerebral are in reality like his third group (p. 781), corticonuclear—*i. e.*, cerebrospinal in so far as they belong to spinal areas. They have a reflex center or, better,[1] a lower short reflex arc in the cord, and in addition a more highly differentiated "upper" reflex arc in the brain (see Fig. 368). Under ordinary circumstances an accompanying excitation of the cerebral arc is essential to the operation of the reflexes. This arc when so stimulated transmits a centrifugal impulse to the spinal reflexes. In a motor hemiplegia of cerebral origin, the lesion (*ab*, Fig. 368) interrupts the centrifugal tract from the brain to the spinal reflex center (this tract is either identical with or situated very near to the pyramidal tract). Hence the cutaneous reflexes upon the paralyzed side disappear. In a transverse lesion of the cord (*cd*, Fig. 368) the cerebral reflex arc is also interrupted, and one would naturally expect a similar disappearance of the cutaneous reflexes; but this does not occur, because the lesion, by interrupting the sensory condition in the cord, in a measure dams the sensory stimulation. Therefore the peripheral impulse must find a path in the region of the lower cord segments. It usually selects the usual path—*i. e.*, the formed spinal reflex arc of the corresponding cerebrospinal reflex—and so the cutaneous reflexes become purely spinal.

This explains how many of the preserved reflexes—*i. e.*, the abdominal and cremaster—retain their complete physiologic distribution. It also explains how other reflexes, by means of the damming up of exciting pulses at the lesion, attain both abnormal intensity and distribution by transmission of the impulse to neighboring paths.

The stasis of the excitation at the level of the lesion is sufficient to

[1] Compare p. 927 *et seq.*, Segmental Localization. The expression "reflex center," employed merely for the sake of brevity, is inaccurate and incorrect, and the conception according to which it was first used is no longer tenable.

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account for the abnormal intensity of the reflexes in transverse lesions, even without the assumption of abolition of the reflex inhibitory tracts (although the latter shall not be contested).

On the other hand, in cerebral hemiplegia since extensive by-paths in the whole cord and a great part of the brain are open to centripetal excitation (Fig. 368, lesion *ab*), there is no reason for an actual stasis, and likewise no ground for assuming a pure spinal stasis of reflexes which otherwise come from the brain. These, therefore, from lack of cerebral excitation, disappear, or through insufficient exertion appear weak. Perhaps we can assume that the centripetal impulses here are dispersed so completely through the wide-open tracts of the nervous system that they become inert and run, like a lightning-rod, into the ground. The writer can claim more than a mere diagrammatic representation for this theory of damming back and jumping over of sensory irritation in the cord, because Golgi has demonstrated the existence of branched sensory collaterals (Fig. 368) and has shown that paths for discharge are open upon all sides, and that it depends only upon the intensity of the resistance which of these pathways will be selected by the stimulation—*i. e.*, the reflex. The diminution of the reflexes sometimes observed in very acute, especially traumatic, cord affections is, according to this theory, to be attributed to inhibition or to injury of the lower cord segments from circulatory disturbances, etc.

The author's theory, contrasted with Jendrassik's, simplifies the scheme of the reflexes. We should differentiate physiologically between only two groups of reflexes. The first group would comprise *purely spinal* or, better, *purely nuclear* reflexes because some of them occupy the region of the cranial nerves, and would include the tendon, periosteal, and joint reflexes. The second group, the cerebrospinal—*i. e.*, cerebronuclear—reflexes, would include both the normal, simple, cutaneous, and mucous membrane reflexes and the combined reflexes of Jendrassik's third group—bladder, rectal functions, etc. In the second group the brain and spinal cord (or the cerebral cortex and cranial nerve nuclei) act normally together—*i. e.*, the activity in a lower (nuclear) reflex arc is, under physiologic conditions, discharged by the cortex. In transverse lesions of the cord, reflexes of this second group may proceed merely by the spinal path, and so be increased or even deformed by reflex damming. This conception seems to the writer the only one which corresponds to our clinical experience. That of Jendrassik is essentially opposed to the facts in so much as it does not recognize the short spinal path for cutaneous reflexes which has been localized by pathologic and experimental findings (see Table upon p. 930 *et seq.*), and which is made use of for the purposes of local diagnosis.

DIAGNOSTIC AND PROGNOSTIC SIGNIFICANCE OF QUANTITATIVE ALTERATIONS OF THE REFLEXES.

The demonstration of the presence of a reflex is of greater diagnostic significance than the demonstration of its absence, because its presence is

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conclusive evidence of an intact reflex arc; whereas, although its absence may mean that the arc is interrupted, it may also mean that the reflex is affected by a mere inhibition or by remote influence from some circulatory disturbance. Similarly, an increase of a reflex is ambiguous. The latter may be caused by lesions which directly stimulate the reflex centers or tracts, or by those which remove inhibition or injure inhibitory fibers. The pathologic relations of the reflexes are therefore evidently complicated. Only a comparatively few types will be mentioned.

The relation of altered reflex to lesions which are situated in the lower (nuclear) reflex arcs is perhaps the most distinct (see p. 782 *et seq.*, and Fig. 368). Any such lesion, whether of the sensory limb, nucleus or motor limb, is capable of diminishing or destroying the reflexes. Such reflexes as the cutaneous, which are purely cortical, and which pass through the lower and upper reflex arcs, require intactness of the lower reflex arc for the proper display of the reflex, because, according to the author's idea, the upper reflex arc has no functional independence, but merely incites the lower reflex arc to activity. So anatomic lesions of its nuclear arc cause a diminution or disappearance of that reflex tendon. The disappearance of the tendon reflexes in tabes, and the disappearance of all the reflexes in peripheral neuritis and other peripheral paralysis, are examples in point. On the other hand, an accentuation of the reflexes results from an increase of irritation in the lower reflex arc—*e. g.*, in tetanus, in hysteric and neurasthenic conditions, and occasionally in the beginning stages of neuritis, especially while associated with hyperalgesia. The last may sometimes occasion difficulty in diagnosis.

The reflexes in cerebral hemiplegias and in transverse lesions of the cord have been so fully discussed in the preceding section (p. 781 *et seq.*) that only a few diagnostic points need be added. Cerebral hemiplegias can sometimes be diagnosed by the diminution of the cutaneous reflexes, and the alterations of the tendon reflexes (increase or decrease) upon the paralyzed side, even during the "stroke," when the patient is still unconscious, and while, therefore, the motility cannot be directly tested. The abdominal reflexes, particularly the cremaster in men and the crural in women, are especially significant, unless these reflexes are absent upon both sides on account of inhibitory influences. In the latter event the criterion fails, and the condition is evidently more serious. Preservation of the cutaneous reflexes upon the paralyzed side should always be considered a relatively favorable prognostic sign, because it shows that the upper reflex arc through the cerebrum, whose motor limb is practically identical with the voluntary motor tract, has not been entirely destroyed (see p. 781).

In transverse lesions of the cord we generally find an accentuation of the reflexes whose nuclear arc lies below the injury. A decided increase of the cutaneous reflexes—*i. e.*, the appearance of pathologic skin reflexes—implies a serious lesion. On the other hand, a decided diminution of the reflexes originating beneath the lesion shows either

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that an inhibitory influence proceeds from the injury or that there exists a coincident involvement of the spinal segment situated below the lesion. Such an involvement may be the result of circulatory disturbance or of a distinct longitudinal lesion of the cord. If traumatic transverse lesions cause a disappearance of the tendon reflexes of the lower part of the body, Bastian, Kocher, and others consider that this lesion is then probably complete. The author's theory would attribute this to a decided inhibitory action in the lower segment, and perhaps also to a circulatory impairment from lesion of the spinal arteries. At all events, later in the course of these cases, although the continuity of the cord is not re-established, the tendon reflexes sometimes reappear. Our discussion upon the causes of the accentuation and variation of the cutaneous reflexes depending upon transverse lesions presents an important consideration for the operative treatment of spinal cord compression (spondylitis, etc.)—viz., the nearer the reflex approaches to the normal, the more liable the cord is to be merely compressed and otherwise intact. Again, the more profound and extensive the cross-lesion, the more pronounced will be the signs of what we have called reflex damming. In other words, decided accentuation or variation or loss of the reflexes argues in favor of serious lesion and against simple compression. The preservation of certain reflexes is very important in cross-lesions, because it enables us to localize the level and the extent of the lesion (p. 928 *et seq.*). The loss of them is, as we have seen, less serviceable for localizing diagnosis, because of so many indirect influences which can affect them.

In spastic paralyses it is very difficult to make any diagnostic use of certain reflexes. If the paralyzed muscles are strongly contracted, neither cutaneous nor tendon reflexes can be elicited from the very tense muscles. Sometimes this is especially noticeable in tetanus, where the permanent tension of the muscles prevents the ordinary reflexes, and yet the appearance of jerks at any irritation leads us to conclude that there is an accentuation of the reflexes.

The behavior of the vesical and rectal reflexes will be specially considered on p. 939 *et seq.*

QUALITATIVE ALTERATIONS OF THE REFLEXES; PATHOLOGIC REFLEXES.

Many so-called pathologic reflexes should be regarded from what was said upon p. 783 as deformations of the normal reflex, depending upon an encroachment of the reflex impulses upon pathways which become accessible to the impulse only because an obstruction is intercalated in the ordinary reflex tract in consequence of reflex damming. Frequently by this procedure the original reflex is only modified, so that, although disfigured, we can still recognize it. In other cases, however, reflexes occur, in quite an analogous way, entirely as pathologic phenomena. It is impossible to enumerate here all those which are observed in transverse lesions of the spinal cord; but there is a pathologic plantar reflex which is so frequently observed in such lesions that it deserves a brief notice. Irritation of the sole of the foot without stimulating the plantar reflex of the toes or even dorsal flexion of the ankle-joint often causes extensive contraction of the external rotators of the thigh. Not infrequently such movements are combined with reflex movements of quite distant muscle territories—*e. g.*, of the abdomen, of the leg, or of the arm. This pathologic reflex can

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frequently be excited by irritating other parts of the extremity, such as the thigh.

Babinski's phenomenon or the Babinski reflex (phenomen des orteils) consists of a dorsal flexion of the toes, but more especially of the great toe [and a simultaneous flexion of the other toes.—ED.], when the sole of the foot is irritated by pricking or stroking with a moderately sharp instrument. This reflex comes under the head of the pathologic plantar reflexes, and is sharply contrasted with the normal plantar flexion of all the toes to irritation of the sole of the foot. Babinski found that this reflex depended nearly always upon a lesion of the pyramidal tract. H. Schneider,[1] [and especially Walton and Paul, *Journal of*

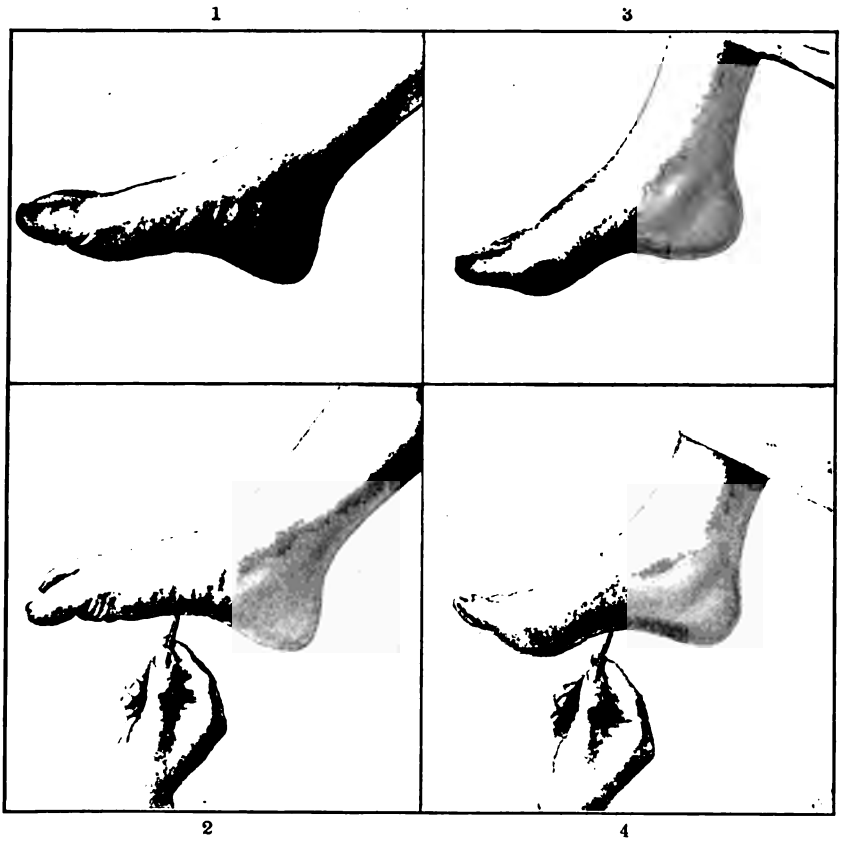

FIG. 299.—Plantar reflex: 1, Foot at rest; 2, plantar flexion (normal); 3, foot at rest; 4, dorsal flexion of great toe (Babinski's reflex).

Nervous and Mental Diseases, 1903.—ED.] and many other authors since, have confirmed Babinski's original observation. Schneider explains this reflex as follows: In the ordinary reflex of the sole of the foot the plantar flexion of all the toes depends upon a cortical component of the reflex; whereas the dorsal flexion (especially of the great toe) in Babinski's reflex depends upon a spinal component of the reflex. With a lesion of the pyramidal tract the reflex for the plantar flexion is interrupted, but not that for the dorsal flexion. Babinski's reflex may also be produced by an increase of the spinal component of the reflex

[1] *Berlin. klin. Woch.*, 1901, No. 37.

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just as well as by a lesion of the pyramidal tract; therefore, its demonstration does not prove absolutely a lesion of the pyramidal tract. It seems to the author, however, that the hypothesis of deformations of the normal reflex which he has advanced will explain the transformation of the original plantar reflex into Babinski's reflex quite as well as Schneider's hypothesis.

In transverse lesions of the spinal cord, the frequent occurrence of ejaculation and erection reflexes, excited by light stroking of the genital or anal region, of the perineum or of the thigh, should be mentioned, as well as bladder and rectal emptying from stroking a bed-sore, etc. The testicle reflex described by Kocher in spinal-cord affections consists in a lateral bending of the spinal cord toward the irritated side when the testicle is firmly pinched. The author can confirm the appearance of this reflex in transverse lesions of the spinal cord. Whether it should be considered as pathologic is not easy to determine, because the attempt in healthy individuals—*i. e.*, those with normal sensibility—is very painful and possibly injurious.

Other abnormal reflexes may occur both from cutaneous and from tendon reflexes without assuming congestion, but simply increase of reflex irritability. In this way the impulse can no longer be limited within the stimulated territory, but diffuses itself according to Pflüger's laws in horizontal and then in longitudinal directions (p. 777). These phenomena really belong to the section upon Quantitative Alterations.

V. EXAMINATION FOR TROPHIC DISTURBANCES.

1. TROPHIC DISTURBANCES OF THE MUSCLES.

(*a*) Increase in Volume of the Muscles; Hypertrophy and Pseudohypertrophy.

True hypertrophy of the muscles—*i. e.*, an enlargement with increased strength—is a very rare pathologic condition. It occurs, however, in Thomson's disease and in true congenital muscular hypertrophies. The latter are rarely observed, and still very imperfectly understood. In most cases the pathologic increase in volume of the muscles is not an actual hypertrophy, but a pseudohypertrophy, since it depends not upon an increase of the contracting muscular fiber, but upon a growth of interstitial connective tissue and of fat. The variety of muscular atrophy known as pseudohypertrophic progressive muscular atrophy furnishes the best example of this pseudohypertrophy. Now and then other myopathic forms of chronic progressive muscular atrophy show pseudohypertrophy in certain of the affected muscles.

(*b*) Decrease in Volume of the Muscles; Muscular Atrophy.

Inactivity Atrophy; Simple Non-degenerative Atrophy.—This means a diminution of the contractile substance which goes on in every muscle that is not used. An absolute increase of interstitial connective tissue does not accompany it, and for this reason this type is designated as a non-degenerative atrophy. Paralysis, mechanical fixation of an extremity, or a restrained position of the latter from some painful affection may gradually produce inactivity atrophy. This atrophy becomes profound only when the immobility is quite absolute. The differentiation of inactivity atrophy from degenerative atrophy is easy without the aid of electrical examination, because the volume of that part of the body which shows the atrophy and

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which is no longer moved is diminished *in toto;* while in degeneration atrophy frequently individual muscles or groups of muscles are spared or are predominantly involved in the atrophy.

The above rule, that an inactivity atrophy which depends upon partial inaction of the muscles never attains a very high degree, does not hold good in the case of paralysis appearing in a growing individual. The physiologic growth then seems to be decidedly inhibited by limited inaction—*e. g.*, we frequently observe very marked atrophies in the cerebral paralysis of children, and we can definitely determine that these are not degenerate by means of the anatomic localization of the primary lesion and by electric and anatomic examination of the muscles. It should nevertheless be remembered that some of the marked atrophy consequent upon cerebral paralysis in children is possibly also of a degenerative nature, and is brought about in the manner indicated in the footnote on p. 790.

Degenerative Atrophy.—Degenerative muscular atrophies are distinguished from inactivity atrophies by the presence in the affected muscles of a pathologic proliferation of interstitial connective tissue, which develops as well in those idiopathic muscular atrophies called progressive as in the so-called atrophic paralyses.

Progressive Muscular Atrophies.—These include *myopathic, neuritic,* and *spinal* (better nuclear) forms, depending upon whether the muscles are primarily diseased or whether they atrophy secondarily to a chronic neuritis or to a chronic degeneration of the large ganglion cells or nuclei of the spinal cord (or the nuclei of the cranial nerves). In all three forms certain muscles and muscle groups gradually disappear. The atrophy affects the muscles individually and increases gradually, but an entire extremity does not show diffuse atrophy until an advanced stage of the process has been reached. The muscular power diminishes in proportion to the disappearance of the muscles. This is in contrast with *atrophic paralysis,* in which the paralysis appears first, and then the atrophy. The question whether a muscular atrophy is myopathic, neuritic, or nuclear is made easier to answer by knowing that each of these forms has a characteristic way of spreading.

(A) Under the *myopathic* form, lately designated by the term *dystrophic,* several types have been described. Three of the most important are:

1. Erb's juvenile muscular atrophy (progressive muscular dystrophy), which begins in the shoulder girdle. There are different types of this group.

2. Leyden-Moebius's juvenile form; this begins in the lower extremities. Pseudohypertrophy is closely related to this form.

3. Duchenne's infantile variety; this begins in the face.

(B) *Nuclear (spinal) muscular atrophy* begins in the small muscles of the hand and involves early the bulbar muscles—*i. e.*, produces the picture of bulbar paralysis (better, bulbar atrophy).

(C) The *neuritic* or *neural type of progressive muscular atrophy* is still the least understood. It begins most frequently in the lower

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extremities, in the domain of the peroneal nerve (peroneal type), and causes a pes varus or equinovarus. Ordinarily, disturbances of sensibility are associated with this form.

Other methods of differentiating these three groups are as follows: *Fibrillary twitching* (p. 748) and *reaction of degeneration* (p. 812) are demonstrable in the spinal and neuritic forms, but rarely in the myopathic. Disturbance of sensibility, even though very slight, points to the neuritic type. Myopathic muscular atrophy is, as a rule, hereditary, and affects almost exclusively young individuals. The same is true for some of the neuritic cases; but the spinal form is almost entirely confined to older people without neuropathic heritage.

The examination of the nervous system by means of the newer methods based upon more minute histology of the ganglion cells will probably determine that the boundaries of the individual forms are in no way so sharply defined as we have thus far assumed. In this regard the entire subject of muscular atrophies, especially its neuropathology, needs anatomic revision.

Secondary Degenerative Muscular Atrophies After So-called Atrophic Paralyses.—Paralyses are described as atrophic when the cause of the paralysis deprives the muscles not only of the impulse of the will, but also of the trophic influence of the cells of the nerve nuclei—*i. e.*, the cells of the cord situated in the gray anterior horns.[1]

To avoid confusion with the *simple inactivity atrophies* (p. 788 *et seq.*), we should remember that the *secondary degenerative atrophies* depend entirely upon a lesion of the peripheral nerves—*i. e.*, the paralysis is located either in the nucleus or peripherally to it (nuclear and peripheral paralyses).[2]

These atrophies ordinarily follow the onset of the paralysis quite rapidly—*i. e.*, within a few weeks. They affect individual muscles very differently, depending upon the degree of the paralysis, and ordinarily attain a very high grade, even to complete disappearance of individual muscles. They present the reaction of degeneration often even before the diminution of volume has become evident (see p. 812). They are also frequently associated with fibrillary twitchings (p. 748). Such atrophies are always an indication of severe paralysis and require a long time for recovery—many months—even in favorable cases. The prognosis is not always absolutely unfavorable in lesions located peripherally to the nuclei, since the regenerative power of peripheral nerves is exceedingly good. The prognosis of the degenerative atrophies which arise from a paralysis of the nuclei themselves is, on the other hand, absolutely hopeless, because in such cases regeneration does not occur

[1] The ordinary assumption that these nuclear cells preside over this trophic influence is scarcely correct. However, as centers of spinal reflexes—*i. e.*, of the tendon reflexes—they probably preserve the muscle tonus and prevent a complete muscular inactivity if the muscles are paralyzed above the nuclei.

[2] In reference to the rare cases of degenerative muscular atrophy following upon lesions of the central motor neuron, and its possible explanation in the extension of the descending degeneration to the peripheral neuron, the reader is referred to the article of Steinert ("Cerebrale Muskelatrophie," *D. Zeits. f. Nervenheilk.*, 1903, vol. xxiv., parts 1 and 2).

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(acute, subacute, and chronic poliomyelitis). If a degenerative paralysis does recover, the volume of the muscle gradually returns, following the reappearance of motility and improvement in the electric irritability.

2. TROPHIC DISTURBANCES OF THE SKIN.

Ordinary Decubitus.—This occurs in all sorts of severe diseases, and especially in transverse lesions of the spinal cord. It consists of necrotic processes in the skin and subcutaneous tissues of those parts which endure the principal weight of the body while lying in bed—viz., the sacrum, the trocanters, and the heels. In mild cases there is simply an exposure of the corium from destruction of the epidermis; in more severe cases, a deep necrotic destruction of the tissues even down to the

FIG. 300.—Perforating ulcer in tabes. *x*-ray plate showed involvement of the bone (Dr. J. J. Putnam, Massachusetts General Hospital).

bone. The phenomenon is practically a pressure necrosis. Since decubitus is never found in health, we are naturally inclined to consider it due, in a certain sense, to some trophic disturbance, not to an actual affection of trophic centers or trophic nerves, as it is so common in diseases which have no nervous involvement, but rather to the generally depressed nutrition, in which the skin shares. Decubitus is frequent in transverse lesions of the spinal cord because the enforced quiet is often responsible for impoverished nutrition, the difficulty in moving the patient favors more constant pressure upon the dependent places, and, even if movement is possible, the impaired sensibility prevents recognition of inequalities, such as folds in the bedclothes, which a healthy person would instantly avoid. Disturbance in the functions of the bladder and rectum, so frequent in these conditions, often keeps the sacral region unclean, and so gives rise to infection.

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Acute Unilateral Decubitus.—The trophic influence of the nervous system seems to be more striking in the case of the acute unilateral decubitus. This consists of a rapidly increasing necrosis of the skin over the sacrum, which is observed upon the side of motor involvement in severe cerebral hemiplegias, and upon the side of the sensory paralysis in spinal hemiplegias. The unilateral character of the phenomenon shows the influence of the nervous system, but at the same time the actual trophic elements of the nervous system are not apparently responsible. It is quite possible that in cerebral hemiplegias the essential fault is disturbance of sensation, which robs the patient of the instinctive protection against considerable pressure upon the

FIG. 301.—Saber shin (Dr. Joseph Collins, New York City Hospital).

anesthetic side. Acute unilateral decubitus is almost always an unfavorable prognostic sign, apparently because it arises only in very marked paralysis; but recovery sometimes occurs.

Changes in the Skin Over Paralyzed Parts.—The skin over peripherally paralyzed parts, more noticeably the hands, frequently presents a characteristic thin, atrophied, shining appearance, the so-called *glossy skin*. Conversely, an increase in the subcutaneous fat frequently masks the muscular atrophy, especially in the cerebral, but also in the spinal paralyses of children.

Other Trophic Changes of the Skin.—It is impossible to

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discuss here all the changes of the skin which we meet with in diseases of the nervous system. We shall merely mention the presence of pigmentation, of abnormalities in the formation of the epidermis and in the growth of the hair, of deformities of the nails (onychogryphosis), of the loss of the nails (alopecia unguium), of herpes zoster, of symmetric gangrene (maladie de Raynaud, syringomyelia), of panaritium (Morvan's disease; syringomyelia), of Dupuytren's contracture, and of perforating ulcer of the foot (see Fig. 300). All these occur predominantly with diseases of the peripheral neurons, and for their description we must refer to special pathology.

3. TROPHIC DISTURBANCES OF THE BONES AND JOINTS.

In all paralyses occurring in early youth, whether peripheral or central (cerebral and spinal paralyses of children), the growth of the bone

FIG. 302.—Tabetic arthropathy of left ankle (New York City Hospital).

is impeded; on account of such an inhibition of growth and the associated degenerative or inactivity atrophy, the paralyzed extremity is much smaller.

An abnormal *fragility* of the bones, which is responsible for the so-called spontaneous fractures, occurs in tabes dorsalis and syringomyelia.

Joint affections are observed in the most diverse diseases of the nervous system. They depend frequently upon purely mechanical

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causes (destruction of a joint on account of the suspended paralyzed extremity, or of contractures), yet sometimes they are attributed to actual trophic disturbances. Even here this expression does not mean that the disturbances depend wholly upon a lesion of the nervous system, nor can we consider that a nervous apparatus exists whose only function is trophic. The joint affections which occur in tabes, and which we designate as *tabetic arthropathy*, should be included under such trophic disturbances. They are ordinarily characterized by their onset, which is abrupt and painless, by the presence of a considerable

FIG. 303.—Tabetic arthropathy of the knee (Dr. Joseph Collins, New York City Hospital).

fluid effusion, by loosening of the joint, and by a growth of bone and cartilage, leading to deformity of the joint. The knee-joint is most commonly affected, and soon becomes an actual tottering joint.

These specific peculiarities of tabetic arthropathies argue against the purely mechanical explanation which has been so frequently assumed—viz., that the joint suffers repeated injuries merely on account of the ataxia. Other reasons for assuming a trophic influence are the characteristic wearing away of the bones (eburnation fracture of the bones in the direct neighborhood of the diseased joint) and their insensibility to tuning-fork vibrations (p. 765). Even here the author does not consider it necessary to assume the existence of special trophic nerves to explain the joint affections and the bone brittleness of tabes, because

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hemiplegia be situated in the pons, peduncles of the brain, or the internal capsule. In the beginning the paralyzed extremities ordinarily appear warmer and redder; later, colder and more cyanotic than normal. What causes these peculiarities is not yet fully understood. Since the cerebrum, as shown by the action of psychic irritation, possesses an influence upon the vasomotors, the initial heat and redness can be explained by assuming a paralysis of the vasomotor tracts of the brain. The vicarious action of the vasomotor centers of the spinal cord, however, quickly transforms the condition. The coolness and cyanosis of the skin of the paralyzed side, which appear later and which ordinarily continue as long as the paralysis lasts, have nothing to do with the vasomotor action (though this is opposed to the ordinary supposition), but depend upon a stagnation of the venous blood, caused by the immobility of the extremities. Muscular action in movement of an extremity, as is well known, has great influence in propelling the venous blood forward.

In *transverse lesions* of the spinal cord, the higher the lesion the more profound is the vasomotor paralysis of the lower half of the body. In lesions of the oblongata, the paralysis of the cutaneous vasomotor center from dilatation of the vessels may lead to a fatal sinking of the blood-pressure, accompanied by marked cyanosis and coolness of the peripheral parts. In transverse lesions situated below the oblongata, the paralysis of the vasomotors (vasoconstrictors) affects the territory innervated by that part of the spinal cord situated below the lesion, because the vasoconstrictor impulses of the oblongata, except those supplying the head, run downward through the spinal cord and leave the cord along with the motor nerves for the same territory to merge into the sympathetic system. The territory showing motor paralysis then also shows vasomotor paralysis. This is made evident by increased temperature and redness of the paralyzed limb. Yet this paralysis is ordinarily slight and often transitory, because the vasomotor apparatus situated beneath the lesion, as well as the sympathetic fibers of the spinal cord arising above the lesion, are able to functionate vicariously for the nerves which have been cut off. Later, the paralyzed parts frequently become cool and cyanotic on account of the immobility, as in cerebral hemiplegia. *Priapism*, so frequently observed in transverse lesions of the spinal cord, probably depends upon a vasomotor paralysis. The vasoconstrictor fibers of the face probably first issue from the spinal cord in the region of the upper dorsal cord (according to animal experiments on the spinal cord, see p. 934). Therefore they may be affected in all lesions of the spinal cord situated above the sixth dorsal nerves. As yet we know nothing definite about the behavior of vasodilator nerves in lesions of the brain and spinal cord.

Tache cérébrale, Trousseau's spots, dermographism, are the different names applied to a cutaneous vasomotor phenomenon of unexplained origin. It probably depends, however, upon changes in irritability of the vasomotor nerves. It consists of a deep-red color of the skin, often accompanied by the formation of a wheal wherever the skin is irritated—*e. g.*, by light scratching of the finger or the head of a pin. It occurs in purely functional diseases, sometimes in brain diseases (especially meningitis), and not rarely in spinal cord diseases.

We must refer to special pathology for the description of the characteristic redness of the skin observed in erythromelalgia.

VII. EXAMINATION OF DISTURBANCES OF SECRETION.

Abnormalities of sweat secretion are common, but as yet we cannot accord them much diagnostic significance. Hemihyperhidrosis and hemianhidrosis—*i. e.*, unilateral increase or absence of sweat production—often occur physiologically in otherwise healthy persons. In such a case a suspicion of a disease of the sympathetic system is natural. We also observe hemihyperhidrosis in syringomyelia. In hemiplegias

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of cerebral origin, sweating of the affected side is sometimes more, sometimes less, marked than upon the healthy side; frequently, however, it is entirely normal. A decided tendency to increased perspiration is often noted in extremities which are affected with an *acute polyneuritis*, even when there is no fever. This is peculiar and of some diagnostic importance.

We know so little about the alteration of urinary secretion in diseases of the nervous system that it is impossible to draw diagnostic conclusions. A pale, abundant urine of low specific gravity (urina spastica) generally follows attacks of *epilepsy* and *hysteria*. *Transitory glycosuria*, or even *diabetes mellitus* ending in death, often accompanies diseases of the brain, particularly when situated in the posterior cerebral fossa. *Diabetes insipidus* has a predilection for neuropathic individuals, especially neurasthenics.

Pathologic variations of the *salivary secretion* are mentioned in the section upon Examination of the Facial Nerve.

VIII. EDEMA IN NERVOUS DISEASES.

It is not certain that the acute idiopathic edemata really depend upon the nervous system, but certainly the name "angioneurotic" edema (see p. 52), rather prejudices one in favor of such an origin. The edemata which complicate actual nervous diseases are: (*a*) The so-called "*blue edema*" of hysteria. This is discussed under the heading of Angioneurotic Edema (p. 52), and its occurrence was utilized as an argument for assuming that hysteric symptoms are frequently to be attributed to vasomotor disturbances. (*b*) Whether the edema of the paralyzed members in all kinds of *paralysis* is due to vasomotor paresis must be determined in each case; but the immobility and lack of muscular activity, leading to venous stasis (as explained upon p. 796), is generally a sufficient explanation. (*c*) The edema of the paralyzed members in *polyneuritis*. A vasomotor origin in such cases is especially plausible, because if the peripheral vasomotor fibers are involved, the vasomotor disturbance would be exceptionally pronounced, since, as contrasted with cerebral and spinal paralysis, the vicarious action of the auxiliary vasomotor centers, with the exception of those situated on the vessels themselves, is cut off. Still, with the edema of neuritis, we should also consider the possibility of an inflammatory origin.

IX. METHOD OF TESTING THE MECHANICAL IRRITABILITY OF NERVES AND MUSCLES.

1. MECHANICAL IRRITABILITY OF MOTOR NERVES.

Contraction of muscle may sometimes be produced by striking with a percussion hammer the nerve supplying it. This occurs in health under the appropriate conditions—*i. e.*, when the nerve runs superficially upon some firm underlying tissue. This mechanical irritability is increased in *tetany*, especially in the facial nerve (facial phenomenon, Chvostek's phenomenon), and more rarely in *writer's cramp*.

2. MECHANICAL IRRITABILITY OF MUSCLES; IDIOMUSCULAR MECHANICAL IRRITABILITY.

Only vigorous percussion will stimulate muscles in health. Such a stimulation causes a sudden, quick contraction of the bundles of

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fibers which lie in the long axis of the muscles near the point struck. At the same time a smooth local swelling is produced at the spot tapped, or in the long axis of the muscle at a greater or less distance. After the lapse of some seconds this swelling very gradually disappears, or first merges into a series of waves running along in the direction of the fibers.

In all *cachectic conditions* (tuberculosis, carcinoma, etc.) the idio-muscular irritability is apt to be increased and the muscle swelling quite pronounced.

The mechanical irritability is also increased wherever muscle exhibits reaction of degeneration with increased galvanic irritability (see p. 814). The longitudinal wave-like contractions are here exceptionally slight, and much slower than the normal. This phenomenon is called mechanical reaction of degeneration.

X. METHOD OF TESTING THE ELECTRIC IRRITABILITY OF NERVES AND MUSCLES.

1. GENERAL DISCUSSION.

Electrical examination of sensory nerves, including the nerves of special sense, has not yet developed anything of practical value. The following description will therefore be limited to the electrical examination of motor nerves and muscles.

For this purpose a faradic (induced, interrupted) or a galvanic (constant) current is employed almost exclusively. One of the many modifications of the Du Bois-Raymond sliding apparatus with one or two zinc-carbon cells will furnish the faradic, while for the constant current the stationary Léclanche batteries are most useful. These latter are expensive and cannot be transported, so that a portable dry battery with sulphuric-acid-zinc-carbon elements is better adapted for the practitioner's needs. Chardin, of Paris, makes one of the best of the latter type, and also supplies a satisfactory induction apparatus. [Satisfactory and adequate batteries are to be had from dealers in electrical apparatus in nearly all the larger cities of this country.—ED.]

For stimulation we employ flat electrodes or those with a bulb on the end. They are covered with chamois leather, and before being used should be moistened with warm water. If salt is added to the water the current strength is increased, but the electrodes soon become tarnished and spoiled. Different sizes of button-shape and flat electrodes are required, and some of the former should be made with a contact contrivance for opening and closing the current.

For an accurate electrical examination we need an appliance to measure the strength of the galvanic current. Until recently this purpose has been served by a galvanometer divided, according to the absolute strength of current, into milliampères. The apparatus of Gaiffe (Paris), Edelman (Munich), or Edison (New York), can be recommended. The galvanometer is an essential for electrotherapeutic purposes; but the volt meter offers many advantages for electrodiagnosis (see later, p. 806 *et seq.*).

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Medical batteries are supplied with a commutator, or current changer, for quickly changing the poles, and with a contrivance for connecting or disconnecting the individual cells in order to increase or decrease quickly the strength of the current. Finer gradations are obtained by inserting a fluid rheostat into the main circuit. Although quite practical with the ordinary galvanometer, a rheostat in the main circuit cannot be used with the volt meter—*i. e.*, a galvanometer with a very strong resistance in the lateral circuit—and so is not practicable for diagnostic purposes. If we use the volt meter we must employ either a rheostat in lateral circuit or, still better, the "Gaiffe reducteur de potentiel."

The polar method of electrical stimulation is nowadays ordinarily employed for electrical examination. One pole is placed upon the spot to be stimulated, the other upon an indifferent spot, as far distant as possible—*e. g.*, upon the abdomen or the breast. The examination is thus essentially simplified by disregarding the direction of the stream. For all practical purposes both poles of the faradic current may be said to act alike, although not with equal strength. With the galvanic current, on the other hand, there is a fundamental difference between the two poles. This difference is apparent in the so-called laws of contraction of motor nerves and muscles. The cathode of the opening induction current acts most powerfully; therefore it is agreed to employ this for faradic stimulation. On the other hand, in every case where we use the galvanic stream, we must test cathode and anode stimulation separately and compare the results.

The electrode should not be too large if the object is to stimulate isolated nerves or muscles. Too small electrodes are impracticable, because with them the intensity of the current is so great that the pain of the examination is materially increased. For most purposes a button-shaped electrode of 1 to 2 cm. diameter answers the purpose. If we wish to compare the results of different electrodiagnostic examinations uniform electrodes should be used, since the stimulation effect depends not only upon the strength, but also upon the intensity of the current (see p. 807). Erb and Stintzing recommend so-called normal electrodes for all electrical examinations. Erb's normal electrode is 10 sq. cm. in size (a circular surface of 3.6 cm. diameter). Stintzing's electrode, which is serviceable for stimulating small muscles—*e. g.*, of the hand—has a circular surface of 3 sq. cm. (1.8–2 cm. diameter). The indifferent electrode should be as large as possible, in order to decrease the intensity of the current through it, even when a strong current is employed, and so to lessen the pain caused by the passage of the current. The pain is due essentially to electrolizing the skin.

There are some exceptions to the rule given above, that the indifferent, large electrode should be applied as far as possible from the point to be stimulated. These exceptions occur frequently enough to merit mention—*e. g.*, when employing strong currents in diminished irritability, if we should apply the indifferent electrode to the abdomen and then attempt to stimulate the small muscles of the hand, the effect would

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be very confusing. The current passing through the nerves and muscles of the arm would produce contraction of the muscles not only of the hand, but also of the arm; the entire arm would naturally be jerked about, and so confuse us. In such a case it would be better to apply the indifferent electrode to the other surface of the same hand. The employment of the largest possible indifferent electrode establishes a great difference in current intensity between the two electrodes, so that an almost exclusive, and practically a polar, action results at the differentiating electrode. Such devices should be made use of upon other parts of the body.

A complete electrical examination, evidently, must include testing each muscle and nerve. It consumes a great deal of time, because control tests must be made in order to verify results. In fact, the electrical examination of an extensive paralysis requires more time, skill, and patience than any other kind of examination, if it is to be accurate and thorough, and leads to many serious errors if it is carried out hastily or carelessly. An accurate testing of one muscle is of more service than the careless examination of all the paralyzed muscles. Multum is better than multa; quality than quantity. A complete examination of all the muscles implicated in a paralysis is impracticable for a busy practitioner, especially if, in the interest of prognosis, continued changes in the reaction during the course of the disease are to be followed. Testing some few muscles and nerves is, fortunately, sufficient for diagnostic purposes.

A complete electrical examination of a nerve muscle should include testing of both muscle and nerve by the galvanic as well as by the faradic current. A nerve generally reacts the same to galvanism as to faradism, so that, if anything has to be slighted, it is advisable to omit the galvanic test. Still, the latter has here, as elsewhere, a distinct advantage over faradism, because with it the amount of stimulation can be measured.

The term faradic stimulation, as employed here and in what follows, implies the use of a rapid induction current (tetanic contraction) from a rapidly vibrating sliding apparatus. Single induction shocks can also be obtained from this instrument by fastening the hammer, and then opening and closing the primary current by hand. The latter method is particularly useful and practical in peripheral palsies with a decided diminution of irritability, because it is much less painful, and because degenerated muscles retain a reaction much longer to individual interruptions than to an interrupted tetanic current. This is a point worth remembering in treating such paralyses.

There are certain places scattered over the surface of the body which, when stimulated by a small electrode, evoke the best response from one definite nerve or muscle. These are the so-called "motor points" determined by Duchenne, Erb, v. Ziemssen, and others, and make possible the individual stimulation of single motor nerves and muscles. They are represented in the accompanying figures (305 to 309). Their location has been confirmed by the writer's own examinations. Most

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FIG. 305.—Motor points on the head and neck:

1. Occipitalis.
2. Retrahens aurem.
3. Posterior auricular nerve.
4. Splenius.
5. Spinal accessory nerve.
6. Sternocleidomastoid.
7. Trapezius.
8. Circumflex nerve (deltoid).
9. Long thoracic nerve (serratus magnus).
10. Brachial plexus.
11. Lower branch.
12. Facial nerve (trunk)
13. Upper branch.
14. Middle branch.
15. Temporalis.
16. Frontalis.
17. Corrugator supercilii.
18. Orbicularis palpebrarum.
19. Nasal muscles.
20. Levator labii superioris.
21. Zygomaticus major.
22. Orbicularis oris.
23. Masseter.
24. Levator labii inferioris.
25. Depressor labii inferioris.
26. Depressor anguli oris.
27. Hypoglossal nerve (in the depths).
27a. Platysma.
28. Sternohyoid.
29. Omohyoid.
30. Phrenic nerve.
31. Sternothyroid.
32. Erb's point[1] (deltoid, biceps, brachialis anticus, supinator longus).
33. Anterior thoracic nerve (pectoralis major).

motor points for nerves are situated where the nerve lies superficially and at some little distance from other nerves. The motor point of a muscle is usually at the point where the motor branch of the nerve

[1] Erb's point is situated 2 to 3 cm. above the clavicle, somewhat to the outer side of the posterior border of the sternocleidomastoid, immediately in front of the transverse process of the sixth cervical vertebra.

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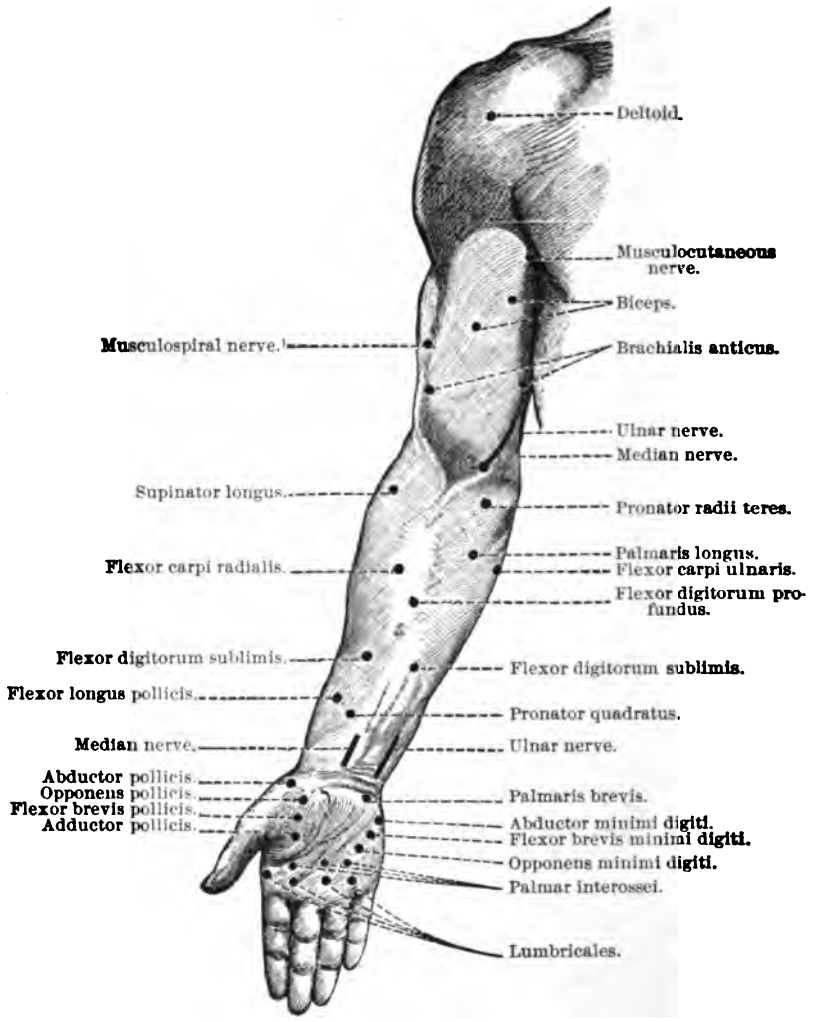

FIG. 306.—Motor points on the anterior aspect of the arm.

enters the muscle belly. In point of fact, it is here that the nerve is generally stimulated. Therefore, when endeavoring to obtain a pure muscular reaction, it is a good plan to keep as far as possible from the

[1] This point should be sought for in the external bicipital groove, at the junction of the middle and lower thirds of the arm. According to Erb, it is midway between the external condyle and the insertion of the deltoid. It is frequently difficult to locate, because the nerve easily rolls from beneath the electrode, and possibly also because the electrode is lifted away from the nerve by the contraction of the biceps and triceps. A very fine electrode should be employed for this examination. It should be noted that the trunk of the musculospiral nerve may also be irritated in the axilla at the inner border of the upper extremity of the coracobrachialis. Erb's point (see Fig. 305) may also be employed for the supinator longus. These points possess a special clinical interest, as will be shown on p. 819.

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motor points, unless the nerve irritability is lost. In this way stimulation will produce local contractions only in the individual bundles which are stimulated.

In all electrical tests it is essential to determine three factors: First,

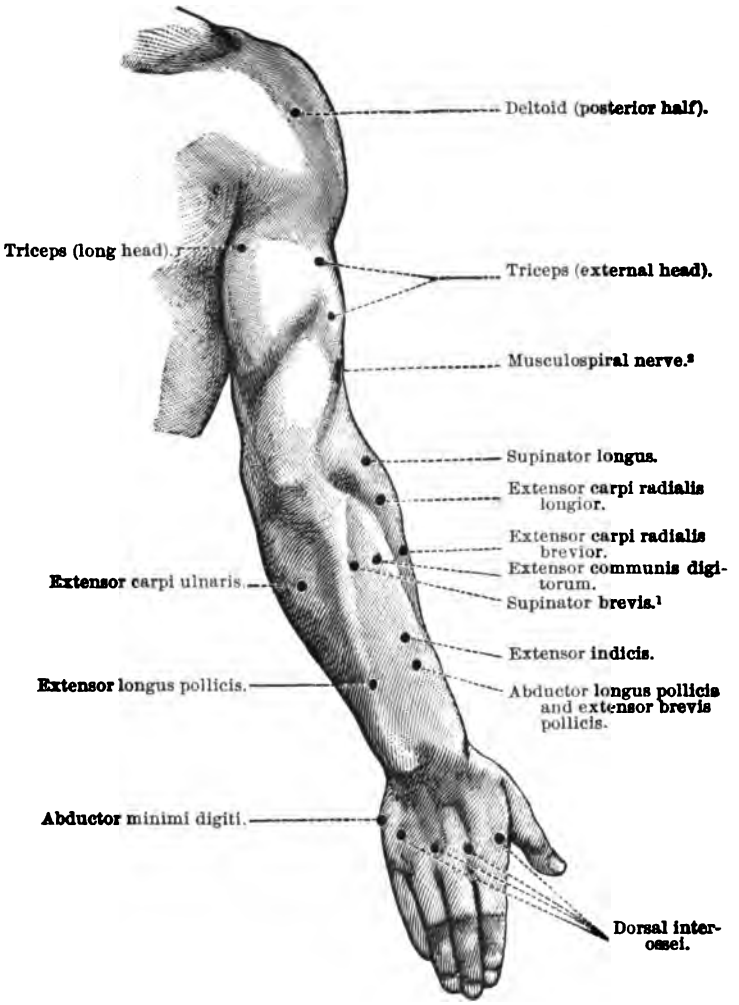

FIG. 307.—Motor points on the posterior aspect of the arm.

whether both the motor nerve and the muscle react to the faradic and to the galvanic current; secondly, whether the reaction to either current is quantitatively altered—*i. e.*, whether it is increased or, as most frequently happens, diminished; and finally, whether any qualitative alteration in

[1] It is possible to irritate the supinator brevis by itself only when the extensor communis digitorum is atrophic and fails to respond to the electric current (as in lead palsy, for example).

[2] See note to preceding figure.

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irritability can be demonstrated—*i. e.*, any deviation from the ordinary type of contraction. With the galvanic current the effect of each pole must be noted.

The "dosage"—*i. e.*, the strength of current employed—must always be estimated in examining for quantitative changes.

This has been done heretofore in using the galvanic current by read-

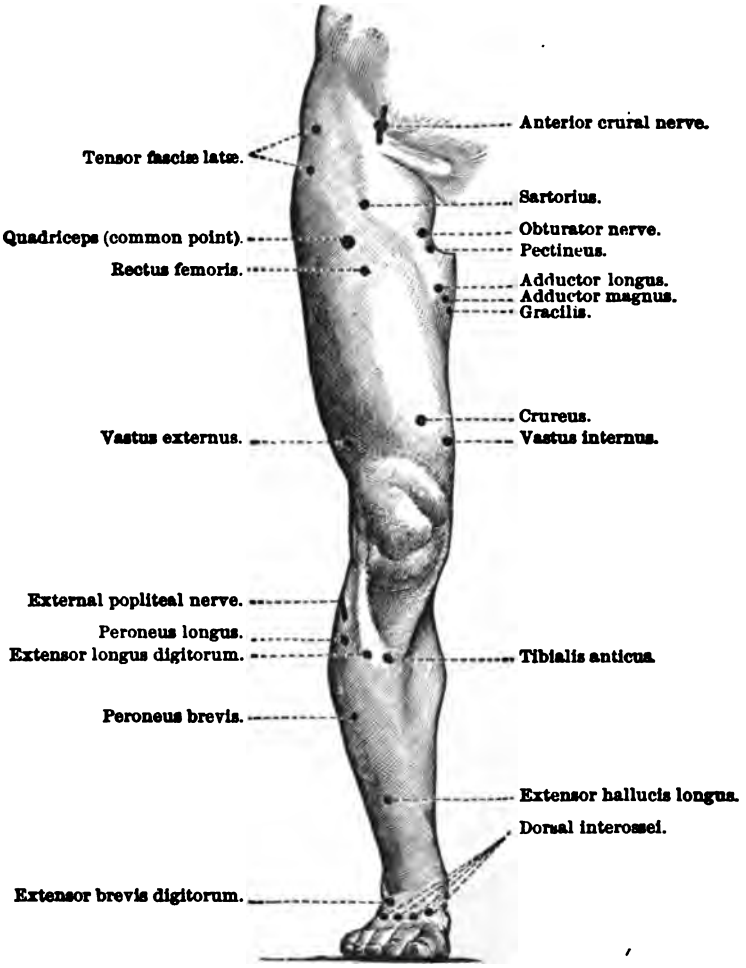

FIG. 308.—Motor points on the anterior aspect of the leg.

ing the strength of the current. The latter changes so very rapidly that in closure contracture it must be read immediately. In examination of the opening contraction, it is read directly before the contraction begins. Before taking the reading, the current should be allowed to flow through the body and the galvanometer until the needle of the latter becomes quiet. In a well-made instrument adapted for medical purposes, with a good rheostat, this will occur in a few seconds.

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Dubois (Bern)[1] has recently shown, however, that in estimating the galvanic irritability of a nerve or muscle it is more accurate to make the calculation in volts than in ampères, for the same movement of the muscle always takes almost the same voltage, while the current rises with the resistance interposed.

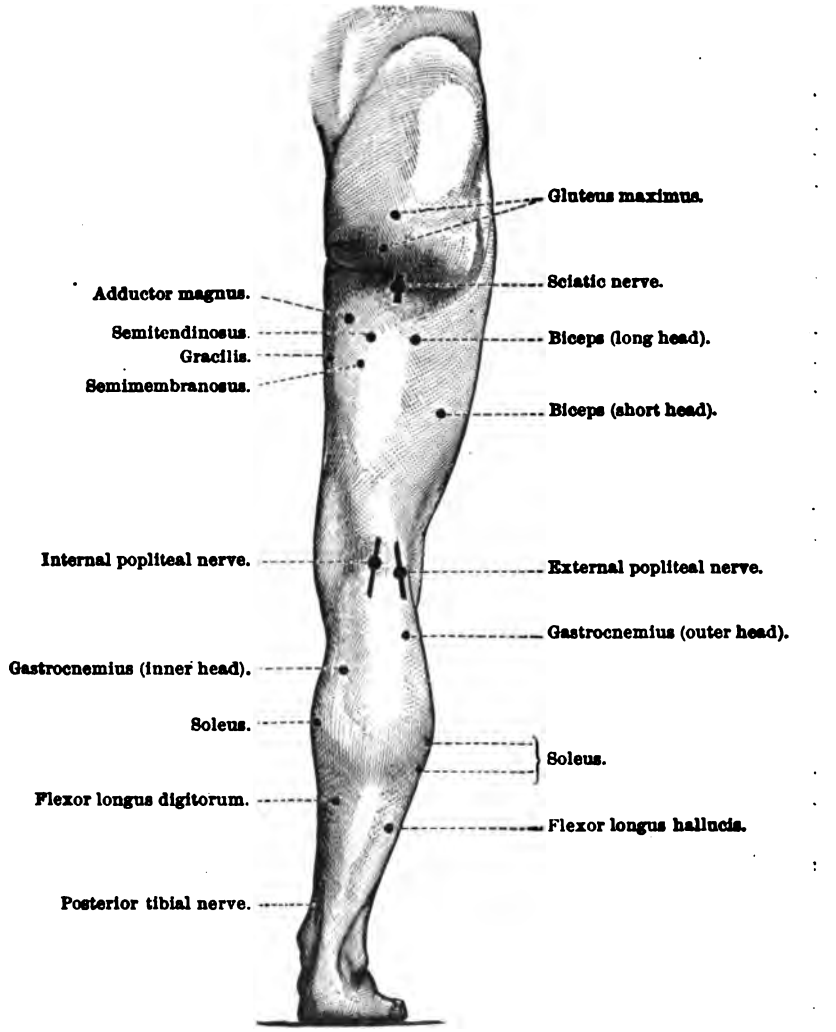

FIG. 309.—Motor points on the posterior aspect of the leg.

This coincides with the experience of Cornaz,[2] pupil of Dubois. He found that measurements made in volts conformed much more nearly than when made in ampères when he tested the same nerve of the same individual at different times, or similar nerves of different people, or similar nerves of the two sides of the body. Dubois explains

[1] *Arch. de physiol.*, Oct., 1897.

[2] *J. A. Diss.*, Bern, 1898.

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this phenomenon by the following fact, which he established experimentally (for the details his monograph must be consulted): The resistance of the human body, in consequence of its great "capacity," has practically no influence on the amount of stimulation, which depends upon potential. So that if the metallic resistance of the current circuit does not change, and if the current strength varies only with alterations in the electromotive force, the stimulation can be determined with considerable accuracy by the volt tension. To be sure, this is no longer the case if rheostat resistance is interpolated into the main circuit and the action of the current is thus graded, as was formerly the custom in electrodiagnosis. Dubois has shown that such resistance, whether fluid or metallic, produces strong auto-induction. Therefore it must cause a change not only in the current strength, but also in the increase of the current. Under these conditions neither the volt tension nor the current strength can be considered a measure of the irritative effect, so that this old procedure loses its value. Examining the same muscle repeatedly with the same strength of current but with varying rheostat resistance would therefore explain many previously inexplicable contradictions in electrodiagnosis.

Hence, in employing the volt meter it is advisable to regulate the galvanic apparatus so that the resistance in the main current will not vary with a gradation of current activity. In other words, in addition to the employment of the element transformer, the current activity should be graded only by altering the volt tension. The latter effect can be accomplished by inserting a rheostat into the lateral circuit, or, still better, by Gaiffe's "reducteur de potential," constructed according to the principle of the rheochord.

Instead of the galvanometer, Dubois recommends substituting the volt meter, combined with the "reducteur de potential," for electrodiagnosis (though not for electrotherapy). The volt meter is practically a galvanometer situated in the lateral circuit, but of so great a resistance that the resistance of the battery does not enter into consideration. Gaiffe's instrument is especially serviceable for electrodiagnostic purposes, because a simple alteration will in a few seconds change it to a galvanometer with *accurate divisions*. The volt meter possesses another great advantage. Its needle does not oscillate with the opening and closing of the current, so that the reading may be made during the examination. This is much more trustworthy than the estimation of the current strength, which can be read only after the stimulation is finished or with opening stimulation before it begins.

This combination of volt meter with "reducteur de potential" has been used at the author's clinic, and has been entirely satisfactory.

It should be noted that the accuracy of the measurement of galvanic reaction by means of the volt meter has recently been questioned by Mann,[1] who recommends, as the method most free from objection, the employment of condenser discharges. Since this method has not yet been extensively employed, the author will simply refer the reader to the article by the above-mentioned author.

[1] *Berlin. klin. Woch.*, 1904, No. 33.

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The induction current, as Dubois has shown, has faults, even with the modern simplified gradations of the apparatus. Unfortunately, it must be confessed that thus far no method has been devised to estimate the physiologic action of the induction current in a universally efficient way—*i. e.*, in figures independent of the apparatus employed. The physiologic action of one induction apparatus cannot be compared with the action of another, so that for quantitative examination or for comparison of results we should always employ the same apparatus. The distance that the secondary coil is moved will measure the current strength; provided, of course, that the elements or cells are freshly prepared. Since the individual centimeters of the scale usually placed upon the induction apparatus correspond to entirely different variations in the effect of the current, it would be much better to graduate the scale also in absolute units, according to the method of Kronecker.[1] It must not be supposed, however, that such a graduation will enable us to compare different induction apparatuses, since the rapidity of increase in the individual induction currents, and their consequent irritating effects, are quite different, being dependent upon the quality of the coils as well as upon the electromotive force of the elements which run the apparatus. The absolute graduation simply possesses the advantage that, if we always use the same machine with freshly prepared elements, we obtain a better idea of the effect of the current.

Even with a faradic current the bodily resistance varies physiologically, because the stimulation takes place under variable conditions in a circuit of great capacity; but such variation does not produce any decided difference in the effect of the stimulation any more than with galvanism (see above). Therefore it seems unnecessary to follow many authors' advice and measure the skin resistance galvanically, as well as the distance of the secondary coil, when applying the faradic current. But in any event the electrodes must be well moistened, in order that the contact may be as complete as possible.

It is quite as important, in determining the dosage of the faradic as in determining that of the galvanic currents, to employ electrodes of definite size; because, naturally, the intensity with which the current impinges upon a given spot is quite as important as the current strength and the volt tension. The current intensity is equal to the current strength divided by the square surface of the electrode. Therefore, under otherwise equal conditions, an electrode of 2 sq. cm. surface provides only half as great a stimulation effect as one of 1 sq. cm. surface. Such an estimation is accurate only for the skin surface directly in contact with the electrode. In penetrating the skin the current is, of course, diffused over a much greater area, and since most of the muscles and nerves to be stimulated lie at a certain depth, the size of the electrode is in reality not so important as this discussion would lead one to think. At all events, in order that the conditions may be kept as constant as possible, electrodes of the same size should be employed in comparative examination, and

[1] *Zeits. f. Instrumentenkunde*, 1889.

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their dimensions should be mentioned in the description. The so-called "normal" electrodes (p. 799) are the most satisfactory.

The technic of applying the electrode is naturally important, for a larger electrode set against the body surface at an angle naturally acts like a smaller one. Again, one-sided pressure should be avoided, the pressure should not crowd the soft parts too much, and the well-moistened surface of the electrode should be in intimate contact with the body.

If, as is sometimes the case, especially in employing a portable galvanic battery, we wish to differentiate the poles quickly, it can be done very easily by grasping the electrodes one in each hand, and then estimating which electrode causes more intense burning on closing the current. The one that does will be the cathode. Of course, the electrodes should be of the same size and equally well moistened. Again, the cathode turns moistened violet litmus paper blue; the anode turns it red. With the faradic current a stronger irritation is produced by a cathodal opening, and after the passage of some current a blue spot will appear upon the litmus paper. The opening of the induced current, on account of its greater strength, is of chief importance for the collective action of the alternating stream.

The electrode to be employed in such tests should be of polished metal; for this will give sufficient current strength for the slight electrolytic action of the induction current; and we need not fear any discoloration of the litmus paper by the acid metallic salts which always collect upon old electrodes.

The following abbreviations are employed to tabulate the results of an electrical examination:

X mm. *D* = distance in millimeters that the secondary has been withdrawn from the primary coil.
M. A. = milliampères.
V. = volt.
Ca. C. C. = Cathodal closing contraction.
An. C. C. = Anodal closing contraction.
An. O. C. = Anodal opening contraction.
Ca. O. C. = Cathodal opening contraction.
Ca. C. T. = Cathodal closure tetanus.
Ca. C. C. = 2 *M. A.* signifies that a minimal cathodal closure contraction will be produced by 2 milliampères of current.
Far. C. 90 mm. *D.* signifies that a minimal faradic contraction will be excited when the secondary coil is withdrawn 90 millimeters.
Ca. C. C. > *An. C. C.* signifies that the cathodal closure contraction is greater than the anodal closure contraction, etc.

2. METHOD OF TESTING THE QUANTITATIVE ELECTRICAL IRRITABILITY OF THE NERVE MUSCLE.

We test the quantitative electrical irritability of a nerve or of a muscle by estimating how strong a galvanic, and then how strong a faradic current is required to produce a minimal contraction (see p. 803 *et seq.*). With a galvanic current the cathodal closure contraction is normally the easier to obtain; hence, it is ordinarily employed for

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this estimation. When a maximum current produces no further contraction, the condition is called exhausted irritability. Since this condition holds good only for that particular maximum current strength or volt tension employed, it is advisable to determine both of these.

Physiologically, the irritability of different nerves and muscles, as well as that of the same muscle or nerve, varies considerably in different individuals. It is not by any means easy, therefore, in any given case to determine whether the results of the examination overstep the normal limits, and, if so, how much. The following method seems the most practical: We estimate the irritability and then compare it with that of a healthy person, or, if the disturbance is unilateral, with that of the healthy side. The latter is, of course, always more accurate. Symmetric points and an identical position must, of course, be selected. Erb and Stintzing have shown that the normal differences in irritability upon the two sides are very slight. In comparing the results with those found in a healthy person, decided differences only should be regarded as significant.

Galvanic Current.

Normal Estimations to be Used in Comparing the Galvanic Irritability of the Two Sides.—The following figures represent the maximal differences of the galvanic current strength which is needed to stimulate actively the normal nerve of both sides of the body. They are copied from Stintzing, and were estimated with his normal electrode of 3 sq. cm.:

Maximal Physiologic Differences between the Galvanic Irritability of the Two Halves of the Body Expressed in Current Strength (after Stintzing).

Ramus frontalis of the facial nerve	0.7 M. A.
Nervus accessorius	0.15 "
Nervus medianus	0.6 "
Nervus ulnaris 2″ (above the olecranon)	0.6 "
Musculospiral nerve	1.1 "
Nervus peroneus	0.5 "
Nervus tibialis	1.1 "

Rates of the Volt Tensions which are Required for a Minimal Stimulation of the Corresponding Normal Nerves of the Two Sides of Body (after Dubois and Cornaz).

These figures are important:

Faciales (maxim.)	100:122	(corresponding	ratio of current strength	=100:129)
Medianus "	100:117	("	"	=100:503)
Musculospiral "	100:112	("	"	=100:145)
Ulnaris "	100:116	("	"	=100:253)
Peroneus "	100:130	("	"	=100:175)

Normal Estimations to be Used in Comparing the Galvanic Excitability of Different Individuals.—The following figures represent the averages which Stintzing computed from an examination of 58 healthy individuals. They are useful when we cannot compare the two sides of the body.

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Limits of the Normal Irritability Expressed in Current Strength (after Stintzing).

Ramus frontalis of the facial nerve,	irritated by	0.9 — 2.0 M. A.
Ramus zygomaticus of the facial nerve,	"	0.8 — 2.0 "
Ramus mentalis,	"	0.5 — 1.4 "
Nervus accessorius,	"	0.1 — 0.4 "
Nervus ulnaris 2″ above the olecranon,	"	0.2 — 0.9 "
Musculospiral nerve,	"	0.9 — 2.7 "
Nervus peroneus,	"	0.2 — 2.0 "
Nervus tibialis,	"	0.4 — 2.5 "

Limits of the Physiologic Irritability of Corresponding Nerves of Different Individuals and of the Same Individual at Different Times, Expressed in Volt Tension and in Current Strength (after Dubois and Cornaz):

MAXIMAL DIFFERENCES.

	VOLTS.	Ratio.	MILLIAMPÈRES.	Ratio.
Facialis.				
1. In different individuals,	3.8 — 9.4	(100 : 247)	0.8 — 3.0	(100 : 375).
2. In the same individual at different times,		100 : 126		100 : 190
Medianus.				
1. In different individuals,	4.4 — 14.2	(100 : 323)	0.2 — 2.7	(100 : 1350).
2. In the same individual at different times,		100 : 212		100 : 1000
Musculospiral Nerve.				
1. In different individuals,	5.2 — 12.8	(100 : 246)	0.8 — 2.5	(100 : 246).
2. In the same individual at different times,		100 : 152		100 : 225
Ulnaris.				
1. In different individuals,	1.6 — 7.8	(100 : 487)	0.1 — 1.9	(100 : 1900).
2. In the same individual at different times,		100 : 260		100 : 575
Peroneus.				
1. In different individuals,	4.0 — 10.5	(100 : 265)	0.6 — 1.8	(100 : 300).
2. In the same individual at different times,		100 : 225		100 : 225

When a weaker current strength or volt tension excites a minimal contraction, the stimulability is increased; when a stronger one is required, the stimulability is diminished.

As mentioned above, the measurement by volt tension is the more accurate method—*e. g.*, a minimal volt tension which will produce the necessary stimulation will correspond to a much greater current strength if the resistance is slight than if the resistance is high (according to p. 805 *et seq.*). The milliampère value will, in the former case, conceal a diminished, and, in the latter case, an increased, stimulability.

Faradic Current.

Every examiner will gradually become accustomed to his own apparatus, so that he knows what differences come within physiologic limits. Stintzing found that with his own apparatus the maximal physiologic difference for all nerves examined was 15 mm. D. This is a rather indefinite statement, since this 15 mm., even with Stintzing's apparatus, corresponds to entirely different amounts of irritation, dependent upon the position of the secondary coil, and these values, moreover, do not obtain when another apparatus is employed. Differences which exceed

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a physiologic maximum should be regarded as pathologic. If a shorter distance is necessary to excite a minimal contraction, it shows a diminution of irritability; if a greater distance is required, an increase of irritability.

3. METHOD OF TESTING THE QUALITATIVE ELECTRICAL STIMULABILITY OF THE NERVE MUSCLE.

The irritability of motor nerves is subject only to quantitative alteration. But with the muscles, quantitative changes are often accompanied by qualitative alterations, which require a special description.

(*a*) Normal Conditions.

The qualitative normal reaction of motor nerves and muscles to a galvanic stimulation is expressed in the following so-called law of contraction:

The Normal Law of Contraction for the Motor Nerves with a Galvanic Current.

WEAK CURRENT.

Ca. C. C. . . . . . *An. C.*: no effect.
Ca. O.: no effect . . . . . *An. O.*: no effect.

MODERATE CURRENT.

Ca. C. C.: vigorous . . . . . *An. C. C.*: weak.
Ca. O.: no effect. . . . . *An. O. C.*: weak.

VERY STRONG CURRENT.

Ca. C. T. . . . . . *An. C. C.*: vigorous.
Ca. O. C.: variable . . . . . *An. O. C.*: vigorous.

The normal law of contraction for the muscles with a galvanic current differs from the above only by the fact that the opening contraction is either obtained with great difficulty or, more commonly, cannot be produced. The contraction of a muscle to galvanism, whether the muscle is stimulated directly or indirectly through the nerve, normally appears very suddenly, like lightning. Yet some differences occur, depending upon whether the differential electrode is applied to the motor point of the muscle or at some distance from it. The latter (avoiding the motor point as far as possible) is the only way in which we can excite a pure muscular contraction. Although sudden, the contractions are somewhat less lightning-like, and the distinction between *Ca. C. C.* and *An. C. C.* is less pronounced. A stimulation from the motor point itself should be regarded really as the stimulation of a nerve.

Normal Laws of Contraction for Motor Nerves and Muscles with the Ordinary Faradic Current (the rapidly interrupted and alternating induced current).—Faradic stimulation of both muscle and nerve is tetanic—*i. e.*, soon as *D.* (the distance) is diminished enough to produce any contraction at all, the muscle remains contracted so long as the alternating current flows through it or through its motor nerve. The onset and the cessation of the tetanic muscular contraction correspond to the closure and the opening of the current, and are sudden and

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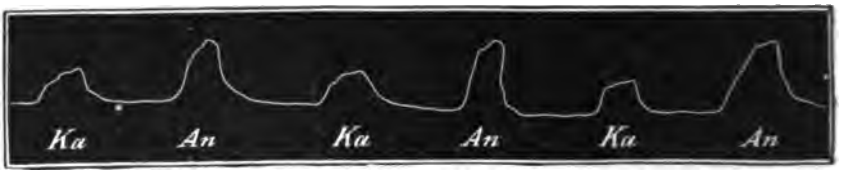
lightning-like. The two poles of the faradic current differ only in their quantitative effect—*i. e.*, the pole at which the opening induction current has its cathode is somewhat stronger. The faradic current produces a much weaker contraction when applied near a motor point than when applied directly to it. Qualitatively, on the contrary, the effect is identical.

(*b*) Pathologic Conditions.

Reaction of Degeneration (D. R.)—The essential factor in the reaction of degeneration which occurs in peripheral and muscular paralyses is the condition of the muscles (see p. 817 *et seq.* in regard to its significance). This reaction, in the broadest sense of the term, presents very different modifications. The two cardinal symptoms common to all forms are the following:

1. The essentially different reaction of the muscle to prolonged and to transitory stimulation—that is, to galvanic and faradic. The muscle

(*a*) Healthy girl. *Ca. C. C.* decidedly greater than *An. C. C.* Contraction lightning-like.



(*b*) Poliomyelitis reaction of degeneration. *An. C. C.* much greater than *Ca. C. C.* Contraction slow.

FIG. 310.—Myographic curves of galvanic closure contraction from a direct muscular stimulation in the peroneal region: (*a*), Normal; (*b*), reaction of degeneration (taken from Kast).

either does not react at all to the faradic current, or its reaction is very much weaker than to the galvanic current. Dubois's experiments, however, show that single shocks of a very strong current will produce a contraction even where the reaction of degeneration is complete. He believes that rapidly succeeding shocks—*i. e.*, with a freely vibrating hammer—so fatigue the muscle that it cannot contract.

2. The contraction no longer exhibits a lightning-like character, but is slow and vermiform. An after-contraction may sometimes be noticed to persist after the original effect has ceased; and in a pronounced example a tetanic contraction will be excited which will persist most of the time while the current is applied.

The other characteristics of the reaction are very variable; they will be mentioned below.

The two peculiarities just outlined are common to all forms of reac-

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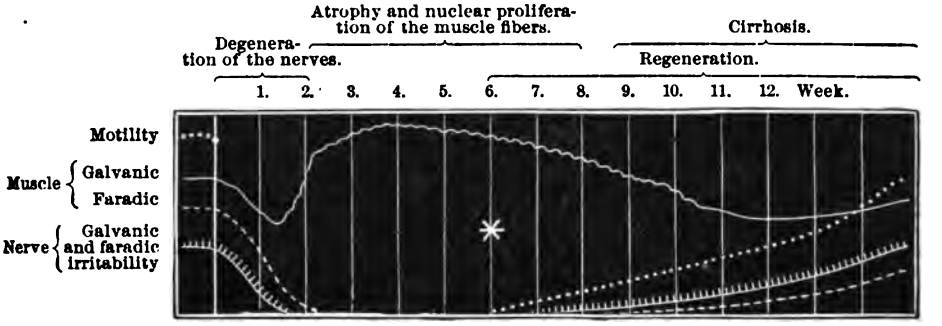
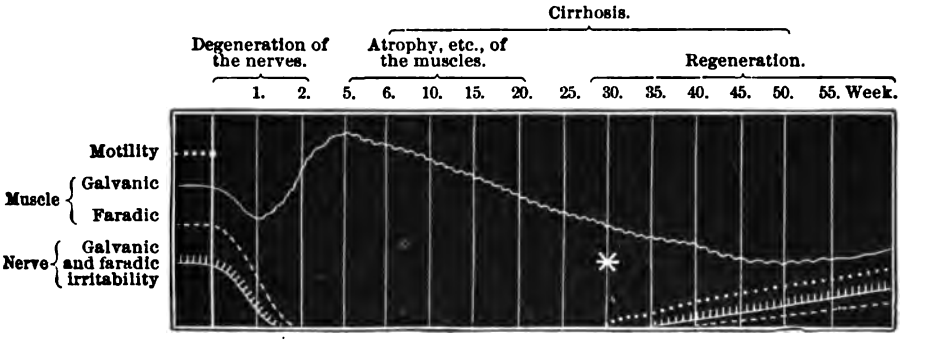
tion of degeneration, and are to be regarded as the clinical expression of a degenerative atrophy of the muscle due to some peripheral lesion (see p. 789 *et seq.*).

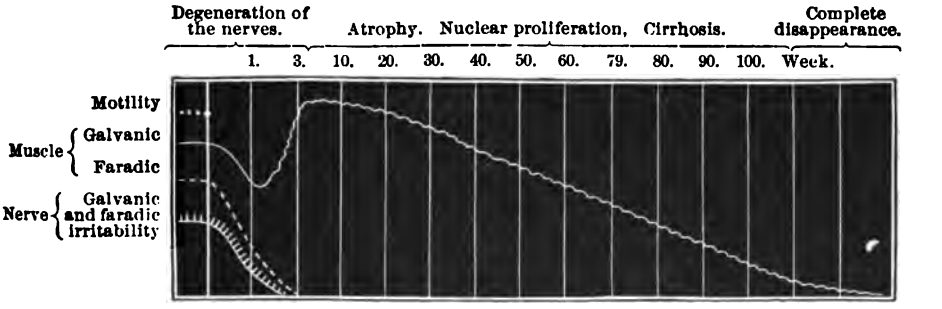
The complete reaction of degeneration may be schematically described as follows:



(a) Paralysis with a relatively early recovery of the motility.



(b) Paralysis with a later recovery of the motility.



(c) Irreparable paralysis. Disappearance of motility persists.

FIG. 311.—Diagram of the course of irritability in peripheral paralysis with complete reaction of degeneration: The wavy character of the line which represents the galvanic irritability signifies that the irritability is qualitatively modified—*i. e.*, there is a D. R. Where the line is even the condition is qualitatively normal. The asterisk marks the return of voluntary motility. The histologic changes in the nerve and muscle are printed at each stage above the curves. The figures above the curves correspond to the number of weeks which have elapsed since the onset of the paralysis. The abscissæ of the later course of the paralysis had to be shortened in different degrees for the three curves on account of the space, so that the curves cannot be exactly compared as to their length (Erb).

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Faradic test: Excitability of the nerves lost.

Faradic test: Excitability of the muscles lost.

Galvanic test: Excitability of the nerves lost.

Galvanic test: Excitability of the muscles immediately after the onset of the paralysis quantitatively normal, later increased, and finally often decidedly diminished. The contraction is slow. *An. C. C.* is excited more rapidly than *Ca. C. C.* *An. C. C.* > *Ca. C. C.* for the same current strength or volt tension.

The myographic curves (Fig. 310) illustrate these quantitative changes of the muscle reaction.

The duration of complete reaction of degeneration in peripheral paralysis is very typical. It has been studied thoroughly in severe facial paralysis of rheumatic origin, and has been graphically represented by Erb (Fig. 311).

Mechanical reaction of degeneration, mentioned upon p. 798, occurs principally where there is complete electrical reaction of degeneration with increased irritability.

Partial (Incomplete) Reaction of Degeneration.—In this form the faradic and galvanic irritability of the nerves and the faradic irritability of the muscles are not lost, but merely diminished. A slow contraction is ordinarily caused only by galvanic excitation of the muscles, although sometimes faradic excitation with isolated shocks at some distance from the motor point will produce the same effect. A scheme of the incomplete reaction of degeneration follows:

Faradic: Nerve. Muscle.	Irritability merely diminished. Contractions not slower, except sometimes in the case of the faradic muscle contractions when the motor point is avoided.
Galvanic: Nerve. Muscle.	Like the complete reaction of degeneration.

The course of an incomplete reaction of degeneration in peripheral paralyses is represented in Fig. 312.

Incomplete Reaction of Degeneration with Forced Indirect Slower Contractions.—In this form, as distinguished from the simple incomplete reaction of degeneration, all the contractions are slowed, not only those excited by galvanic muscular stimulation, but also those excited by faradic stimulation of the muscles and by faradic or galvanic stimulation of the nerves.

Mixed Reaction of Degeneration.—When some of the fibers of a muscle preserve a normal reaction, while others present a reaction of degeneration, the result is called a *mixed reaction of degeneration.* In such cases we cannot examine each type of fiber separately, so that the resulting mixed reaction is frequently difficult to understand, and it is quite impossible to pick out which of the fibers show a normal and which a degenerate reaction. Many instances of incomplete reaction of degeneration really belong to this variety.

Peculiar Electric Reactions of Certain Old Peripheral Palsies as Described by Placzek.—Placzek,[1] and subsequently Bernhardt,[2] have described a peculiar phenomenon which is in rare cases observed as a termination of severe peripheral facial palsies. In contrast to the usual findings, the electrical reactions of the nerve muscle in some of these old facial palsies reappear, although the paralysis of voluntary motion remains permanent. This phenomenon is highly interesting from a theoretic standpoint, and the author would regard it as analogous to

[1] *Berlin. klin. Woch.*, 1893. [2] *Ibid.*, 1903.

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the behavior of the so-called mild peripheral palsies, in which the paralysis of voluntary motion exists together with maintained electrical excitability of the peripheral parts. Both conditions may be explained by the supposition that changes exist in the peripheral nerves which block voluntary impulses, but do not disturb the peripheral excitability of the nerve or muscle. In the old palsies exhibiting this phenomenon there can be no doubt that peripheral regeneration has taken place, but that the blockading of voluntary impulses nevertheless permanently remains. This is difficult to understand if we cling to the old theory of the necessity of efferent irritations for the trophic integrity of the nerve muscle. This old theory, however, does not seem to obtain in all cases, since Bethe has shown that, when the union of divided nerve fibers is prevented in young animals,

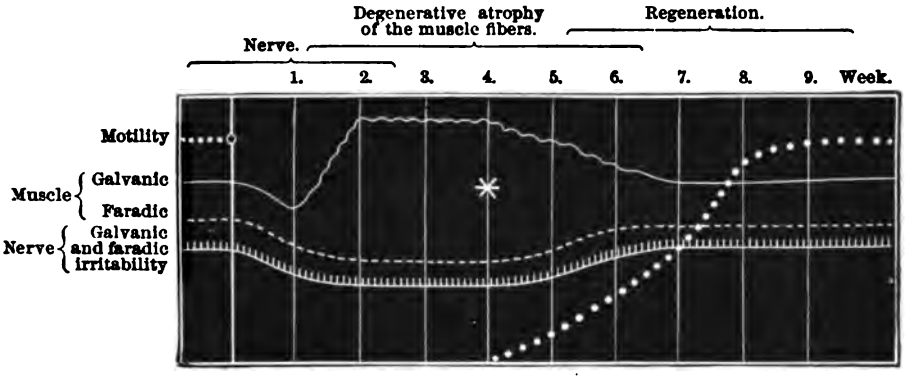

FIG. 312.—Diagram of the course of irritability in partial reaction of degeneration. See the explanation of Fig. 310. The faradic and galvanic irritability of the nerves and the faradic irritability of the muscles are only slightly diminished. Motility returns early. Compensation early and complete. Nerve degeneration probably absent.

a peripheral regeneration may nevertheless occur, so that the nerve fibers regain their electrical irritability.

Myotonic Reaction (Erb).—This occurs only in Thomsen's disease (myotonia congenita). It is characterized by a peculiar persistence of the contraction after the cessation of the stimulation. The contractions are slow and frequent, and, when the galvanic current is steadily applied to the muscles, are often characteristically rhythmic and wave-like. Even very weak currents will excite these phenomena (muscle hyperirritability). The anodal closing contraction (An. C. C.) of the muscle is frequently more vigorous than the cathodal closure (Ca. C. C.).

Neurotonic Reaction.—A few years ago Marina[1] and Remak[2] independently described a rare form of reaction which they called *neurotonic reaction*. Marina found it in *hysteria*, and Remak in a case, in all probability, of *progressive muscular atrophy*.

In this reaction without any increase of the quantitative minimum irritability, anodal opening contraction appears very early from stimulation of the nerve but not of the muscle. There is also a special tendency of the nerve to cathodal closure and anodal opening tetanus. The closure tetanus can be prolonged beyond the opening of the current, and even the faradic tetanus of the nerve may persist after stopping the stimulation. These characteristic phenomena depend essentially upon nerve irritation, and they cannot be excited by stimulation of the muscle.

Reaction in Tetany.—A quantitative increase of the electrical irritability of the nerve trunks is found in this disease, although it does not affect the mus-

[1] See *Neurol. Centralbl.*, 1896, No. 17. The older Italian publications upon this subject by Marina are mentioned here.

[2] *Neurol. Centralbl.*, 1896, No. 13.

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cle, or, at least, not nearly to so great an extent. The quality of the reaction of the nerve is frequently, though not constantly, altered—*e. g.*, a neurotonic reaction appears with a tendency to anodal opening and cathodal closure contraction and a prolongation of the contraction. The essential difference is that in tetany, as contrasted with neurotonic reaction proper, a quantitative increase of irritability accompanies the qualitative changes.

Characteristic Reaction in Certain Traumatic Neuroses.—Rumpf[1] has applied the unfortunate term "traumatic reaction" to this peculiar phenomenon. For some time after the cessation of a vigorous faradic stimulation the muscle will exhibit a characteristic fluctuating movement, made up of alternating fibrillary and clonic contractions. In many cases these complex results occur even during the stimulation. Each contraction may spread from the muscle which is directly stimulated and become generalized. In these patients similar appearances are observed to follow vigorous efforts and the action of cold. (See p. 748, Fibrillary Contractions in the Healthy.)

Myasthenic Reaction.—A peculiar electrical condition of the affected parts accompanies *myasthenia gravis pseudoparalytica* or *asthenic bulbar paralysis*, as the disease is usually called. It was first described by Jolly.[2] When a tetanic induction current is permitted to act upon a muscle, whether directly or through the nerve, the contraction gradually diminishes and finally disappears. This electrical phenomenon is entirely analogous to the pathologic fatigue of the muscle after voluntary impulses, which is so characteristic of this disease. A similar phenomenon can be elicited experimentally in muscles poisoned with protoveratrin.

4. DIAGNOSTIC SIGNIFICANCE OF THE DIFFERENT ELECTRICAL REACTIONS.

The myotonic, neurotonic, "traumatic," and tetany reactions have been sufficiently described above, and, so far as our present knowledge goes, each is limited to its corresponding disease.

In *psychic* or *hysteric paralysis* the electrical irritability remains normal. This is also a general rule for all paralyses which depend upon a lesion of the voluntary tracts above the nucleus—*i. e.*, above the gray anterior horn (*cerebral hemiplegias, transverse lesions of the spinal cord*). Sometimes, however, in these cases, especially if the paralysis has persisted for some time, the electrical irritability is considerably diminished.

The condition during the first few days following a *peripheral paralysis* (see Figs. 311 and 312), and that during the entire course of the *milder forms*, will illustrate very typically a simple diminution in the electrical irritability. When the peripheral interruption of conduction is complete—*i. e.*, when the paralysis is severe—reaction of degeneration will generally take the place of simple diminution of irritability.

Some cases of *polyneuritis* and of *lead-poisoning* furnish striking exceptions to the above statements. In some cases, instead of the expected reaction of degeneration, not only is the irritability diminished, but the diminution is so pronounced that even direct contract of the galvanic current excites no response. This proves that in these cases the muscles are affected by the toxins directly, and not alone through

[1] *Deutsch. med. Woch.*, 1890, No. 9, p. 165.
[2] *Berlin. klin. Woch.*, 1895, vol. i., p. 2, *et seq.*
[3] Prognosis will be discussed separately upon p. 819 *et seq.*

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involvement of the nerve. In fact, there are many other reasons for such an hypothesis. This supposition is at least probable in those cases which recover, despite a marked diminution of the direct galvanic muscular irritability. At all events, in incurable cases of polyneuritis and lead-poisoning, the disappearance of the galvanic irritability, and the rapidly appearing terminal stage of the reaction of degeneration, must be considered to be the expression of definite secondary degeneration of the muscle (see Fig. 311).

In the *myopathic forms of progressive muscular atrophy* simple diminution of electrical irritability is the rule.

A simple increase of electrical irritability is a comparatively rare phenomenon. It does occur in quite recent *paralyses of neural origin*, and in *tetany*. It then depends chiefly upon nerve excitability. Increased excitability of the nerve or muscle, with coincident qualitative change, should not be confused with such a condition. (Reaction of Degeneration, Myotonic Reaction, Tetanic Reaction, see above.)

The various alterations in reaction of degeneration (in the widest sense of the term) occur only in cases in which the muscle is affected by a break in conduction between it and its so-called trophic center[1] (situated in the nucleus—*i. e.*, in the anterior horns), or where a lesion of the center itself occurs, or where the muscle is primarily degenerated. The degeneration of the muscle, and with it the reaction of degeneration, may be absent, even when the lesion is so situated, provided it be slight and transitory. Degenerative changes in the muscle are found in all instances of reaction of degeneration and, in fact, seem to be physiologically expressive of such a reaction. If complete, the degeneration also spreads to the nerve. A partial reaction of degeneration, on the contrary, which depends upon a moderately severe disturbance of conduction, accompanies a degeneration of the muscle with normal or, at most, only slightly degenerated nerves. The retention of nerve irritability in partial reactions of degeneration seems to depend upon the preservation of the medullary sheaths.

In accordance with the above, the most frequent lesions which produce the various types of the reaction of degeneration are *nuclear* or *peripheral paralyses*. The *spinal* and the *neuritic muscular atrophies* also often lead to partial reactions of degeneration. Yet numerous divergences in the electrical results occur in individual cases of these muscular atrophies, because in them, as contrasted with actual paralyses, each fiber is successively and, in a certain measure, individually diseased. Therefore each muscle we examine contains a series of fibers in different conditions of irritability, and the resulting reaction will depend upon whether seriously injured, slightly affected, or intact fibers predominate. In *spinal* and *neuritic muscular atrophies a mixed reaction* is the rule, whereas a *partial or complete reaction of degeneration* is much commoner in *peripheral palsies*.

Many cases, however, of spinal or neuritic muscular atrophies show

[1] See p. 790, note, for the significance of this center—*i. e.*, how its trophic character is regarded.

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absolutely no signs of reaction of degeneration as—*e. g.*, if the fibers which are seriously affected are very rapidly and quite completely destroyed, so that the reaction to stimulation actually comes from intact fibers or from those only slightly affected, and is merely quantitatively diminished. We might call this result, which is by no means uncommon in spinal neuritic muscular atrophies, a sort of latent reaction of degeneration. The same peculiarity is presented in *bulbar paralysis* which in its origin is identical with the *spinal form of muscular atrophy*, and with the closely related *amyotrophic lateral sclerosis*. Reaction of degeneration may or may not be present. The *myopathic forms of muscular atrophy* exhibit similar variations. Here and there a suspicion of reaction of degeneration does occur, although from the nature of the cause we should suppose that it was impossible. This apparent inconsistency may depend upon the presence of many seriously affected fibers which still react to stimulation at the time of the examination. If many such fibers are present, reaction of degeneration will result; if few, the reaction will depend upon the normal fibers, and simply be normal or but slightly diminished. The occurrence of such variations—*i. e.*, reaction of degeneration in purely myopathic forms, and, conversely, its absence in spinal and neuritic forms of muscular atrophies—makes it plain that we must not rely upon the electrical examination to decide the form of the muscular atrophy, but turn to other clinical evidence, such as heredity, age, mode of extension of the disease, etc.

From what has been said, it is plain that it is much more important for diagnosis to demonstrate that the reaction of degeneration is present than that it is absent. A *positive reaction of degeneration* (this is perhaps the most important law in electrodiagnosis) *completely excludes a central or supranuclear origin* for the disease). The absence of reaction of degeneration, however, even if we except the cases where the lesion is too slight to produce it, does not always permit the exclusion of a nuclear or subnuclear cause of disease.

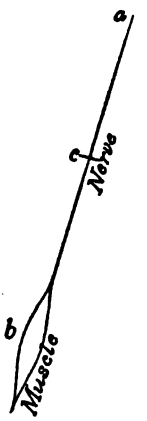

FIG. 313.

There is another difficulty which makes it sometimes impossible to recognize reaction of degeneration, even when the muscles are paralyzed from peripheral lesion. This is the exceptionally diminished muscular excitability, even to the galvanic current, in the late stages of the reaction of degeneration. Frequently the batteries are either too weak to excite contractions, or so strong a current is required that either the pain or the electrolytic action of the current upon the skin prevents the examination. Such an exceptionally diminished irritability should be considered for clinical purposes as equivalent practically to the reaction of degeneration. (See p. 816, Direct Injury to the Muscles by the Toxins.)

We can sometimes utilize an electrical examination to locate some

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obstruction to conduction in a peripheral nerve with its muscle (Fig. 313). If some obstruction to the conduction—*i. e.*, some injury—occurs at *c*, no contraction will result from a stimulation between *a* and *c*, but will from one between *c* and *b*. In this way the lesion can be localized at *c*, provided the nerve is accessible to examination throughout its course. The test will fail, of course, if *c b* is no longer capable of being stimulated—*e. g.*, in a more degenerative lesion where the nerve below *c* soon loses it irritability. In a comparatively fresh or mild paralysis there will be at least a partial reaction of degeneration. This condition is well illustrated by the so-called "drunkard's paralysis" or "sleep paralysis" of the musculospinal nerve, in which we can sometimes locate the injury at the point of origin of the radial nerve, because ordinarily the radial nerve can be stimulated directly above this point—*i. e.*, in the axilla, or, for the supinator longus, at Erb's point (see note to Fig. 306).

5. PROGNOSTIC SIGNIFICANCE OF THE ELECTRICAL REACTION.

Nothing could be more misleading than to base the prognosis upon the alteration in the electrical reaction without at the same time heeding other facts, especially the spinal anatomic diagnosis—*e. g.*, a rheumatic facial paralysis even with a typical reaction of degeneration frequently recovers; whereas a cerebral hemiplegia with perfectly normal electrical reaction is often incurable. Again, a myopathic muscular atrophy with normal electrical reaction is no less incurable than a spinal muscular atrophy with reaction of degeneration. Many similar instances could be mentioned.

At the same time electrodiagnosis is often of incalculable value in determining the prognosis, provided that we compare only the electrical examinations of cases limited to the same type, and draw our conclusions only after several careful examinations repeated at some little interval of time from each other. A few examples will make this evident.

Rheumatic facial paralysis, which Erb has studied so carefully in regard to the sequence of its electrical conditions, supplies us with an excellent example of the prognostic value of the electrical examination. If in this disease reaction of degeneration can still be demonstrated after about fourteen days, it is evident from the diagram upon p. 813 that at least some months, or under some conditions even as much as a year, will elapse before perfect recovery will ensue, or the case may be incurable. If after the lapse of fourteen days no more pronounced alterations of the electrical excitability occur, the paralysis is slight, and may recover in a few weeks. If after the lapse of fourteen days only a partial reaction of degeneration occurs, we can count upon recovery in eight to nine weeks (according to the scheme, Fig. 312). If the affection is severe and associated with reaction of degeneration, a single electrical examination is not sufficient to determine whether it is curable or not. We must follow the entire course of the electrical

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changes. If after the lapse of about thirty weeks we can determine no return of the motility, and no improvement in the electrical irritability, or perhaps a still further impairment, the prognosis would be very grave indeed (Fig. 311 *c*). Conversely, the slightest improvement in the electrical reaction—viz., an increase of the galvanic irritability in the nerve or the muscle supplied by it, or even cessation of its further depression—is of favorable significance.

The above-mentioned prognostic laws apply only to those peripheral paralyses which, like a rheumatic facial paralysis, owe their existence to a single injury—*i. e.*, whose anatomic causes neither persist nor progress. It would be obviously illogical to apply this law to other types of facial paralysis, such as a paralysis caused by a tumor or by an osteitis of the petrous bone, or even to the facial paralysis of a bulbar palsy. The distinction is that the rheumatic facial paralysis tends to recover, whereas these others tend to persist and progress.

The prognosis of a case of *infantile spinal paralysis* (poliomyelitis acuta) is also very materially assisted by the results of an electrical examination. For, just as in rheumatic facial paralysis, the cause is in operation but a short time; even in the very severe cases of poliomyelitis the cause persists and progresses for only a few weeks, and after that the disease itself is stationary. If after fourteen days to three weeks no reaction of degeneration can be detected in certain of the affected muscles, we can safely apply the same prognostic law to them that we have just mentioned for a rheumatic facial palsy. Reaction of degeneration is, however, much more serious a sign in poliomyelitis than it is in a rheumatic facial palsy, for experience has taught us that in infantile paralysis those muscles which show reaction of degeneration never recover. The reason is probably that the lesion is located in central organs, which have little or no recuperative power.

With *lead palsy* reaction of degeneration almost always appears promptly, or, if this is not so marked, at least a pronounced diminution of the muscular irritability to the galvanic current (see pp. 816 and 818) promptly results; either effect is equally significant from the prognostic standpoint. If no pronounced changes in the electrical stimulability occur some time after the onset of a lead palsy, the prognosis will be favorable, just as in the rheumatic facial palsies. In lead paralysis, however, there is nothing absolutely unfavorable in the appearance of reaction of degeneration or in the pronounced diminution of electrical irritability mentioned above. Only examinations repeated at considerable intervals suffice to determine the prognosis in cases of lead paralysis with reaction of degeneration. If the reaction constantly progresses the prognosis will be grave; if, on the contrary, it remains the same the prognosis will be less serious; and if it evidently improves the prognosis will be favorable. For experience has taught us that when once an improvement commences in a lead paralysis, it almost always proceeds to complete recovery, and that partial recoveries are much less common. The same is found in polyneuritic and diphtheric paralyses.

We scarcely need mention that in polyneuritis there is always a

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very decided tendency to recover just as soon as the determining causes stop; and, of course, this fact, as well as the result of electrical examination, must be kept in mind in making the prognosis, which must be sharply determined for each individual muscle, and not for all those affected.

In central paralyses due to lesions in the supranuclear neurons, the results of electrical examination are of much less prognostic importance than in disturbances due to lesions of the peripheral neurons. For the electrical irritability is not, as a rule, very much altered in these cases even when the prognosis has from the onset been grave. However, an unfavorable prognosis should be given in disease dependent upon central lesions (cerebral hemiplegias, transverse lesions of the cord) which lead to a pronounced depression of the electrical irritability, because they point to a decided secondary involvement of the peripheral motor neurons. This involvement may be corrected up to a certain point by appropriate electrical treatment.

B. SPECIAL PART.

I. EXAMINATION OF THE DIFFERENT CRANIAL NERVES.

In examining the cranial nerves, the most convenient and logical plan is to take them up one after the other in their anatomic sequence. In the case of the nerves to the eye muscles, it is, however, more practical to discuss them all together, beginning with the third pair, the oculomotor.

1. CRANIAL NERVE: OLFACTORY.

To test this nerve we employ substances with different odors, such as cologne, asafetida, oil of anise, etc. We request the patient to smell each of them, closing first one nostril and then the other. It is rather convenient at the same to test the trigeminus, employing for this purpose acetic acid and ammonia. The two sides should be tested separately. If any differences are made out, we must not attribute them to the olfactory or the trigeminus until we have convinced ourselves by a careful rhinoscopic examination that they do not depend upon a local alteration of the mucous membrane.

Among other conditions, cerebral pressure will generally produce a paralysis of the olfactory. Huguenin, judging from his own experiments, considers that this condition possesses "diagnostic significance similar to that of the choked disk." A unilateral diminution of smell, provided no local cause exists in the nasal mucous membrane, appears most frequently as an accompaniment of the *hemianesthesias of hysteria*, and of the *traumatic neuroses*. It is purely functional. The so-called *capsular hemianesthesia*, which depends upon lesions of the posterior part of the internal capsule, is not ordinarily associated with any disturbance of smell. The olfactometer constructed for physiologic examinations has very little clinical utility, because gross changes, those easily demonstrated without any instrument, are the only ones of any diagnostic importance.

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II. CRANIAL NERVE: OPTIC.

1. **Determination of Acuteness of Central Vision.**—Snellen's or Pflüger's well-known test types are the most convenient and suitable for testing the acuteness of central vision. Landolt's new vision tests are more conveniently carried about, and therefore better adapted for examining patients in bed. Errors of refraction must first be corrected. Ophthalmologic text-books must be consulted for more exact directions. We should remember, however, that even if the acuteness of vision is

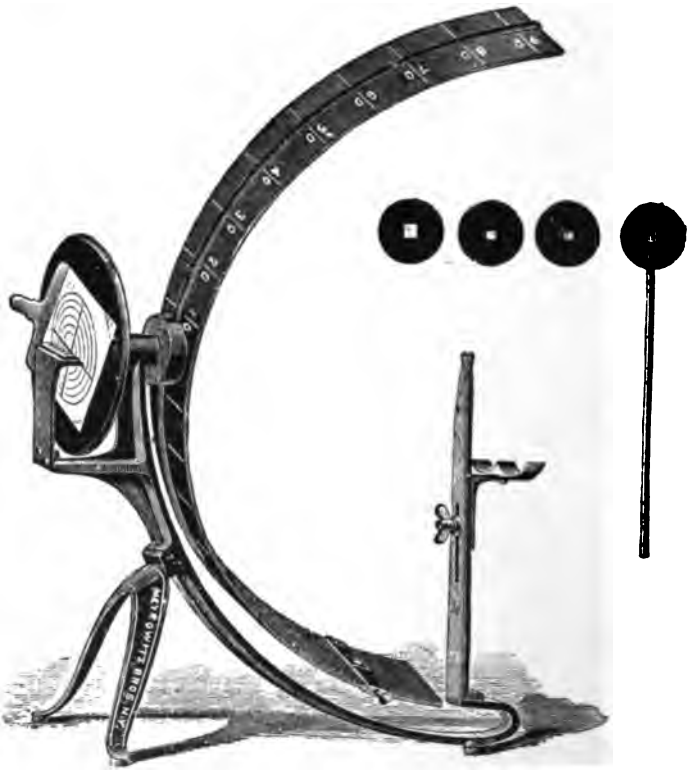

FIG. 314.—Perimeter: The examination may be made with the carrier which moves along the semi-circle, or the test objects may be carried along this by means of dark disks attached to a long handle, each disk containing in its center the test object. The patient's chin is placed in the curved chin rest; the notched end of the upright bar is brought in contact with the face, directly beneath the eye to be examined, which attentively fixes upon the center of the semi-circle. The other eye should be covered, preferably with a neatly adjusted bandage. The record chart is inserted at the back of the instrument, and, by means of an ivory vernier, the examiner is enabled to mark exactly with a pencil the point on the chart corresponding to the position on the semi-circle, at which the patient sees the test object. The various marks are then joined by a continuous line.

normal, we cannot absolutely exclude the existence of pronounced changes of the retina or of the optic nerve, so that an opthalmoscopic examination is essential (retinal hemorrhages, retinitis, choked disk, optic atrophy).

2. **Testing the Field of Vision**—We employ the *perimeter* (Fig. 314) to determine accurately the field of vision; to demonstrate hemi-

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opia and other visual defects (as central scotomata, quadrant defects and quadrant anopsia); to establish the existence of unilateral or bilateral limitations of the visual field, such as accompany hysteria and other neuroses, especially in the so-called traumatic neuroses; as well as to show the frequently associated fatigue of the retina. Books upon ophthalmology must be consulted for the technical application of this instrument.

We can determine the visual field quite accurately by a very simple device. The patient, with his left eye closed, sits opposite the examiner. The latter closes his right eye. The patient's right eye and the examiner's left eye are now fixed upon each other; the examiner then moves his finger in a frontal plane halfway between the two eyes, from the periphery into the field of vision. He can then directly compare the patient's field of vision with his own. Of course, the distance of the finger from each eye should be exactly alike. With but one eye it is difficult to be accurate about this distance, so that it is advisable for the examiner to orient himself by opening the closed eye from time to time. Pronounced defects in the visual field are, according to the author's experience, easily recognized by this method.

Significance of Scotomata or Defects in the Visual Field.—Dufour was the first to call attention to the great difference between "not seeing" (vision nulle) and "seeing indistinctly" (vision obscure). If the visual defect consists of a simple lack of perception of sight (not seeing), without obscuration of the affected areas in the visual field, the trouble must be attributed to a functional or anatomic lesion of the visual center in the cortex. Very likely the patient appreciated it for the first time during the examination. Should the patient, on the contrary, complain that the defect in the visual field does not appear to be complete, but merely an obscured area (in which case the patient is annoyed by this condition and is conscious of it), we should then naturally conclude that the visual center is intact, and that the visual defect depends upon some involvement of the visual conducting apparatus, either of the refracting media, the retina, the optic nerve, the optic tract, or the visual fibers. "Seeing indistinctly" is nothing more than the reaction of the intact visual centers to a faulty transmission of optical impulses; whereas "not seeing" is a consequence of the lack of optical appreciation of the object in question, and naturally it occurs only in functional or anatomic lesions of the visual center. This distinction between "not seeing" and "seeing indistinctly" is of importance in the differential diagnosis between *central* and *peripheral hemiopia*. Thus, the observation that the hemiopic scotoma in scintillating scotoma or in ophthalmic migrain is an absolute defect and not an obscuration of the visual field entirely agrees with the supposition (probable from other reasons) that the process is central and located in the meninges or in the cortex.

Visual Color Fields.—We are often assisted in the diagnosis of hysteric and other neurotic conditions by determining the visual color fields. These are frequently contracted and their sequence disturbed. The visual field for blue is normally the largest; but in the above

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affections we frequently find that other colors encroach upon its borders. The text-books upon eye diseases show how to determine these conditions by means of the perimeter.

Compare section 4 for the typographic diagnostic significance of defects in the field of vision.

3. **Ophthalmoscopic Examination** (see p. 703 *et seq.*).

4. **Topographic Diagnosis of Lesions in the Course of the Optic Fibers.**—Fig. 315 represents diagrammatically the course of the optic fibers from the retina to the occipital lobes. The fibers ari-

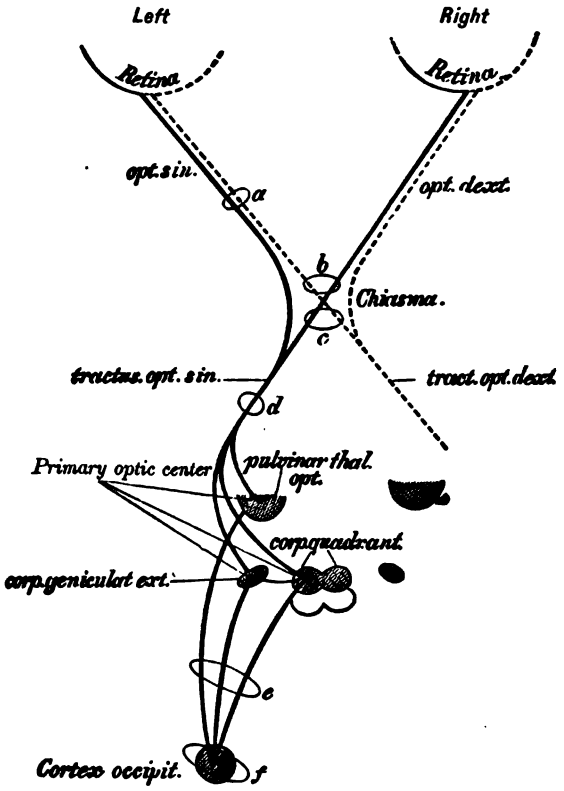

FIG. 315.—Diagram of the course of the optic fibers.

sing from homonymous retinal halves form the optic tract by a semi-decussation at the chiasm. Then they proceed, in part direct, but mostly indirect, through the so-called primary optic centers to the occipital cortex.

Without further explanation, the diagram (Fig. 315) makes it plain that a focal lesion (*a*) of the optic nerve will cause unilateral blindness; a lesion (*b* or *c*) in front or behind the chiasm will cut off the nasal halves of the retina and produce a temporal hemiopia; a lesion (*d*, *e*, or *f*) will cut off both left-sided retinal halves and produce a homonymous right-sided hemiopia.

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Grasset has modified Fig. 315 hypothetically in Fig. 316 in order to explain the occurrence of unilateral blindness from a lesion in the extreme posterior (sensory) portion of the internal capsule. This condition is sometimes observed instead of a hemiopia. For the sake of simplicity the optic fibers are represented in this diagram as uninterrupted—*i. e.*, the primary optic centers are omitted. The diagram assumes that the fibers which have not already crossed at the chiasm finally decussate between the corpora quadrigemina and then directly return to their original side; both *a* and *b* are supposed to be situated in the posterior part of the internal capsule. Hence a lesion in the internal capsule will, according to its position, produce either a crossed blindness—*i. e.*, *amblyopia* (lesion *a*) or a *homonymous hemiopia* (lesion *b*). Recent observations, however, seem to prove that lesions of the internal capsule probably do not produce hemiopic

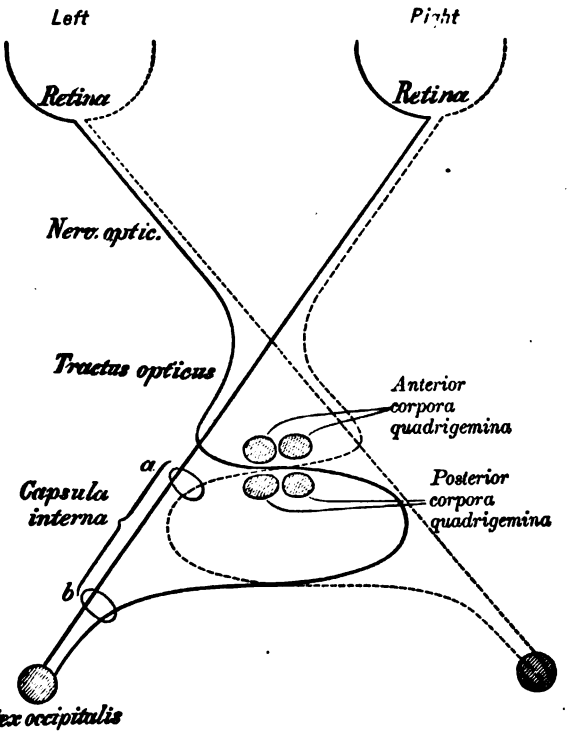

FIG. 316.—Grasset's modification of the preceding diagram: The optic fibers are depicted for simplicity sake, as if they were uninterrupted, without intercalation of the primary optic centers.

disturbances of sight. Henschen has recently examined the innervation of the different retinal quadrants—*i. e.*, the course of the visual fibers of each retinal quadrant in the trunk of the optic nerve and in the optic tracts. By dividing the retina into four quadrants with a vertical and a horizontal meridian he found that the visual fibers of each of these quadrants run as a compact bundle in the optic fibers. This circumstance can sometimes be utilized in establishing, by aid of an exact representation of the visual field, a local diagnosis of peripheral interruptions of the visual fibers.

The following diagrams from Henschen[1] illustrate this point:

5. Detection of Simulated Unilateral Blindness.—Unilateral blindness is quite frequently simulated. Ordinarily we can

[1] See Salomonsohn, *Deutsch. med. Woch.*, 1900, No. xlii., p. 677.

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discover it very readily by using the stereoscope. The instrument is placed in front of the two eyes; different letters are arranged so as not to coincide in stereoscopic union. The patient cannot recognize to which eye the individual pictures belong. If, now, he simulates a one-sided blindness, the simulation is evident, because he reads the characters which could be seen only by the supposedly blind eye quite as well as

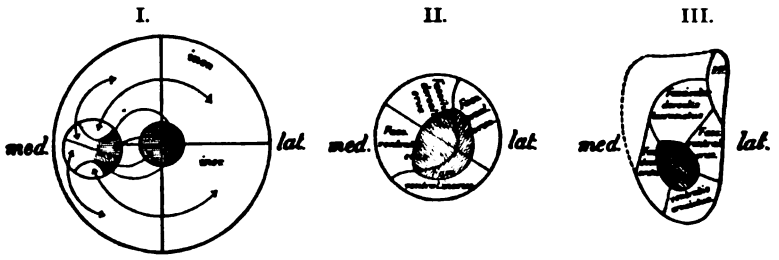

FIG. 317.—I. The four retinal quadrants: To the left is the position in cross-section of the fibers in the vascular part (anterior) of the optic nerve. The shaded parts correspond to the macula lutea—*i. e.*, the transmission of central vision. The arrows show the way the retinal quadrants correspond to the individual parts of the optic nerve. This arrangement of the fibers can be similarly made out at the part of the optic nerve which is visible to the ophthalmoscopic (optic disk) examination. *incr*-retinal quadrants with uncrossed fibers.

II. Cross-section of the optic nerve in the non-vascular (posterior) portion. Here the macular bundles (represented by shading) lie in the center of the nerve, and the fibers corresponding to the individual retinal quadrants are arranged like quadrants about an imaginary axis running in the center of the nerve.

Explanation of the terms in the figure: The fasciculus dorsalis cruciatus corresponds to the internal superior retinal quadrant. The fasciculus ventralis cruciatus corresponds to the internal inferior retinal quadrant. The fasciculus dorsalis incruciatus corresponds to the external superior retinal quadrant. The fasciculus ventralis incruciatus corresponds to the external inferior retinal quadrant.

The figure also shows that the quadrant arrangement is retained in the non-vascular part of the optic nerve—*i. e.*, by imagining the quadrant picture of the retina seen from in front (I) with the upper end of the vertical meridian rotated outward about 45°.

III. Cross-section of the posterior part of the optic tract. *P. F.*, Fibers of the pupillary reflex (see p. 842 *et seq.*). Other abbreviations have the same meaning as in II. The four quadrant bundles are bound together again, but still distinct. They surround the shaded bundle in the center, which corresponds to the tract of the macular fibers. The arrangement of the quadrant bundles in III. is very similar to that in the cross-section II., except that the pupillary fibers do not appear in the latter.

the other. There are, however, some cases of hysteric unilateral blindness with monocular vision which are really not simulated. In such cases the above would not show anything. Appropriate stereoscopic pictures for such an examination with the stereoscope will be found at the end of Dr. M. Burchardt's treatise, "Practical Diagnosis of Simulation" (Berlin, Enslin, 1891).

III. IV. VI. CRANIAL NERVES; THE OCULAR MUSCLES' NERVES, INCLUDING THE SYMPATHETIC; MOTOR INNERVATION OF THE EYE REGION.

1. Functions of the External Ocular Muscles.

The *III. cranial nerve* (oculomotor) supplies the following muscles: Levator palpabræ superior, rectus superior, rectus inferior, rectus internal, and oblique inferior, the pupillary sphincter, and the muscles of accommodation (both the latter from the short branch of the ciliary ganglion).

The *IV. cranial nerve* (trochlear) supplies the superior oblique (trochlearis) muscle.

The *VI. cranial nerve* (abducens) supplies the external rectus muscle.

The muscles which move the eyeball have the following functions:

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I. Internal rectus: movement of the eyes inward without any wheel motion.

II. External rectus: movement outward without wheel motion.

III. Superior rectus: movement upward and inward with rotation of the upper extremity of the vertical meridian inward.

IV. Inferior oblique: movement downward and outward with rotation of the upper extremity of the vertical meridian outward.

V. Inferior rectus: movement downward and inward with rotation of the upper extremity of the vertical meridian outward.

VI. Superior oblique: movement downward and outward with rotation of the upper extremity of the vertical meridian inward.

The external and internal recti suffice for directing the eyes in the horizontal axis, but movement in the vertical axis (upward and downward) requires the joint action of one rectus and one oblique. The principal function of the obliques is, therefore, to limit the tendency to wheel rotation of the eye, which would take place without their action. Any vision directed obliquely between the vertical and the horizontal axes necessitates the joint action of three different muscles. One of them acts to counteract the tendency to wheel rotation.

2. Paralysis of the Muscles which Move the Eyeball.

The disturbances of function which may result from a paralysis of the eye muscles can be essentially determined from the preceding description of their function. If one or more muscles of the eye are paralyzed, the eye will remain immobile when attempting to follow the direction of vision ordinarily accomplished by the muscles affected. To demonstrate readily the existence of a paralysis of the eye muscles, the patient should follow with his eyes (but with the head absolutely fixed) the movement of the examiner's finger in every direction, while the examiner compares the excursions of the two eyes. This is easily accomplished by observing the position of the corneal margin in relation to the angle of the eye, a procedure generally sufficient for a hasty test. A slight paresis may, however, be hidden by the tendency such a patient has to fuse the double images. Therefore, in any doubtful case, it is well to test each eye alone, covering the other eye. A slight weakness will sometimes be disclosed by having the patient attempt the extreme positions, and then observing either that the position cannot be retained for any length of time or else that it produces an accompanying tremor of the globe (nystagmus), although the weakness is not sufficient to diminish the size of the excursions. In testing the internal recti we must especially note their behavior in attempted convergence of the eye (see p. 836).

In complicated muscular paralyses of the eyes it is often difficult to form a judgment as to the function of the oblique muscles, because they are in such intimate association with the recti. In such a case, and especially in distinguishing the more delicate disturbances of the eye movements, testing the torsion of the eyeball is especially significant. If the eyeball moves normally, no axial deviations occur, because the

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axial deviating components common to all the eye muscles, excepting the external and internal recti (p. 827), are counteracted by the associated action of the other muscles. Just as soon as this extremely finely balanced associated action of the eye muscles is injured by the paralysis of such muscles as possess axial deviating components, axial deviations of the bulb must ensue. The determination of such axial deviations or movements of rotation in a definite visual direction, and the ascertaining which muscles are responsible, permit conclusions in regard to the finer defects of movement. These cannot be made out in an examination of the gross excursions of the eyeballs, for in complicated paralyses it is especially difficult to recognize the functions of the obliques in the movements of the eyeball, because the direction in which these muscles pull is affected by the action of the two recti muscles. In order to draw diagnostic conclusions concerning rotation it is only necessary to keep in mind that rotation with a tendency of the upper extremity of the vertical meridian inward, depends upon the superior rectus and the superior oblique, while the reverse rotation depends upon the inferior rectus and the inferior oblique. If a rotation occurs in any individual movement of the eye, we naturally infer that the fault depends upon the muscle, which by its help should prevent such rotation, and which, unaided and unopposed, would accomplish the opposite rotation. We test rotation by having the patient look upward and outward, and then downward and outward. While looking upward and outward, rotation of the right eye in the direction of the hands of a clock (as seen by the examiner) signifies paresis of the inferior oblique and preservation of the superior rectus; while looking downward and outward, a similar rotation signifies paresis of the inferior rectus with intact superior oblique.

More pronounced paralyses of the eye muscles are also evidenced by strabismus (*paralytic squinting, paralytic strabismus*). Paralytic strabismus can be differentiated from concomitant strabismus by the fact that in the former a deviation of the eyeball from the normal reciprocal position shifts with the direction of vision, whereas in concomitant strabismus it remains the same in every direction of vision.

Patients with paralyses of the eye muscles ordinarily complain of double vision; hence the existence of diplopia and its peculiarities may be utilized for diagnosis. For this purpose it is essential to know to which eye each one of the double pictures belongs. This is determined most readily by putting before the patient's eyes differently colored glasses, which may be conveniently slipped into the spectacle frame used by ophthalmologists. From the patient's statements as to the color of the pictures, it is easy to determine the eye to which each of the double pictures belongs. In many cases, especially in old paralyses, double vision is first discovered by making use of these colored glasses, whereas without this device patients disregard one of the pictures—*i. e.*, they no longer see it. It is quite possible, without the colored glasses, to determine to which eye each of the double pictures belongs, by covering one of the patient's eyes, and then having him say which of the pictures disappears. In paralyses of the eye muscles which act horizontally, the double pictures will stand side by side. These we call *crossed double pictures* (*crossed double vision*), if the picture lying to the left (from the patient) belongs to the right eye. On the contrary, we speak of

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homonymous or *non-crossed double pictures*, if the left picture belongs to the left eye. In accordance with well-known physiologic laws of projection, the appreciated picture—that is, the projection of the retinal picture in space—seems to be inverted. Hence *homonymous* or *non-crossed double pictures* depend upon a crossing of the visual axes in front of the object—*i. e.*, upon convergence; or, in other words, upon abducens paralysis. On the contrary, *non-homonymous* or *crossed double pictures* depend upon divergence of the visual axes—*i. e.*, paralysis of one or both internal recti. The following laws of double vision are applicable for diagnostic use. They depend upon the fact that the projection of the retinal pictures in space seems to be inverted, just as does the corresponding eye:

(1) That eye is paralyzed whose image appears to be deviated from that of the other in any one direction of vision; and (2) the paralysis involves the muscle or muscles in whose direction of pulling, projected into space, the deviated picture appears to move when the direction of vision is altered.

In examining for double vision, it is useful to know that many cases see double only objects which are at a considerable distance from the eye, perhaps because the effort of accommodation in focusing at a near distance simplifies and disregards the second indefinite picture. It is also important to remember that double pictures separated but little from one another are frequently not recognized as such by the patient, but are described by him as "blurred"—*i. e.*, obscure—vision. Closing one eye improves the patient's vision in this type of disturbance.

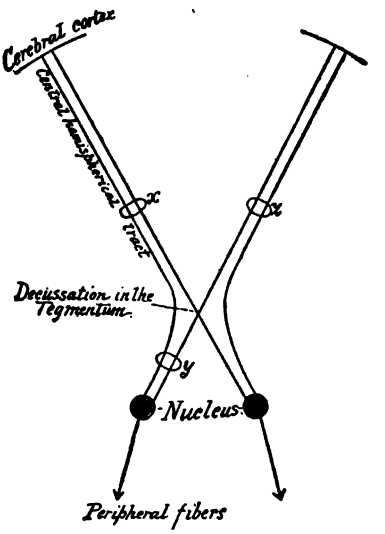

FIG. 318.—Diagram of the bilateral central innervation of the nerves to the eye muscles and most of the remaining motor cranial nerves: The focus *x* causes no evident paralysis, because the tracts of the opposite side remain intact; the lesion *y* would, however, and so would lesions *x* and *z* together. Simultaneous lesions in the latter places (*x* and *z*) cause so-called bulbar paralysis (p. 878).

Besides double vision, patients with paralysis of the eye muscles very frequently complain of *vertigo* (ocular vertigo), and *disturbances of equilibrium* in walking and standing (see p. 882). This is psychical, and depends upon disturbances of orientation in space. According to the author's experience, ocular vertigo depends principally upon paralysis of the ocular muscles which rotate the eyeballs; whereas mere paralysis of the abducens and internal recti ordinarily causes no vertigo. This is comprehensible without further explanation; for, despite the diplopia, the patient with these latter paralyses sees objects at least in normal vertical orientation; whereas in paralysis leading to rotation, objects appear upside down. *Ocular vertigo* differs from other kinds of vertigo, inasmuch as it disappears upon closing the eyes.

If the paralysis is simple, and especially if it is unilateral, the diagnostic laws concerning the significance of the kind of double pictures mentioned above easily lead to a diagnosis; but in complicated binocular paralysis the recognition of the paralyzed muscles from the double pictures alone is often very difficult. Here one of the most reliable methods is to determine the fields of vision by means of the *perimeter*. It is merely an improved method of testing the excursions of the eyebulb in various directions, and is accomplished by fixing the head by means of the perimeter, and estimating those points in the instrument which can be directly (centrally) seen—*i. e.*, at which fine type can still be read.

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Monocular double vision, which is not uncommon in hysteric conditions, must not be confused with binocular diplopia occasioned by paralyses of the muscles of the eye. The former is often attributed to a partial spasm of the ciliary muscle, in consequence of which a portion of the lens acts as a prism and produces a second picture upon the retina. This theory is probably incorrect, for certainly it will not apply to many cases; in those which the author himself has observed the monocular diplopia was evidently a purely psychical phenomenon. Monocular diplopia is therefore a stigma of hysteria, and it is important to bear this in mind,

FIG. 319.—Unilateral ptosis (New York City Hospital).

in any instance of double vision, before making a diagnosis of ocular muscle paralysis, which causes only binocular diplopia.

The diagnostic importance of ocular muscle paralysis is considerable, because experience has shown that it almost always depends upon a lesion of the peripheral motor neurons—*i. e.*, the subnuclear fibers, or the nuclear region itself—and practically never upon a supranuclear lesion, or a lesion of the central fibers. As each one of the ocular

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nerve-muscles must possess a central tract running to the cortex, this peculiarity demands explanation. This may be given by the supposition that the nuclei of the ocular muscles are innervated not by one, but by both hemispheres. Fig. 318 readily explains why a unilateral hemisphere lesion, even if it destroys the central fibers, causes no apparent paralysis of the corresponding eye muscle; although a portion of the innervation for both sides is affected, the intact hemisphere apparently furnishes sufficient innervation for both sides. Therefore, since there is no method of measuring the absolute power of the muscles, the bilateral defect of innervation escapes observation. When the lesion, even though small, occurs at *y*, in the region of the nucleus or below it, all the fibers are interrupted. Still another circumstance may aid in preventing the occurrence of a muscle paralysis in unilateral supranuclear lesions of the hemisphere; it is possible that the central fibers do not run as compactly as is represented in the figure, but are distributed over different points of the cortex, so that a circumscribed lesion could not very easily destroy many of them.

On the contrary, in bilateral hemispheric lesions or in excessive superficial affections of the two sides of the brain (meningitis, etc.), bilateral ocular muscle paralyses may occur as "pseudobulbar" paralyses (p. 876 *et seq.*). A few

FIG. 320.—Bilateral ptosis, normal position (Neurologic Department, Massachusetts General Hospital).

examples in which with unilateral cortical lesions, contrary to ordinary experience, isolated crossed ocular muscle paralyses have been discovered—*i. e.*, ptosis—are explained by assuming that in many individuals the bilateral hemispheric innervation is insufficiently developed, or that the central fibers are more compact in their course and localized. The parietal lobe has been found to have suffered in these few cases of central ocular muscle paralyses. This will be discussed upon p. 834 along with the localization of the center for conjugate ocular movements (see Fig. 339, p. 884).

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In paralyses of the ocular muscles one can therefore generally exclude supranuclear causes, so that a diagnosis of the situation of the lesion is

FIG. 321.—Bilateral ptosis, showing effort to open eye, using muscle of forehead (Neurologic Department, Massachusetts General Hospital).

for the most part limited to a distinction between a nuclear and a subnuclear type. This differentiation in the case of the oculomotor is fre-

FIG. 322.—Bilateral ptosis, showing effort to open mouth (Neurologic Department, Massachusetts General Hospital.

quently simple, because in subnuclear—*i. e.*, entirely peripheral—paralysis the nerve is affected almost always as an entirety; whereas in

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nuclear paralyses the separate functions of the nerve can be affected, so to speak, individually, since the oculomotor nucleus is anatomically situated in functionally different areas. Especially characteristic for most nuclear paralyses is the non-involvement of the pupillary and accommodation fibers of the oculomotor. The following plan, copied from Kahler and Pick, presents the anatomic arrangement of the different parts of the oculomotor nucleus and their relations to the neighboring trochlear nuclei. Its study will facilitate a more exact local diagnosis in nuclear paralyses.

Anatomic Arrangement of the Different Components of the Oculomotor Nucleus.

[Anterior (proximal).]

1. Accommodation.
2. Sphincter iridis.

Medial. { 3. Rectus internus. / 4. Rectus inferior. } { 5. Levator palpebræ superioris. / 6. Rectus superior. / 7. Obliquus inferior. } Lateral.

Trochlearis.

[Posterior (distal).]

3. Ptosis, Including So-called Sympathetic Ptosis.

By ptosis is meant a paralytic drooping of the upper eyelid so that the lid covers the eyeball more or less completely and thereby narrows the ocular aperture. Ptosis ordinarily results from paralysis of the levator palpebræ superioris, supplied by the oculomotor. (See below, Sympathetic Ptosis.) In this connection it is necessary to differentiate paralytic ptosis from a spasmodic condition of the orbicularis oculi by which the upper lid is drawn down over the eye and the ocular aperture narrowed. The distinction is usually easy. In the former, paralytic or true ptosis, the excursion of the upper lid upward is diminished if not entirely prevented; whereas in a spasm of the orbicularis this need not be the case. In a spasm of the orbicularis, moreover, the wrinkles about the eye are ordinarily more prominent, and the eyebrow is placed lower than upon the normal side; whereas in paralytic ptosis the eyebrow, by means of its innervation by the facial, seems instinctively elevated higher than normal to counterbalance the defect.

The so-called *sympathetic ptosis*, which was first described by Horner in 1869, must not be confused with ptosis due to paralysis of the levator palpebræ. Here, although the lid aperture of the affected side is narrower than upon the healthy side and its upper lid hangs lower, it can be demonstrated that the excursions of the levator palpebræ are in no way diminished; but the eyeball often seems sunken into the orbital cavity, the pupil is ordinarily somewhat narrowed, and frequently abnormalities of the sweat secretion and of the vascular innervation appear upon the affected side of the face. This symptom-complex depends upon a paralysis of the so-called Müller muscle (supplied by the sympathetic), comprising the smooth muscle fibers of the superior oblique, inferior oblique, and orbitalis. Stimulation of the two former widens the lid aperture; stimulation of the latter projects the eyeball somewhat forward from the orbit. A paralysis of these fibers causes a diametrically opposite appearance of the eyes to that in Graves's disease (exophthalmos) (Gräfe's symptom, see p. 838). This latter symptom is to be attributed to an irritation of these smooth muscles.

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Congenital ptosis, which is not very rare, is partly sympathetic in nature, and partly dependent upon a congenital (nuclear) paralysis of the levator palpebræ superioris.

4. Conjugate Paralyses and Conjugate Deviation of the Eyes.

The so-called conjugate eye paralysis will be discovered in a binocular examination for the mobility of the eyes (described above) by means of fixation upon a finger held in front. It occurs in cerebral diseases, and consists of weak, deficient, or absent mobility of the two eyes to the same side. These conjugate paralyses are usually due to a lesion situated in a tract which leads possibly from the middle portion of the frontal lobe, adjacent to the anterior central convolution, to the nucleus

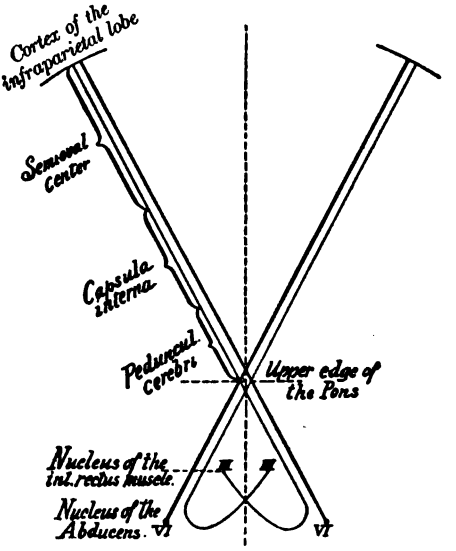

FIG. 323.—Diagram of the tracts for the associated lateral movements of the eyes: The upper half of the diagram, as far as the decussation (at upper edge of the pons), is to be regarded as a frontal section; the lower half of the diagram (the pons), as a horizontal section through the brain. The heavier of the two pairs of lines which intersect each other represents the central tract of the abducens; the lighter, the central tract of the internal rectus of the opposite side. In conformity with an incorrect conception, this diagram represents the infraparietal lobe not only as giving passage to innervating fibers, but also as the center for innervation of ocular movements.

of the abducens of the opposite side and to the nucleus of the rectus internus of the same side. They may probably also be due to a lesion in a fasciculus which is situated in the infraparietal lobe and connects the centers for ocular movement with the center for the optic nerve (Landouzy and Wernicke). Fig. 323 is a schematic representation of the course of this tract based upon pathologic findings (Leichtenstern-Hunnius), and conforms to the old view now proven to be incorrect, that the infraparietal lobe not only gives passage to innervating fibers, but is also the center for the innervation necessary for ocular movements. It will be seen that the fibers for the rectus internus, instead of passing directly to this nucleus, take a circuitous course through the

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region of the opposite abducens nucleus, and that the entire tract decussates in the anterior half of the pons.

In order to explain the way in which this tract can be affected by cerebral lesions at different locations, it is best to simplify the diagram, as in Fig. 324. The arrows mean that the tract proceeding from the left hemispheric cortex supplies the lateral movements of the eyes to the right; the tract from the opposite side, those to the left. With this explanation it is easy to understand that a lesion (x) above the pons will prevent the movements of the eyes to the side opposite the lesion, while, on the contrary, a lesion (y) below the upper edge of the pons will paralyze the movements of the eyes to the same side as the lesion.

Since conjugate paralyses are ordinarily combined with a conjugate deviation of both eyes to the side of the non-paralyzed antagonists,

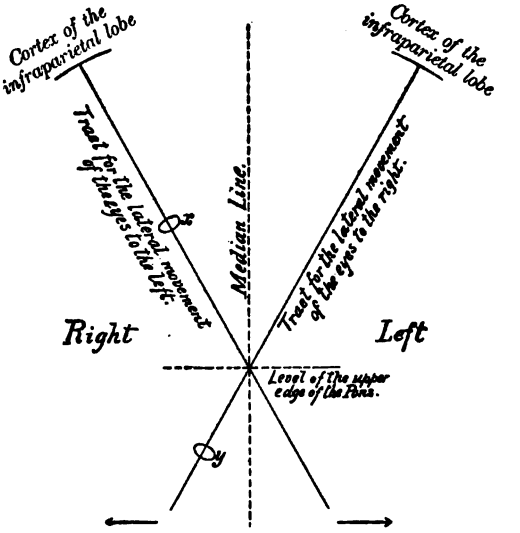

FIG. 324.—The same diagram simplified.

with a lesion x (Fig. 324) an ocular deviation to the right will be noted, and with a lesion y one to the left. Briefly, in lesions above the pons the patient looks toward his cerebral lesion; whereas in lesions of the pons and below the pons he looks away from it.

Conjugate paralysis with deviation of the eyes occurs in cerebral lesions, especially hemiplegia (hemorrhage, softening). Like hemiplegic paralyses of the extremities, it is frequently an indirect focal symptom, depending upon lesions located at various points, and then generally soon disappearing with the subsidence of the remote action. In some cases, however, the persistence of the hemiplegia proves that the entire unilateral voluntary tract is destroyed, and with it the accompanying conjugate ocular tract running through the internal capsule, and yet the conjugate ocular paralysis gradually disappears. We are compelled to

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assume, therefore, that, although most of the tract decussates, there must exist in the other hemisphere an uncrossed tract, possessed of the same function, which in apoplexy is affected more or less strongly by the remote action, but which, after the subsidence of the apoplexy, can take the place of the affected crossed tract. Fig. 324 must therefore be modified in accordance with this theory, as in Fig. 325. Naturally it may also be inferred that the vicarious action of the healthy hemisphere can be improved by constant use.

Besides being due to paralysis, conjugate deviation of the eyes may result from spasmodic action. This cause may be assumed when we

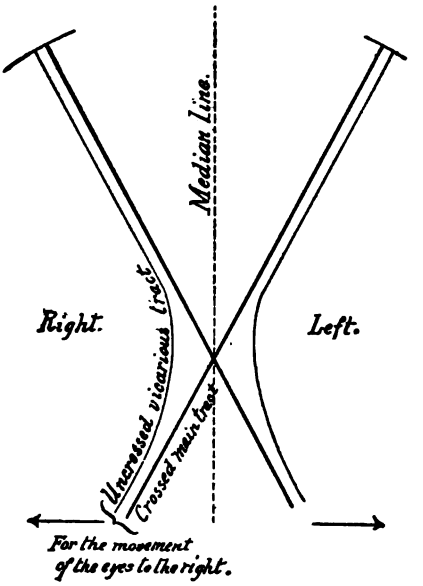

FIG. 325.—The same diagram modified to include the uncrossed vicarious innervation.

note the existence of similar spasms in other muscles upon the side of the deviation. The local diagnostic conclusions from the direction of the deviation are then naturally simply reversed.

5. Paralysis and Weakness of Converging Movements of the Eyes.

Convergence of the eyeballs requisite for binocular vision of near objects is naturally affected or rendered impossible by paralysis of one or both internal recti. However, characteristic conditions also do occur in which the internal recti functionate normally for all conjugate lateral movements of the eyes, but not for converging movements. Such observations have led to the assumption of a separate convergence center localized, it seems probable, in the pons. Its existence is still, however, problematic, because isolated convergence paralysis could quite as well depend upon the paralysis of a special tract supplying each internal rectus as upon the lesion of a center. A lesion of a central tract—*i. e.*, a supranuclear oculomotor and, naturally, a bilateral convergence tract—different from the tract for the conjugated movements of the eyes (Fig. 323) would explain the peculiarity. The only thing which seems to be certain (and this was con-

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firmed by one of the author's autopsies upon a tumor of the pons) is that convergence can be paralyzed by a lesion of the pons, while the other movements (conjugate, see p. 834 *et seq.*) of the internal recti remain intact. In this sense the symptom can be utilized for local diagnosis.

Without characteristic and complete paralysis, a mere weakness and insufficiency of convergence may be of considerable diagnostic importance in neurasthenic conditions and in exophthalmic goiter. In the latter this phenomenon is called *Möbius's symptom.* Insufficiency of convergence, which occurs also in myopia, is experienced subjectively by the appearance of the so-called asthenopic difficulties, by a sense of fatigue, by obscure and double vision when steadily

FIG. 326.—Exophthalmic goiter, showing exophthalmos and goiter (Dr. J. J. Putnam, Massachusetts General Hospital).

looking at near objects, and, on the other hand, objectively confirmed by the exhibition of latent external strabismus for near vision. This latent external strabismus appears when the binocular focusing power fails, and can be demonstrated by fixing the glance upon a near object—*e. g.*, 25 cm.—and then suddenly covering one eye with the hand. With insufficient convergence the covered eye will noticeably deflect outward, since the effort for convergence has become unnecessary. In spite of such subjective and objective confirmation of insufficiency of convergence, the degree of convergence which is transitorily accomplished by vigorous voluntary impulse is in such cases oftentimes considerable, so that, according to the author's experience, the Landolt ophthalmodynamometer does not suffice to demonstrate the insufficiency in exophthalmic goiter.

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6. Nystagmus.

In examination of the eyes one must always be on the lookout for nystagmus, by which is understood a rhythmic oscillation of the eyeball, especially marked when the eyes are in an extreme position. It is much more commonly a lateral than a vertical movement. It is generally an intention tremor. It frequently occurs associated with paralysis of the eye muscles (see p. 827), and without demonstrable paresis in many affections of the eye and of the brain, most commonly, however, in multiple sclerosis. The neurodiagnostic importance of nystagmus is not so great as has been supposed, because it occurs in all sorts of ophthalmologic affections, especially in those in which early in life the sight is quite defective—*e. g.*, in corneal opacity, in cataract, either congenital or early acquired, in congenital iridochoroiditis and retinitis pigmentosa, in coloboma of the choroid and of the retina, and finally in albinismus. [Oftener than true nystagmus in these last-named conditions, we encounter irregular twitching movements of the eyeballs, to which the designation nystagmiform movements is given. —Ed.]

7. Contractions of the Ocular Muscles.

A contraction of certain of the external ocular muscles, evidenced by anomalous positions of the eyeball, plays a part of some importance in hysteria. These positions change so constantly that it is impossible to confuse them with paralyses or with concomitant strabismus; besides, in hysteria, as contrasted with the latter condition, a voluntary movement fails to alter to any considerable extent if at all the position of the deviated eye in relation to the lid aperture. The history will generally aid in any doubtful case. A differential diagnosis between spasm and paralysis might be difficult in some instances unless the pressure of contracture in an adjoining muscle (facial spasm, blepharospasm) should decide the question.

(Compare p. 836 in regard to conjugate deviation of both eyes as a spastic phenomenon.)

The so-called "Gräfe's sign," which occurs in exophthalmic goiter, is probably to be regarded as a spasmodic contracture of the sympathetic superior oblique muscle (p. 833). This sign gives the patient with exophthalmic goiter a very characteristic appearance, and is of considerable diagnostic importance. It is elicited by having the patient look down gradually; as the eyeball lowers, it is noticed that the upper lid does not follow the movement of the eyeball, but either remains still or only moves slightly, thus exposing a more or less broad band of sclera between the cornea and the upper lid. For its demonstration it is important to avoid a dazzling light, because, apparently for protection, this seems to furnish sufficient stimulus to enable a patient to overcome the phenomenon.

8. The Pupillary Phenomena.

Diagnostically these are of the greatest importance.

Diameter of the Pupils.—The size of the pupils varies with the illumination. It is best to observe them in illumination of medium intensity. In doubtful cases we should compare the pupils of the patient with those of a healthy man under the same light conditions.

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Schirmer[1] found that the diameter of the pupil under physiologic conditions varies greatly in different individuals, and particularly at different ages. In an individual case, however, the diameter is constant for wide ranges of illumination (from 100 to 1100 meter-candles), provided that the observation be made after the eye has fully adapted itself to the particular illumination, as it does within a few minutes. If we bear this fact in mind in estimating the diameter of the pupil, we are to a certain extent independent of the existing degree of illumination. This diameter of the pupil, determined several minutes after full adaptation, must consequently be employed as a basis for the recognition of pathologic variations. Schirmer obtains the necessary degree of illumination of 100 to 1100 meter-candles by placing the patient within one meter of a window which is well illuminated by the sun, and yet not exposed to direct sunlight. The pupillary diameter is then determined while the patient relaxes his accommodation and convergence by looking at some distant object. Schirmer states that the observation should not be made when passing clouds are present, since the illumination will vary and may be less than 100 meter-candles. Tange found by observing these conditions that the pupillary diameter varied between 2 and 4 mm., being dependent upon age, refraction, and sex, and that in the majority of cases it was between 2.5 and 3 mm. In advanced life the pupil is usually smaller than in youth.

Pupillary narrowing (*myosis*) is found physiologically during sleep and old age, pathologically as an early symptom in *tabes dorsalis*, and in *progressive paralysis*. Eserin, pilocarpin, opium, morphin, and chloroform (the latter in pronounced narcosis) narrow the pupil. Sympathetic myosis from lesions of the (pupillary) dilating fibers of the cervical sympathetic, from disease of the sympathetic itself or of the oculopupillary fibers connecting the sympathetic nerve with the first dorsal segment of the spinal cord, is of local diagnostic importance.

Pupillary dilatation (mydriasis) occurs in complete loss of consciousness, in severe pain, in dyspnea, in peripheral blindness (especially from optic atrophy and glaucoma), in general oculomotor paralysis, and in some cases of tabes dorsalis and progressive paralysis. Atropin, duboisin, cocain, chloroform (in the early stages of narcosis) dilate the pupil. Children, as a rule, have dilated pupils.

W. Riegel[2] describes as a sign of neurasthenia, under the name of "alternating mydriasis," a dilatation appearing sometimes in one, sometimes in the other eye with a normal illumination.

Irregularity in shape of the pupils may, of course, occur in diseases of the nervous system, but in most cases it depends upon some local disease in the neighborhood of the pupil (synechia).

Inequality of the pupils is rare in health, and when present most frequently depends upon an unequal refraction of the two eyes. It is not uncommon in the various unilateral cerebral affections, in progressive paralysis, in tabes dorsalis, in unilateral disease of the sympathetic, of the oculomotor or of the optic nerve, and in migraine attacks. An inequality of the pupils is fairly common in neurasthenia, and is then usually of a vacillating character.

See p. 846 *et seq.* concerning an inequality of the pupils which depends upon the loss of reaction and dilatation of one pupil.

[1] *Deutsch. med. Woch.*, 1902, No. 13.
[2] *Zeits. f. Nervenheilk.*, 1900, vol. xvii., p. 169.

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On account of the *crossed pupillary reaction* (compare below), both pupils are equally contracted when only one is exposed to light. Yet, not rarely, persons are observed in whom the pupil adjacent to the source of light is narrower than the shaded pupil. Too little attention is paid to the diagnostic significance of this phenomenon. It may be that it is a stigma of neurasthenia.

Anomalies of Pupillary Contraction. — Pupillary Light Reflex.—Exposure of the pupil to illumination narrows not only the pupil of the same side, but also the pupil of the other eye, thus giving rise to a direct- and a crossed-light reflex or reaction. The part of the oculomotor nucleus (iris nucleus, see p. 833) which innervates the iris must be considered as the center of this reflex.

A convenient method of testing the reaction of the pupils to light is as follows: With a moderate illumination (candlelight) in front of the patient's face we alternately expose and then shade one eye with the hand, watching the effect of the light upon that pupil and upon the other pupil; then we do the same thing to the other eye. At the same time the patient must avoid any attempt at convergence or accommodation. If the test with a moderate light does not produce the pupillary contraction, the examination should be repeated with a dazzling light (sunlight, an illumination lens, or a concave mirror).

This customary procedure for the exact quantitative estimation of the reaction of the pupil to light is subject to a number of sources of error, which are due to the fact that the differences between the effect of light upon the macula lutea and upon the peripheral portions of the retina are disregarded, and also to the fact that the narrowing of the pupils by convergence and accommodation cannot be positively excluded. As the result of minute investigation, Schirmer[1] consequently gives the following rules for the accurate study of the reaction of the pupils to light: "The patient should be seated at a distance of one meter from a well-lighted window which is not exposed to the direct rays of the sun. After the eyes have adapted themselves to this illumination, the diameter of each pupil is observed; the reaction of each pupil to light is now determined by holding the hands in front of the open eyes and then rapidly withdrawing one of the hands. The reaction of each pupil to light is next determined while the opposite eye is unshielded from illumination." The reaction is more marked with the first method of examination (with the opposite eye shielded), since the exclusion of the crossed innervation causes the pupil to dilate before the reaction occurs, or, at least, to be more susceptible to light. This procedure is consequently the one best adapted for determining the vestiges of a diminished direct pupillary reaction. The crossed pupillary reaction is then determined by observing the eye while the opposite one is alternately shielded and exposed. This investigation is repeated for each eye. The sum total of the direct and uncrossed pupillary reactions, and consequently the last remnants of a diminished reaction of the pupils to light, is best estimated by shielding both eyes, simultaneously exposing them, and then observing the reaction of both pupils.

See p. 843 *et seq.* concerning the so-called "hemiopic reaction"—*i. e.*, hemiopic rigidity of the pupil.

Fig. 327 represents the course of the light reflex which was formerly accepted as accurate. It was based upon the supposition that the stimulus to the iris nucleus, causing a reflex narrowing of the pupil, originated in the so-called primary optic centers (pulvinar, anterior corpora quadrigemina, and corpus geniculatum externum see Fig. 315).

[1] *Deutsch. med. Woch.*, 1902.

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The bilateral character of the light reflex was attributed not only to the semi-decussation of the optic fibers, but also to the bilateral connection of the iris nuclei (see Fig. 327).

Although the above conception was advocated in the earlier German editions of this work, it is no longer tenable, because it has been proved clinically, as well as experimentally, upon animals, that lesions of the primary centers do not cause any disturbances of the pupillary reflex. We must therefore conclude that the sensory fibers, which transmit the pupillary light reflex, are distinct from the visual fibers, and certainly leave the optic tract before its entrance into the optic centers. Physiologic examinations and clinical observations induced Bechterew[1] to con-

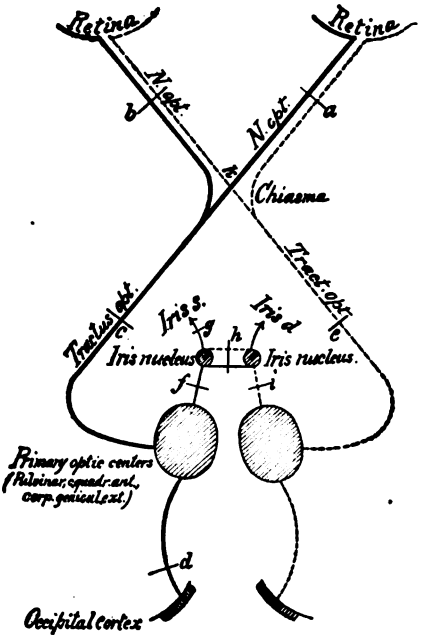

FIG. 327.—Old diagram of the pupillary light reflex, which no longer obtains: The primary optic centers (pulvinar, corpus geniculatum externum, and anterior corpora quadrigemina) are represented as a single center for the sake of simplicity (see Fig. 315).

clude that special pupillary fibers exist in the optics and that these fibers, after separating from the visual fibers at a certain distance behind the chiasm, run through the gray matter near the third ventricle to the iris nucleus.

Owing to the occurrence of hemiopic pupillary immobility, it must be assumed that the fibers of the optic nerve which are involved in the pupillary reflex experience a semidecussation in the chiasm in the same manner as do the visual fibers.

Bechterew's diagram for the pupillary light reflex, which was reproduced in the former German edition of this work, does not sufficiently

[1] *Deutsch. Zeits. f. Nervenheilk.*, vol. xvi., parts 3 and 4, p. 193 *et seq.*

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explain physiologic conditions; it contained no fibers whatever for the direct pupillary reaction, and certain of its details had not been positively confirmed. The author believes that the following diagram (Fig. 328) of the pupillary reaction will explain the physiologic and pathologic facts now at our disposal, and that it is not based upon as many unfounded suppositions. The previously mentioned and more recent anatomic postulates have been regarded in its construction.

The most important lesions, clinically, are indicated in the diagram by the letters *a, b, c, d, e, f, g,* and *h.* The clinical symptoms of these lesions are as follows:

Lesion *a:* Transverse section of one optic nerve. Blindness and

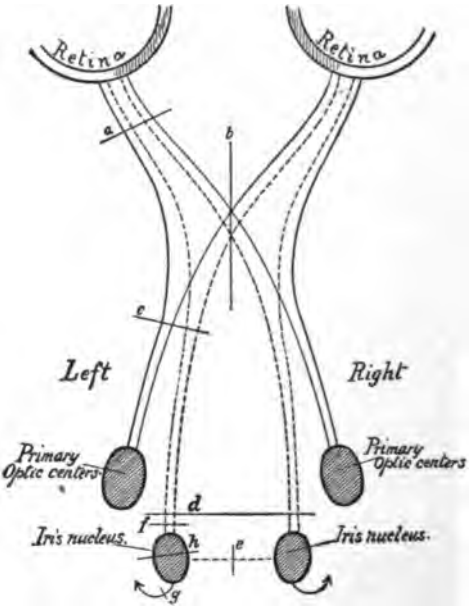

FIG. 328.—Diagram of the pupillary light reflex: The continuous lines represent the visual fibers; the dotted lines, the centripetal fibers for the pupillary light reflex. The central tract for contraction of the pupil by convergence and accommodation is not indicated in the figure; it might be imagined as passing to the iris nucleus from the side.

absence of the direct pupillary reaction in the eye of the same side, with maintenance of the crossed pupillary reaction. The crossed reaction is absent in the opposite pupil.

Lesion *b:* Sagittal section of the chiasm. Bilateral temporal hemiopia. The direct and crossed pupillary reactions are normal in both eyes when tested in the usual manner by diffuse light. Direct and crossed hemiopic pupillary immobility in both eyes (p. 843 *et seq.*) from involvement of the nasal halves of both retinas.

Lesion *c:* Section of one optic tract in front of the primary optic center involving the centripetal fibers for the pupillary reflex. Homonymous hemiopia and homonymous hemiopic crossed and direct pupil-

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lary immobility (see below). Maintenance of the direct and crossed pupillary reaction by the ordinary test with diffuse light.

Lesion *d:* Involvement of all the centripetal fibers of the pupillary light reflex upon both sides after their separation from the visual fibers. Absence of the crossed and direct reactions of both pupils to light with maintenance of visual power, maintained movements of the eyeballs, and maintained reaction of the pupils by convergence and accommodation (Argyll-Robertson's symptom in tabes dorsalis and progressive paralysis, p. 846).

Lesion *e:* The direct and crossed pupillary reactions normal in both eyes when tested with diffuse light. As a result of the injury to the fibers connecting the iris centers, however, only the direct reflex is present when the temporal halves of the retinas are tested, and only the crossed reflex is present when the light falls upon the nasal halves.

Lesion *f:* Section of the centripetal fibers for the pupillary light reflex proceeding from the left halves of both retinas. Homonymous hemiopic pupillary immobility in both eyes upon illumination of the left halves of both retinas. The visual power and the pupillary reaction to convergence and accommodation are maintained in both eyes (see p. 847.)

Lesion *g:* Section of the motor fibers leading from the left iris nucleus to the pupil of the same side. Immobility of the affected pupil to light, both direct and crossed, and also during convergence and accommodation. The opposite pupil reacts normally both to direct and crossed impulses, and also to accommodation and convergence. The sharpness of vision and the visual fields are normal in both eyes.

Lesion *h:* Destruction of the left iris nucleus. Immobility of the left pupil, both to direct- and crossed-light impressions and also to accommodation and convergence. This is associated with hemiopic immobility of the right pupil during illumination of the left halves of the retinas.

A number of direct observations are still necessary to demonstrate the complete accuracy of this diagram. No observations of types *e* and *f*, for example, have as yet been published. In type *h* investigations have not been made to determine whether hemiopic immobility of the opposite pupil is really present from an involvement of the retinal halves corresponding to the injured side. Not until positive statistics have been obtained in reference to these points can this diagram be defended as absolutely correct. If it be found that the lesions *e*, *f*, and *h* do not materialize in actual practice, the diagram must be modified.

Hemiopic Pupillary Immobility (*Hemiopic Pupillary Reflex*).—Fig. 328 furnishes a key to the appreciation of the so-called hemiopic pupillary reaction (Wernicke) or hemiopic pupillary immobility. When one side of the optic tract is injured at the point *c*, or when the centripetal fibers of the light reflex proceeding from homonymous retinal halves are injured in the neighborhood of the point *f*, the result must be that the light reaction from the corresponding homonymous retinal halves of both eyes will be entirely destroyed, whereas from the opposite retinal halves the reflex will remain normal. To this peculiar condition Wernicke gave the name *hemiopic pupillary reaction;* but v. Leyden quite properly substi-

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tuted for this expression the term *hemiopic pupillary immobility*, because, as a matter of fact, when this condition appears, what is observed is certainly an immobility or rigidity. In order to demonstrate this rigidity it is essential, while watching the pupillary condition, that each retinal half be lighted separately.

The ordinary procedure consists in projecting a cone of rays into the eye by means of an ophthalmoscope or of a focusing lens, and observing the reaction of the pupils, according as the light impinges upon the left or upon the right retinal half. A darkened room is, of course, very desirable, if not essential. The difficulty in this depends upon the uncertainty of confining the illumination to one of the retinal halves. If the actual apex of the cone of light does not impinge upon the retina, anterior or posterior circles—radiations—of the light picture may affect the other retinal half or even the macula, and so spoil the test. According to Salomonsohn,[1] the best result can be obtained by reflecting from a concave mirror, placed horizontally beside the eye, a very sharp and vertical flame picture (the latter is collected by the mirror from an illumination situated at one side). Then, by gently rotating the mirror, the reflected light winds around the pupillary edge and impinges upon the retina, at one time from the temporal, at another from the nasal side. It is essential that the patient should always focus at the same distance, preferably into space. On account of the difficulties mentioned above (the dispersed circles of light), the results from this method are oftentimes not very conclusive; hence the following modification may be attempted: A black screen, at least a meter square and with a central opening, is placed vertically in front of the patient in the dark room, at a distance of about 60 cm. (about 2 feet). While the patient focuses upon the opening, an assistant in front of the screen projects a very bright illumination (kerosene lamp) into the visual field, at one time from the right and another from the left side, but not near the focusing point of the patient, and then the behavior of the pupils is noted through the opening in the screen. The lamp should always be held at about the same distance from the eyes as a focusing point, in order to be sure that a sharp flame and not dispersed circles are projected upon the retina; with this method the test can be made as well with one eye as with two. If the pupils of both eyes are contracted only when illumination is at the right side, or only when it is at the left side, right or left hemiopic homonymous pupillary rigidity is present.[2]

The difficulty of both these tests consists in the fact that ordinarily a stimulation of the peripheral portions of the retina by a circumscribed light picture excites only a weak pupillary reaction unless the region of the macula is actually stimulated as well. This objection applies particularly when the sensitiveness of the retina has been diffusely impaired,—*e. g.*, with a choked disk. In such cases the following device may be helpful. Place in front of the patient a screen which rotates upon a sagittal axis, and one-half of which is white, the other half black; then instruct the patient to focus upon a small mark in the half of the screen just at the edge of the border between the black and white, and illuminate the screen very brilliantly by means of a light placed behind the patient's head. Now, if the patient opens and shuts his eyes, hemiopic pupillary rigidity can be demonstrated by the pupillary reaction occurring only when the white side of the screen corresponds to the normal retinal half. This method may be employed for one as well as for both eyes.

In a case of acromegaly where even this device left the author in doubt, he employed the following method with considerable success, and can recommend it as being the best he has thus far used. While the patient focuses upon some object—*e. g.*, his own finger—at a distance of about 30 cm. (10 in.), an assistant

[1] *Deutsch. med. Woch.*, 1900, No. 42.

[2] In the more unusual temporal form of hemiopia, which characterizes tumors in the neighborhood of the chiasm (acromegaly), and hydrocephalus (in which the chiasm is compressed by the enlarged infundibulum), we are compelled to examine for hemiopic pupillary rigidity in the corresponding inner retinal halves (see Fig. 328, lesion *b*). But as a matter of fact the demonstration of hemiopic rigidity has no local diagnostic significance in this form of hemiopia, as it can arise only in the chiasm.

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projects in front of his eyes an electric-bulb light of at least 32-candle power, and stops just before the middle of the visual field; this light must be held so that the plane in which the carbon thread is bent remains sagittal to the eye examined. With hemiopic rigidity the pupillary reaction can be obtained only from the retinal half whose pupillary fibers are preserved. The advantage of employing such an illumination is that by virtue of the linear character of the source of light it remains localized enough, even despite the presence of dispersed rays, to illuminate only one retinal half so long as the lamp does not approach too near the focused point. The great intensity of the illumination is an additional advantage.

The best method of demonstrating hemiopic pupillary immobility is by means of the "pupil tester," described by v. Fragstein and Kempner,[1] which the author has only recently had an opportunity to employ. This is a tubular instrument in which the rays from an incandescent lamp of 8 volts tension are condensed by lenses and diaphragms to a slender but most intense cone of light, the focus of which is situated 4 cm. in front of the anterior extremity of the instrument. With this cone of light it is easy to illuminate either half of the retina by allowing it to pass through the pupil from the temporal or the nasal side. Since the slightly convergent rays cross or become divergent 4 cm. in front of the instrument, an intense and quite circumscribed illumination of an individual portion of the retina is best assured by holding the instrument 3.3 cm. in front of the cornea, so that the focus of the cone of light falls about 7 mm. behind the convexity of the cornea. This situation corresponds to the nodal point of Listing's reduced eye, and rays passing into the eye and directed toward this point are peculiar in that they pursue their original direction through the vitreous humor as though they had not been refracted. With this position of the instrument the cone of light passes into the eye as though no refracting media were present—*i. e.*, the slightly convergent rays continue their original course into the interior of the eye as a slender, slightly divergent cone. This instrument is probably the only one with which it is possible to illuminate still more circumscribed retinal areas, such as a particular quadrant.

The demonstration of hemiopic pupillary rigidity permits us to differentiate a so-called peripheral from a central homonymous hemiopia. While, according to Fig. 328, peripheral hemiopia (location of the lesion at *c*) does not produce hemiopic pupillary rigidity, this is not the case with central hemiopia (location of the lesion at *e*, above the primary optic centers Fig. 315). The hemiopic pupillary rigidity in which a double peripheral blindness occurs, composed of two hemiopias, one side of which is peripheral and the other central, is of special interest and has often been observed. An individual with these two lesions is completely blind. The accurate localization and significance of the visual disturbance is then furnished by the presence of hemiopic pupillary rigidity in the pupil which reacts only to one retinal half.

In those cases where hemiopic rigidity is present both a direct and crossed pupillary reaction can be obtained by testing the pupils in the ordinary way, so that both retinal halves receive light. The reaction may be diminished, but it is still plain enough. In order to avoid overlooking hemiopic rigidity the tests mentioned above must be accurately made, especially in all cases of homonymous hemiopic and bilateral blindness following a cerebral affection. Bechterew has made it clear (Fig. 328, lesion *f*) that homonymous pupillary rigidity may occur without any visual disturbance.

Certain other hypotheses in reference to hemiopic pupillary immobility have been given in the tabulation of the various lesions of the pupillary reflex upon p. 842 *et seq.*

The technical difficulties described above in determining homonymous pupillary rigidity are perhaps the reason that the phenomenon has been so rarely found.

An additional difficulty is furnished by disturbances of Haab's so-called cor-

[1] *Klin. Monats. für Augenheilk.*, 1899.

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tical pupillary reflex, which is brought about by concentration of the attention upon a lateral source of light (see p. 847). All of the procedures for testing hemiopic pupillary immobility are of such a character that the attention of the patient is apt to concentrate upon the source. But this is, of course, the case only when the light falls on the seeing retinal half, and, therefore, if Haab's reflex is lacking, hemiopic pupil rigidity may apparently be present when the blind retinal half is lighted.

Loss of Pupillary Light Reaction (Pupil then Ordinarily Dilated); Rigidity of the Pupil to Light.—A failure of the pupil to react to light occurs in severe disturbances of consciousness of the most varying kinds: In cerebral pressure (in these cases bilateral); in poisoning from the substances mentioned above as dilating the pupils (these unilateral or bilateral, depending upon the poison); in focal lesions which interrupt the reflex pupillary arc according to the method detailed in Fig. 328 (e. g., *motor lesions*); in complete peripheral oculomotor paralysis; in nuclear oculomotor paralysis which affects the iris nucleus, but does not attack the other branches of the oculomotor (see p. 833); in *sensory lesions;* in affections of the retina; and in bilateral optic atrophy or marked bilateral choked disk. Complete loss of pupillary reaction from choked disk is a comparatively exceptional occurrence, and observed only when the choked disk has caused at the same time blindness. The visual acuteness is frequently very well preserved, and corresponds well with the slight injury to the conduction of the sensory fibers belonging to the pupillary reflex. Even when a lesion of the optic nerve diminishes the acuteness of vision decidedly, the pupillary reaction need not be seriously impaired. This is evident without further explanation when we remember (p. 841) that the centripetal fibers of the light reflex in the optic nerve are distinct from the visual fibers. A unilateral loss of reaction is usually associated with a dilatation of that pupil, as compared with the opposite side.

Ordinarily an exact examination of the condition of the optic nerve, on the one hand, and of the nerves supplying the eye muscles, on the other hand, facilitates a differential diagnosis between pupillary rigidity depending upon a lesion of the motor and that depending upon a lesion of the sensory limb of the reflex arc. Fig. 328 should always be consulted to comprehend the diagnostic points. In addition to other characteristics, it should be emphasized that with lesions of the motor limb of the pupillary reflex (paralysis from the muscles of the nucleus of the iris or from the peripheral oculomotor), the narrowing of the pupil to accommodation and the power of convergence (see below) is lost; whereas this is not the case when the loss of pupillary reaction depends upon a lesion of the retina or of the optic tract. In case the lesion is recognized as motor, the question is: Does this lie in the nucleus or its immediate neighborhood, or in the peripheral motor fibers of the oculomotor? This question is frequently decided by the behavior of the ciliary muscle—*i. e.*, by accommodation. In lesions of the nucleus or its neighborhood, accommodation may be preserved on account of the arrangement of the oculomotor nucleus (discussed upon p. 833); whereas with a purely peripheral oculomotor paralysis, which may be situated in the trunk of the nerve or in the short branches of the ciliary ganglion, accommodation will probably fail.

Argyll-Robertson's Phenomenon.—This is an early symptom of tabes dorsalis and of progressive paralysis. It consists in a preservation of the pupillary reaction to convergence and to accommodation with a loss of the reaction of the pupil to light, and is not associated with any impairment of visual acuteness (see under γ). For some unknown cause the pupils are generally narrowed (the so-called spinal myosis of tabetics).[1] Ordinarily the reaction of the pupils to pain is also wanting. Erb called Argyll-Robertson's phenomenon a *reflex pupillary rigidity*, an expression in which the words, according to the writer's opinion, are not used in their ordinary sense, since from it one might conceive of a rigidity occasioned reflexly, not of a rigidity of the reflex. The symptom has not yet been satisfactorily explained by anatomic researches. According to Bechterew,

[1] It has not yet been proved that this is of spinal origin.

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Fig. 328, however, it must theoretically depend upon a lesion of all the centripetal fibers of the pupillary light reflex, after their separation from the visual fibers (lesion *d*, Fig. 328). It cannot depend upon a lesion of the motor limb (lesion *g* or *h*, Fig. 328), because the pupillary contraction in accommodation and convergence persists.

Paradoxic Pupillary Reaction.—This phenomenon, first described by Obersteiner and then by Bechterew, consists in the occurrence of a dilatation instead of a contraction of the pupils to direct and crossed illumination. At times a very insignificant initial contraction precedes the dilatation. Like the Argyll-Robertson phenomenon, this sign occurs principally in *tabes dorsalis* and *progressive paralysis.*

According to one conception, probably erroneous, this dilatation depends upon unnoticed movements of divergence of the eyeball at the moment of illumination (as contrasted with the narrowing of the pupils, associated with convergence and accommodation, see below, γ). Bechterew, however, considers that it is a phenomenon of fatigue, because, under the pathologic conditions mentioned above, a brilliant illumination fatigues, and so inhibits, the pupillary tonus after a scarcely noticeable or absent narrowing.

β. **Pupillary Pain Reflex.**—Severe pain stimulus applied to various parts of the body, but more especially painful irritation of the skin of the neck, will usually dilate the pupils. This dilatation is produced by the pupillary dilator fibers derived from the sympathetic, and in turn from the VIII. cervical and I. dorsal segments (see Fig. 366, p. 926). The reflex is sometimes useful in diagnosis, pointing to an involvement of the roots or of the sympathetic. According to the author's experience, however, the pupillary pain reflex is so inconstant that, generally speaking, a difference between the two sides is the only sign worth heeding.

γ. **Narrowing of the Pupils to Convergence and to Accommodation.**—Physiologically the pupils are decidedly contracted by efforts at convergence or accommodation (it is difficult to separate one from the other). This consensual movement is diagnostically significant; in the first place, because it shows that in testing the other pupillary reactions we must be careful to have the patient avoid convergence or accommodation. Perhaps the best plan is to have him always focus for the same distance, preferably into space. A further diagnostic interest is furnished by the retention of the convergence and the accommodation reflex while the light reflex is absent, which proves that the light reflex is not affected by a lesion of the motor tract.

(See above: Loss of Pupillary Reaction to Light, p. 846; Argyll-Robertson's Phenomenon, p. 846.)

δ. **Westphal's Pupillary Phenomenon.**[1]—This consists in a contraction of the pupil when the examiner, by forcibly holding the lid open, prevents the patient's attempt to close the eye (Bell's phenomenon, p. 863). To appreciate this reaction it is generally essential that the pupil in question should not react to light, or, at least, only slightly, and should not be markedly narrowed. It is easier to recognize the appearance with dilated pupils. Westphal never found the phenomenon in healthy individuals, and only once in the pupils of an hysteric person. He found it several times, on the contrary, in tabes, and in progressive paralysis. The author has repeatedly observed it in tabes.

ε. **Haab's So-called Cortical Pupillary Reflex.**—Thus far no diagnostic significance has been attached to this reflex, though perhaps it may yet be utilized in the diagnosis of cortical disturbances of vision. It consists of a narrowing of the pupils when the patient in a dark room, without any alteration in the position of the eye, concentrates his attention upon a flame placed at one side —*i. e.*, seen indirectly.

[1] *Neurologisches Centralbl.*, 1899, No. 4.

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9. Condition of the Accommodation.

Before testing the accommodation, the acuteness of vision must first be determined and any error of refraction corrected. To correct the latter, if the patient is myopic, we employ the weakest concave lens with which he sees distant objects clearly; if hypermetropic, the strongest convex lens. The acuteness of vision is then determined in the ordinary way. The accommodation is then tested by placing before the eye under examination, at a distance of 25 cm. in a good light, the finest test type which he should be able to read at this distance with his acuteness of vision. (Perfect accommodative power presupposed.) If he can read this type the accommodation is, at least, normal. If he cannot read it there must be defective accommodation. Such a defect will consist of the physiologic presbyopia of his age plus whatever accommodative paresis may exist. The convex lens which the patient requires for reading the selected type at 25 cm. distance (plus the glass which corrects the refraction) measures the defect of accommodation in dioptries. By comparing this defect with that which is found in a patient as a result of the physiologic presbyopia for his age (see the accompanying table), we can decide whether there is a paresis or paralysis of accommodation. Should the patient need a + 4 D. lens to read the type clearly, there exists no power of accommodation.[1] Such a total defect is physiologic after the age of seventy-five years (see table); but in younger individuals it would indicate a complete pathologic paralysis of accommodation. If a patient forty-five years old requires a glass of + 2 D. he has, in addition to his physiologic presbyopia (0.5 D., according to the table), a paralysis of accommodation of 1.5 D., etc.

Age.	Degree of presbyopia—*i. e.*, the physiologic defect of accommodation in dioptries.
Forty-five years	0.50 D.
Fifty "	1.50 D.
Fifty-five "	2.25 D.
Sixty "	3.00 D.
Sixty-five "	3.25 D.
Seventy "	3.75 D.
Seventy-five "	3.75 D.

We find a paralysis of accommodation in total oculomotor paralysis, in lesions of the *accommodation nucleus* (p. 833), and also in *diphtheric paralysis.* Despite the fact that many of the last-named (diphtheric) paralyses are probably peripheral in their localization, they are quite apt to involve the fibers of the oculomotor nerve which supply the pupil and the ciliary muscle.

[1] For the total optical effort required equals that which is necessary to focus upon the retina rays which come from a distance of 25 cm., in an eye rendered emmetropic by the correction of its refraction. This takes place when the convex lens in question renders parallel the rays which enter the eye from that distance. A lens of + 1 D. renders parallel rays which come from a distance of 1 m., a lens of + 4 D. renders parallel rays which come from one-fourth of that distance—viz., 25 cm.

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V. CRANIAL NERVE; TRIGEMINUS.

1. **Motor Trigeminus.**—The motor branch of the trigeminus supplies the muscles of mastication. Their power is tested by having the patient bite upon some object, such as a piece of cork or wood, or by having him exert vigorous movements of the jaw while the examiner attempts to hold the jaw still.

In connection with the action of the separate chewing muscles, we must remember that adduction of the lower jaw—*i. e.*, the closure of the teeth—is accomplished essentially by the temporal and masseter muscles. The external pterygoid pushes the lower jaw obliquely forward out of the glenoid fossa upon the articular tubercle (Gegenbauer). The bilateral action of the two external pterygoids protrudes the lower in front of the upper teeth. The unilateral action of one external pterygoid pushes the jaw to the opposite side, and the alternate action of the two external pterygoids—presupposing, of course, that the temporal muscle pulls the lower jaw back again into the glenoid fossa—comprises the movement of mastication. The external pterygoid also aids in opening the mouth, and this is further assisted by the force of gravity, and by the digastric [1] and the platysma myoides muscles.[2] The internal pterygoid muscle aids the temporal and the masseter in adducting the lower jaw, and also contributes, to a certain extent, in the movement of the jaw forward.

Cerebral *paralyses of the chewing muscles* may be accurately compared to cerebral paralyses of the eye muscles. They are always to be attributed to a cause lying in the neighborhood of the trigeminus nucleus or affecting the efferent trigeminus fibers. This depends upon the fact that above the nucleus the central fibers of each trigeminus are distributed to both hemispheres; in consequence of which a unilateral hemispheric lesion does not necessitate a crossed motor trigeminal paralysis, as the function of the intact hemisphere is sufficient to care for the innervation of both sides. To comprehend this bilateral innervation, the reader should compare Fig. 318, for that diagram applies to the muscles of mastication in the same way as to the eye muscles. On the contrary, a bilateral paralysis of the chewing muscles can be brought about by bilateral hemispheric lesions, such as pseudo-bulbar paralyses (p. 876). In hemiplegia a bilateral defect of innervation of the chewing muscles may perhaps be demonstrated dynamometrically.

Spasms of the chewing muscles occur as accompanying phenomena of general spasms, as the tonic spasms in tetanus and in meningitis, and reflexly from painful affections of the jaw, producing so-called lockjaw.

The so-called *jaw reflex* depends upon both the motor and the sensory trigeminus. It consists in a contraction of the chewing muscles which lifts the lower jaw, and is produced by striking the lower jaw, either directly with a percussion hammer or indirectly through some object applied to the jaw. It can be elicited in most healthy individuals, but is not absolutely constant. If the reflex is increased, a clonus can frequently be elicited by simply drawing the jaw downward (jaw-clonus, masseter-clonus).

2. **Sensory Trigeminus.**—The sensory division of the trigeminus supplies the skin of the face, the mucous membranes of the mouth and nasal cavities, the conjunctiva, and the cornea. It also takes part

[1] The anterior belly is innervated by the third branch of the trigeminus; the posterior belly, by the facial.

[2] Innervated by the facial.

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in the function of taste (chorda tympani), and the function of smell in the nasal mucous membranes. The sensibility of the skin (touch, pressure, pain, and temperature) is tested just as was described above (p. 756 *et seq.*). In testing the trigeminus taste function, a soft brush moistened first with acid (weak acetic acid) and afterward with salt solution is touched to the tongue, and the patient asked to describe what sense of taste he experiences. The two sides of the tongue are compared, and it is readily determined whether the appreciation of taste is equally active and equally quick upon the two sides. Since the trigeminus practically supplies only the anterior part of the tongue, the test should be made there, so as to exclude, so far as possible, the taste fibers of the glossopharyngeal. The patient should keep his tongue protruded, and reply to the examiner's questions by nodding and shaking his head. It is, of course, advisable to include at the same time the examination of the sense of taste of the part supplied by the glossopharyngeal, employing the same method as above, but selecting the posterior part of the tongue. The patient should not breathe during the test, so as to avoid a confusion between the sense of smell and that of taste. The taste fibers of the trigeminus (chorda tympani) may be injured in lesions of the lingual muscle (which they supply), in affections of the middle ear (through which they pass), in certain peripheral paralyses of the facial (see pp. 858 and 860), and finally in lesions of the root of the second or, according to other authorities, of the third branch of the trigeminus, in which the taste fibers are diverted from the facial (see Fig. 337). Testing the sense of smell supplied by the trigeminus has been described already under the olfactory nerve (see p. 821).

The *corneal sensibility* is determined by touching the cornea with the head of a pin. Normally this procedure is very painful, owing to the fact that, according to v. Frey's researches, the cornea possesses no tactile points, but merely countless pain points (see p. 760 *et seq.*). At the same time we should determine the preservation or absence of the so-called "corneal reflex" (lid-closure in touching the cornea). The loss of this may depend upon a lesion of the sensory limb (trigeminus), or upon a lesion of the motor limb (facial) of the reflex arc. Further examination will determine which.

Pareses of the sensory trigeminus occur in peripheral lesions of the nerve, and also accompany the hemianesthesias observed both in hysteria and in focal lesions at the most posterior part of the internal capsule. Disturbances of sensibility which appear in the region supplied by the trigeminus from spinal cord affections involving the ascending (spinal) trigeminus roots are worth noting because of their diagnostic importance. They may occur as low as the second cervical segments (syringomyelia).

(Compare Fig. 356, p. 915, in regard to the distribution of the peripheral skin branches of the trigeminus.)

VII. CRANIAL NERVE: FACIAL.

The facial nerve, probably purely motor, supplies the facial muscles, including the muscle which closes the eye (orbicularis oculi), Horner's tear sac muscle, the

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platysma myoides, the muscles of the skull cap (occipitalis and frontalis), the retrahens, attollens, and transversus auriculæ,[1] the stylohyoid, the posterior belly of the digastric; further, in connection with the third branch of the trigeminus, the buccinator, and finally, by means of the descending palatine nerves, which pass through the sphenopalatine ganglion of the second branch of the trigeminus, it supplies, together with the glossopharyngeus, vagus, and accessory, the muscles of the soft palate. The facial takes the lion's share in the nerve supply of the latter. The palatoglossal and palatopharyngeal muscles (palatine arch and the azygos uvulæ) seem to be supplied mostly by the facial. In the Fallopian canal the facial nerve supplies the stapedius muscle by means of the stapedius nerves. At one part of its course in the temporal bone the chorda tympani is united to the facial nerve, and so brings the taste fibers to the facial, and from it receives the salivary secretory fibers for the submaxillary and sublingual glands (see p. 858). From its motor nucleus the facial receives fibers for sweat secretion, and, according to Goldzieher, even the secretory fibers for the lacrimal glands. These, Köster[2] thinks, probably start from the nuclear region of the glossopharyngeal. At the periphery the facial oftentimes receives sensory fibers of the trigeminus.

(*a*) Paralyses of the Facial.

General Symptomatology of Facial Paralyses.—Upon the paralyzed side in facial paralysis we notice an obliteration of the wrinkles, a loss of the ordinary voluntary movements or their very slight preservation, and under some circumstances even the loss of the emotional movements, the associated movements, and the reflexes. If the paralysis is sufficiently marked, the affected cheek flaps with respiration like a boat's sail, especially during sleep, and if the branches to the eye are affected, the eye remains more or less open, despite attempts to close it, and even during sleep (*lagophthalmus*). In fresh cases the mouth is drawn to the healthy side. If the palate is also paralyzed, it frequently hangs noticeably lower upon the diseased side and seems to crowd to the healthy side, and voluntary or reflex innervation pushes it still more toward the latter. The depression of the uvula so often occurs normally that it is of no importance for the diagnosis of palate paralysis. A unilateral palate paralysis will produce neither a nasal character in the voice nor regurgitation through the nose; but both phenomena will be observed if the palate is bilaterally paralyzed (diphtheric paralyses). If the eye is prevented from closing by an accompanying involvement of the ocular branch of the facial, the normal tear secretion will be affected by a paralysis of Horner's tear-sac muscle; a drooping of the lower eyelid will result and the patients will suffer from a trickling of tears (epiphora), and very likely later from eczematous affections of the eyelid. They may also complain of slight disturbances of vision on account of the corneal suffusion. Blepharoplegia (automatic winking) is exceptional even in complete facial paralysis (including the orbicularis oculi). This signifies that blepharoplegia depends not merely upon facial innervation, but also upon atony of the levator palpebræ superioris. In consequence of the disturbances of tear secretion the nasal mucous membranes become drier than normal, and

[1] According to Heitzmann, the attrahens auriculæ is supplied by the auriculo-temporal branch of the third branch of the trigeminus.

[2] *Arch. f. klin. Med.*, vol. lxviii.

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the sense of smell is, therefore, often affected. The nasal opening of the diseased side often seems narrowed from paralysis of the levator alæ nasi. Because of the paralysis of the muscles about the lips, the saliva frequently trickles from the corner of the mouth upon the paralyzed side and patients are no longer able to whistle. With pronounced facial paralysis the speech may be altered, especially the ability to articulate the labial letters. Statements vary considerably in regard to the condition of the tongue in facial paralyses. Probably facial paralysis as such has no influence upon the position of the tongue, although, since the facial nerve innervates the stylohyoid and the posterior belly of the digastric muscle, its paralysis may have a certain influence upon the position of the hyoid bone. In central facial paralysis the tongue deviates toward the paralyzed side, because here the hypoglossal territory anatomically situated so near by is always more or less affected as well. The deflection of the tongue in central facial paralysis is, therefore, the result of a paresis of the genioglossus muscle, which is supplied by the hypoglossal nerve (see XII. Cranial Nerve, p. 875). Deflections of the tongue occur in peripheral facial paralysis, but these have only an indirect connection with the facial nerve and their explanation is not the same in every case. For instance, in peripheral paralysis upon first glance one would think that the tongue was protruded obliquely, but more careful observation shows that it is really protruded in the middle line, the appearance of deviation being due to the twisted mouth. In other cases the tongue is actually deflected to the healthy side, which is exactly opposite to the condition in central facial paralysis accompanying genioglossus paralysis. Hitzig has proved that this depends upon the patient's effort to protrude his tongue obliquely in order to keep it in the middle of the twisted mouth. This can be confirmed easily by correcting manually the assymetry of the mouth. The patient then protrudes the tongue exactly in the middle. Paralysis of the platysma myoides is most readily recognized by having the patient attempt to project the lower lip upwards as far as possible. Normally the platysma assists in this movement, and the contracted fibers stand out quite plainly under the skin of the neck, so that the contrast between the healthy and paralyzed side can be readily recognized. A paralysis of the muscles of the ear (retrahens, attollens, and transversus auriculæ) and of the frontalis and occipitalis muscles can be readily recognized, but only in those patients who can move the ears and the scalp voluntarily. Yet, sometimes a paralysis of the ear muscles of one side causes a noticeable drooping of that ear.

The clinical picture of facial paralysis varies, moreover, according to the position of the paralysis, whether above the nucleus—*i. e.*, in the central neuron, or in or below the nucleus—*i. e.*, in the peripheral neuron. It becomes necessary, therefore, in the following discussion, to differentiate accurately the symptomatology of these two types of facial paralysis, and to defer the discussion of the affections of secretion and taste as well as certain accompanying troubles with the hearing, until the peripheral type (the one which they accompany) is taken up.

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Central (Supranuclear) Facial Paralysis.—As is well known, the cortical center of the facial nerve lies at the foot of the central convolution, and from this point its fibers pass intermingled with the other pyramidal fibers through the internal capsule to the facial nucleus of the opposite side. In its course it is quite frequently injured (cerebral hemiplegia). Any lesion situated above the nucleus causes what we designate as central facial paralysis, involving the muscles of expression of the lower half of the face, and the corresponding half of the palate upon the affected side, whereas the secretory and taste functions of the facial are not affected at all, because the fibers supplying them do not

FIG. 329.—Facial asymmetry simulating hemiplegia: The distorted nose and a groove in the lower front teeth are responsible for the apparent deviation of patient's tongue to the right (New York City Hospital).

join the facial nerve until it reaches the periphery (see Nuclear or Peripheral Facial Paralysis). Nor does this paralysis include the upper branch of the facial, which supplies the muscles for closing the eyes and the forehead muscles, for reasons about to be mentioned. In regard to the condition of the tongue compare above, p. 852.

The most weighty factor in differentiating between a central[1] and a peripheral paralysis is the non-involvement of the upper facial branch (for the forehead and eyes) in the former. This peculiarity is to be

[1] "Central" and "cerebral" should not be confused. A subnuclear or peripheral facial paralysis may be located within the brain—*i. e.*, still be cerebral, although it is below the nucleus and therefore not central, but peripheral.

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explained by the supposition that only the lower part of the facial (the branch to the face) possesses an actual crossed innervation from the cerebral cortex, whereas, the upper branch, like the eye muscles (see p. 833) and the motor trigeminus, is equally innervated or nearly so by both hemispheres, so that any defect of innervation from one hemisphere is concealed or compensated for by the activity of the other.

A diagram of the central facial innervation made in accordance with this supposition is given in Fig. 330. This clearly explains how a unilateral cerebral lesion at (a) paralyzes only the lower, not the upper

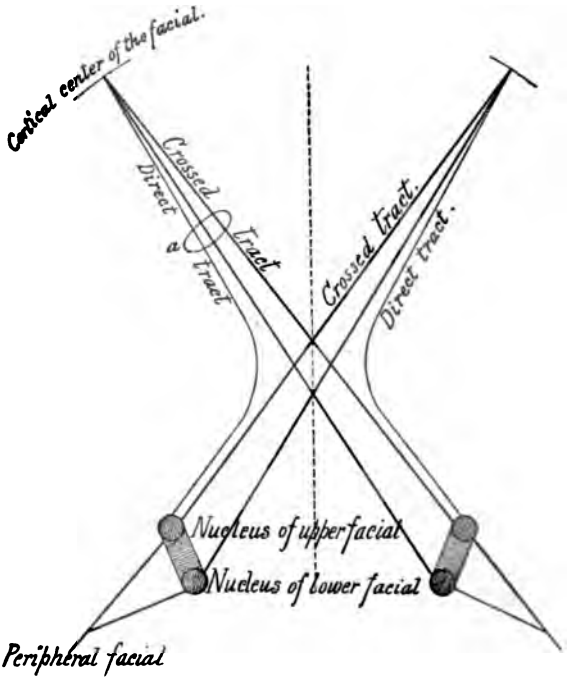

FIG. 330.—Diagram of the central innervation of the facial nerve. The upper branch is supplied from both hemispheres, more from that of the opposite side. The lower branch is supplied almost exclusively from the opposite hemisphere.

facial of the opposite side, since the latter is still sufficiently innervated by the uncrossed tract.

The hypothesis responsible for Fig. 330, viz., that the facial possesses a separate nucleus for its lower and another for its upper branch, is really not based upon anatomic proof. But experience shows that a bulbar paralysis, a disease of the motor nuclei of the oblongata, affects almost exclusively the lower facial, which makes it probable that the nucleus of the latter exists functionally isolated, even though it is impossible to demonstrate this macroscopically. We have attempted to express this individuality as simply as possible in Fig. 330, by representing the two facial nuclei as connected.

It is, moreover, incorrect to assume that in a central paralysis the

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upper facial escapes entirely, for a more minute examination shows that a slight weakness can ordinarily be demonstrated. This consists in a less vigorous closure of the eye upon the affected side, and in the patient's inability to close the eye of the paralyzed side alone, although before the injury this was possible (*Signe de l'orbiculaire*, Revilliod). From this it is evident that both hemispheres influence the upper facial, but that the effect of the crossed fibers outweighs that of uncrossed, although perhaps only to a slight extent. Fig. 330 suggests this by the heavier line representing the crossed fibers of the upper facial.

Probably the central innervation of the stapedius muscle, which plays a rôle in the symptomatology of peripheral facial paralysis, behaves in the same way as that of the upper facial, so that in consequence of the bilateral innervation of the stapedius the symptom of hyperacusis (p. 858) cannot be attributed to central facial paralysis.

In addition to the escape of the upper facial innervation, a central facial paralysis is further differentiated from a peripheral paralysis by the way in which certain voluntary movements, the emotional and the reflex, take part in the paralysis. To explain this point we must assume that tracts which are separated at least for a part of their course are responsible for each different kind of movement. Fig. 331 will serve as an illustration.

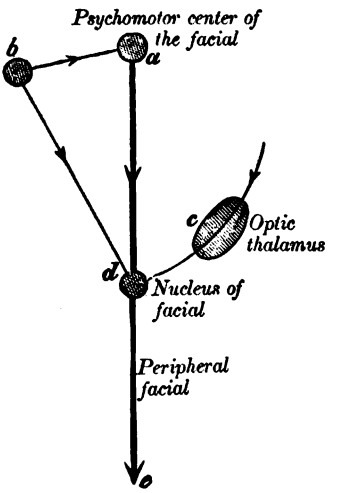

FIG. 331.—Diagram of the three functionally different central paths of the facial nerve and of its reflex arc.

(*a*) Represents the psychomotor center of the facial, (*b*) represents another psychomotor center—(*e. g.*, the arm-center), (*c*) the optic thalamus, (*d*) the facial nucleus.

The tract (*ade*) represents the voluntary facial tract.

It has been assumed oftentimes that associated movements—*e. g.*, grimaces accompanying movements of an arm—depend upon hypothetical tracts like *bd*. To prove their existence it has been argued that a slight central facial paralysis is sometimes evidenced only by a weakness of the associated movements, as compared with those of the healthy side, thus assuming the injury of a tract (*bd*) while the actual facial tract (*ad*) is unaffected. But this manifestation of a weakness of the associated movements in the facial territory in certain cerebral facial paralyses can also be explained in another way. The associated movements can be assumed to pass along the tract (*bad*). Then with a slight lesion of the voluntary tract (*ad*) the voluntary impulse suffers no appreciable opposition, but the weaker associated impulse running over (*bad*) does exhibit a defect. The observation that a similar effect has been noted in mild cases of peripheral paralysis argues for this explanation. Hence it seems to the author superfluous to imagine

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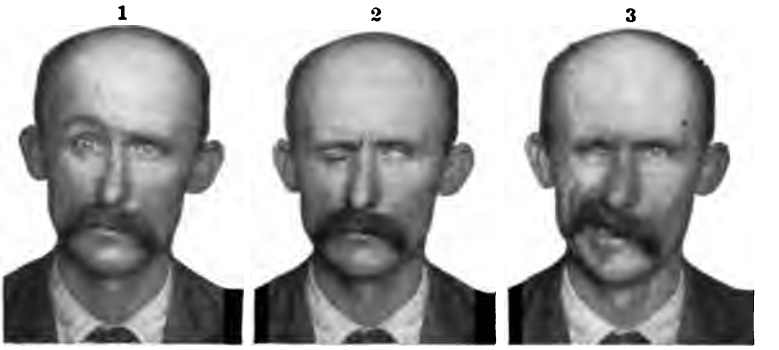

FIG. 332.—Facial palsy of left side: 1, Bilateral attempt to raise eyebrows; 2, bilateral attempt to close eyes; 3, smiling (Church).

the existence of tracts like (*bd*) in order to explain associated movements.

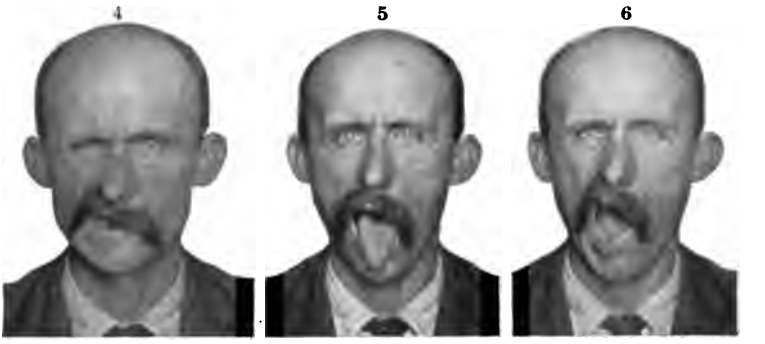

FIG. 333.—Facial palsy of left side: 4, Forced effort to uncover teeth on both sides; 5, protruded tongue in middle line; 6, mouth open more widely on sound side in bilateral effort (Church).

The facial innervation of mimicry is probably from the tract (*ad*) through the optic thalamus. This is determined from experience in diseases of the optic thalamus or of its neighborhood, which cause

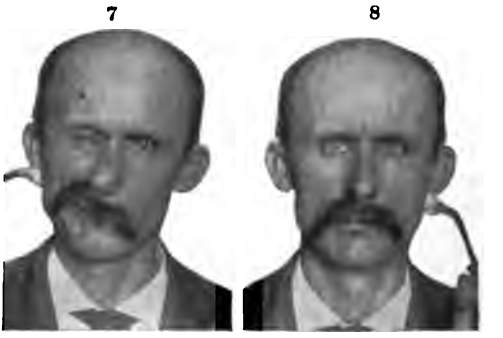

FIG. 334.—Facial palsy of left side: 7, Effect of faradism on sound side; 8, non-effect of same current on paralytic side (Church).

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isolated paralysis of the facial mimetic movements. Moreover, if the thalamus region is intact, ordinarily no, or only a very slight, involvement of the mimetic movements occurs in facial paralysis.

Besides the relative freedom from involvement of the upper branch, a central facial paralysis is characterized by intact reflexes and by the fact that the actual voluntary movements and the spontaneous movements need not be affected to the same degree, whereas without question in lesions of the peripheral neurons situated somewhere along the line (*de*) (*i. e.*, in nucleoperipheral paralysis), every single function, together with the reflexes, appears to be equally affected.[1]

In order to differentiate a central from a nucleoperipheral facial paralysis, it is obviously necessary to distinguish separately the different kinds of facial movements, to carry out the electric examination in accordance with the rules mentioned upon p. 798 *et seq.*, and to determine the presence or absence of degenerative atrophy of the paralyzed

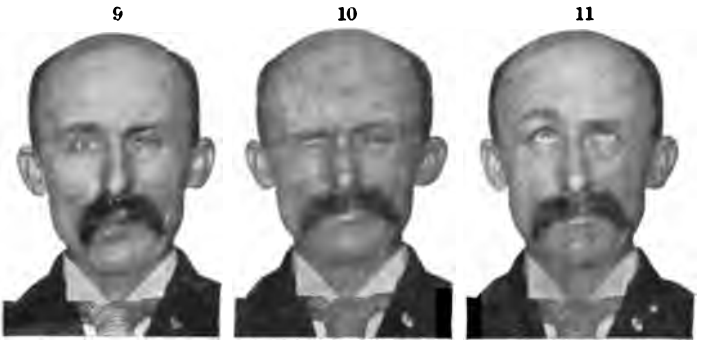

FIG. 335.—Same case six months later: 9 Shows late contracture on the paretic side while the face is at rest; 10, contracture in the lower half of the face increased by gently closing the eyes, and at the same time shows weakness about left eye; 11, contracture increased by raising brows, showing overaction of zygomatici and weakness of frontalis on left side (Church).

muscles in accordance with p. 789 *et seq.* Of course neither atrophy of the facial muscles nor electric changes occur in central facial paralysis.

Nucleoperipheral Facial Paralysis.—The general symptomatology of facial paralysis mentioned above (p. 851 *et seq.*) must be amplified in the case of nucleoperipheral paralysis by the description of certain other disturbances of function peculiar to this type.

As most important may be mentioned the paralysis of the stapedius muscle. The motor fibers for the supply of this muscle leave the facial nerve within the petrous bone in order to reach the tympanic cavity (see Fig. 337 p. 861). Although they probably also accompany the facial in its intracranial course, a stapedius paralysis is actually observed only in the peripheral form of facial paralysis, probably because the

[1] This is true, however, only for very decided and complete nucleoperipheral facial paralyses. In incomplete peripheral paresis, one frequently observes merely that the facial behaves differently for different kinds of movements—*e. g.*, that the paralysis is recognized only in laughing or in associated movements. Perhaps this can be explained by supposing that in the different kinds of movements (voluntary, spontaneous, associated and reflex movements) the impulses vary in their strength.

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stapedius muscle, like the forehead and eye branches of the facial, is innervated by both cerebral hemispheres (see p. 854). This paralysis of the stapedius, observed in certain localizations of peripheral facial palsy of which we shall speak later more fully, is sometimes manifested by a characteristic peculiarity of hearing, designated as hyperacusis. In this condition the patient hears the deep tones (and sometimes others) louder upon the paralyzed side, and oftentimes in a very annoying fashion; sometimes the sound impression is combined with a sensation of pain. Lucae has attributed this phenomenon to an increased tension of the tympanic membrane producing a rise of the labyrinth pressure, because the action of the tensor tympani (innervated by the trigeminus) can be no longer counteracted by its antagonist, the stapedius. This explanation coincides with the modern conception, introduced by Zimmermann, which assumes that the tympanic membrane, together with the chain of ear-bones and their muscular apparatus, serves much less for sound transmission than for a so-called accommodation of the acoustic organ to the different kinds of sound impressions, and for the protection of the internal ear against too intense sounds, associated with marked pressure variations in the labyrinth fluid. Patients with paralysis of the stapedius muscle also occasionally complain of experiencing subjective noises in the ear from movements of the facial or chewing muscles.

Other characteristic appearances arise in peripheral facial paralysis when the fibers of the *chorda tympani* are injured. These consist partly of disturbances in the sense of taste upon the anterior half of the tongue, partly of disturbances in the salivary secretion of the submaxillary and sublingual glands. Patients frequently complain of a defective sense of taste upon one side of the tongue, or of an abnormal dryness of the corresponding half of the mouth (see p. 850 *et seq.* for the more exact method of testing the sense of taste). To examine the chorda tympani's function of salivary secretion more accurately the following procedure is advisable: The patient opens his mouth and lifts the tip of his tongue so as to expose the openings of the ducts of the submaxillary and sublingual glands (ordinarily united at the sublingual caruncle). In case he is unable to do this, the tip of the tongue is held up by a pair of forceps such as are used in chloroform narcosis. The sublingual caruncle is then carefully dried upon both sides of the frenum with absorbent cotton, and while the examiner watches carefully the openings of the ducts, the patient inhales deeply the fumes from a sponge saturated with acetic acid, and held close to the nose. If the chorda functionates normally the saliva flows from both sides freely, as a result of the reflex; if the chorda is paralyzed upon one side the flow occurs only upon the healthy side or much more profusely upon that than upon the injured side.

The excretory ducts are occasionally so close together that the above method will not enable us to determine whether saliva comes from one or both openings. In these cases the author has succeeded in gently clamping the excretory duct upon the unaffected side with a cilia-forceps, after which it is easy to observe whether saliva escapes from the opposite opening.

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In connection with the anatomic relations of these two functions of the chorda to the facial, we should emphasize the fact that the function of secreting saliva belongs to the facial from its origin at the base of the brain up to the exit of the chorda tympani. According to Köster[1] it probably does not arise from the facial nucleus itself, but from the nuclear region of the portis intermedia Wrisbergi belonging to the glossopharyngeal. The taste fibers of the chorda tympani, on the contrary, accompany the facial from the periphery through the tympanic cavity, merely a tiny distance apart, and leave the latter again near the geniculate ganglion in the petrous bone (Fig. 337), according to one supposition (continuous blue line) in order to connect with the sphenopalatine ganglion by means of the superficial large petrosal nerve, and from there with the second branch of the trigeminus, but according to another less probable view (dotted blue line) in order to connect with the third branch of the trigeminus or with the glossopharyngeal by means of the communicating nerve to the tympanic plexus.

Our knowledge of the functions of the peripheral facial has been still further perfected by Goldzieher. He found that at the base of the brain the trunk of this nerve contains fibers for the secretion of tears, and that therefore an injury to the facial at this point results in drying up the tear secretions[2] upon the paralyzed side. These secretory fibers must emerge from the facial further down in the region of the geniculate ganglion and pass by means of the greater superficial petrosal nerve to the sphenopalatine ganglion and from there to the lacrimal glands by means of the communication between the subcutaneous malar and the lacrimal nerves. Goldzieher's suppositions have been confirmed by an observation of Franke,[3] as well as by the extensive clinical and experimental work of Köster. According to the latter the lacrimal secretory fibers, like the salivary secretory fibers, probably arise from that part of the glossopharyngeal nucleus belonging to the portio intermedia Wrisbergi, and mingle with the facial directly at its exit from the brain. To test the lacrimal secretion, he recommends tickling the nasal mucous membrane with a feather or with a fine brush, and then observing the secretion. The amount of the tear-flow secreted can be best estimated by collecting it upon a piece of filter paper introduced into the conjunctival sac.

Köster, in addition, showed that disturbances of the sweat secretion upon the paralyzed half of the face appear not uncommonly in peripheral facial paralysis. This, too, is to be attributed to the fact that from its nucleus the facial conducts fibers for the secretion of sweat. In peripheral facial paralysis, these fibers may be stimulated (hyperhidrosis) as well as paralyzed (anhidrosis). These phenomena may be confused with the influence of the sympathetic upon the sweat secretion of the face.

[1] *Deutsch. Arch. f. klin. Med.*, vol. lxviii., 1900.

[2] Only intermittent secretion from the tear glands, such as occurs in weeping or in reflex tears, not a continued conjunctival secretion.

[3] *Deutsch. med. Woch.*, 1895, p. 33.

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Slight disturbances of hearing which occur in lesions of the facial nerve in the neighborhood of the geniculate ganglion are worth mentioning; these depend upon the proximity of the internal ear to the facial nerve at this point (the cochlea is separated by a bone about ¼ mm. thick). Hence inflammatory changes of the facial can easily have an effect upon the internal ear.

The distinctions between nucleoperipheral—*i. e.*, facial paralysis in the region of the peripheral neurons—and central facial paralysis have

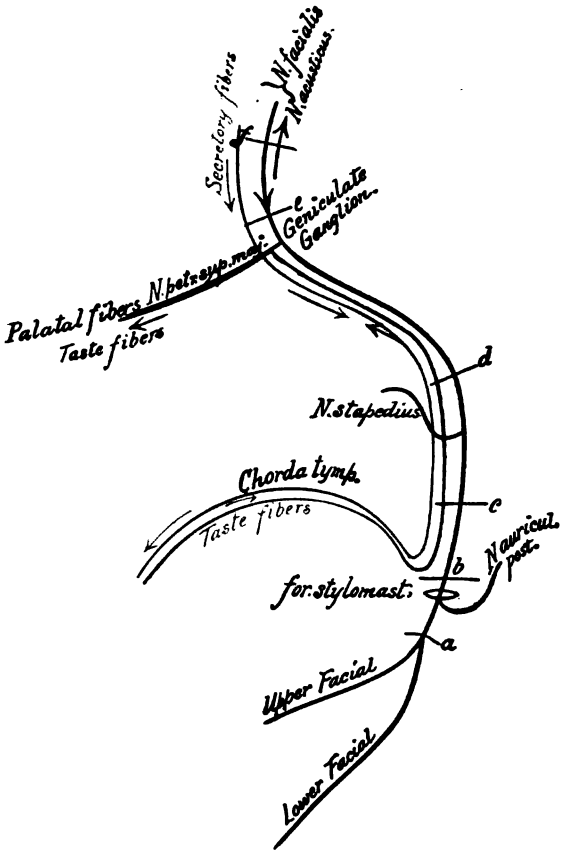

FIG. 336.—Diagram of the peripheral nerve fibers of the facial for clinical use.

already been emphasized in describing the latter (p. 853 *et seq.*). From this it is evident that the essential factors in the distinction are the definite paralysis of the forehead and eye branch of the facial in the nucleoperipheral forms, disturbances in the secretory and taste functions, the condition of the tongue (p. 852), the condition of the reflex and spontaneous movements (p. 855), and the electric and trophic conditions of the muscles. After the nucleoperipheral nature of a facial paralysis has once been recognized, the essential interest centers in

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determining the exact location of the lesion in the course of the nucleoperipheral tract. This is best made out by referring to the diagrams (Figs. 336 and 337). Fig. 336 serves for general orientation; Fig. 337, for a comprehension of the anastomoses of the facial nerve.

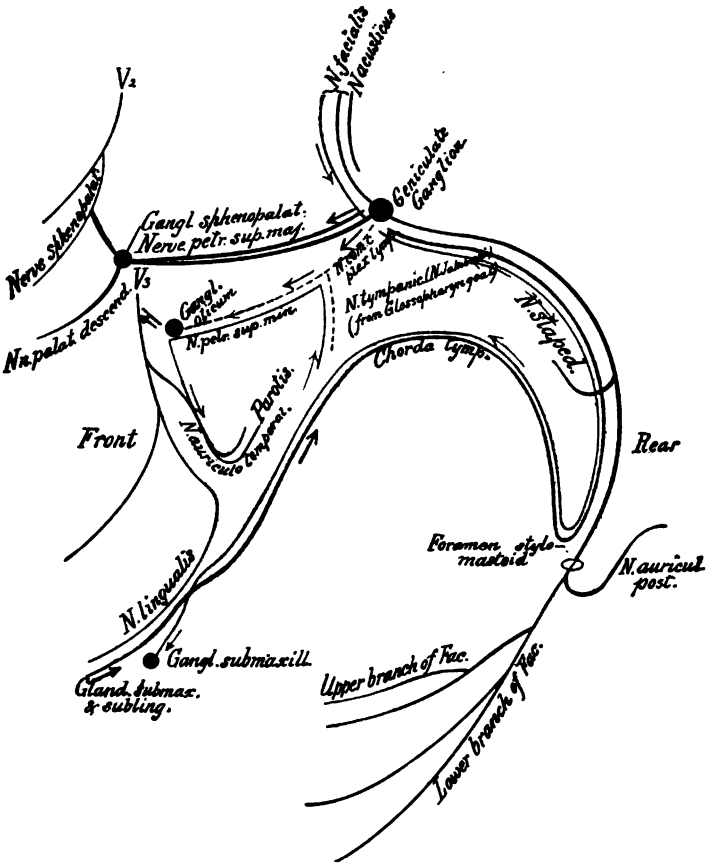

FIG. 337.—Diagrammatic plan of the peripheral facial nerve and its connection with other nerves:

——— Motor fibers of facial.
——— Other cranial nerves.
——— Secretory fibers of the facial to the submaxillary and sublingual glands and of the glossopharyngeal to the parotid.
——— Taste fibers of the facial from the chorda tympani.
- - - - - - Central course of the same from the geniculate ganglion to another less probable connection. (Termination through the nervus tympanicus (Jacobson's) in the glossopharyngeal or through the otic ganglion in the third branch of the trigeminal.)

In Fig. 336 it is evident that a lesion situated at (*a*) will affect the so-called mimic branches of the facial, including the platysma myoides; if it is situated at (*b*), above the exit of the facial from the stylomastoid foramen, the posterior auricular nerve will also be paralyzed (this supplies the occipital muscle and the attolens, retrahens, and transversus

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auricular muscles).[1] As many people cannot move these muscles voluntarily, paralysis of them is oftentimes shown only by an electric examination, and then, of course, only when the electric irritability of the paralyzed muscle has been affected. If the lesion is situated at (*c*), the taste and the salivary secretory fibers of the chorda tympani are also paralyzed. This becomes evident in a diminution of the trigeminus taste appreciation upon half of the tongue in its anterior portion and possibly in a dryness of half of the mouth (see below for a more exact method of testing this function). The sensations of touch and pain in the anterior half of the tongue are sometimes diminished, since the chorda tympani also contains fibers for the appreciation of touch and pain in the tongue. With a lesion at (*d*), the stapedius muscle, supplied by the stapedius nerve from the facial, shares in the paralysis (see above, p. 857, in relation to appearances so caused). Should the lesion be situated still farther above at (*e*), that is above the geniculate ganglion, the disturbances of taste just mentioned escape, because the taste fibers have left the facial at this point, but the palate half will be paralyzed to voluntary and to reflex (see below) impulses, and the secretion of tears (see above) may be affected. Disturbances of hearing from an extension of the process to the cochlea may result from lesions in the neighborhood of the geniculate ganglion. If the paralysis is situated at (*f*), at the base of the brain, where the acoustic and facial nerves are so closely approximated, the former is oftentimes involved with the latter, and in addition to the involvement of the tear secretion, diminution of hearing and disturbances of equilibrium from lesions of the fibers of the ramus vestibuli are added to the symptoms of facial paralysis. When the lesion is localized still higher above—*i. e.*, is nuclear—at least in its most usual form (*bulbar paralysis*), the coincident involvement of other bulbar nerves, the bilateral character of the paralysis, and the very unequal involvement of the individual branches of the facial make the picture sufficiently characteristic. In bulbar paralysis, the territory of the lower facial (see p. 854) is most affected, especially the lip musculature. The facial palsy due to a lesion in the pons, another nucleoperipheral palsy since the peripheral motor neuron is also affected, is usually characterized by paralysis of the extremities upon the opposite side (alternating or crossed paralysis). This is due to the involvement of the psychomotor tract for the extremities (the pyamidal tract) before its decussation, although at this point the facial fibers have already crossed. Between this location and the cortex is the region for central facial paralysis.

In addition to these local diagnostic points (in regard to the special symptomatology of nucleoperipheral facial paralysis), the following facts will be of assistance in completing the clinical picture:

Lagophthalmus, due to paralysis of the eye branch, is a very characteristic sign in most cases of peripheral facial paralysis, and one which furnishes a sharp contrast to central facial paralysis. This con-

[1] According to Heitzmann, the attrahens auricular muscle is supplied by the auriculotemporal nerve from the third branch of the trigeminus.

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sists in the eyes remaining open upon the paralyzed side, despite attempts to close it. In fruitless endeavors to close the eye the globe assumes the so-called sleeping position, upward and outward, more rarely upward and inward, beneath the upper lid, so that patients believe that they have completely closed the eye.

This so-called "*Bell's phenomenon*," just described, is to be attributed to an associated movement of the globe, whose object is to protect the eye. It occurs physiologically, as is shown by its appearance during sleep in a healthy individual, as well as by the fact that it also occurs when a healthy person attempts to close the eye when the closure is mechanically prevented. The sign is more evident in paralysis of the eye branch of the facial, not only because the eye remains open, and so we see it more plainly, but also because the movement appeared to be increased, probably because, after interruption of conductivity in the domain of the facial branches, the motor impulses radiate more intensely through the pathway of the associated movement in a way very analogous to other associated movements. Bell's phenomenon has some prognostic significance, as one of the earliest signs of improvement in facial paralyses is a retrogression of this sign. This is probably to be attributed to an improvement in the conduction function of the facial fibers, whereby a smaller proportion of the motor impulses are diverted into the branch for associated movements; with this improvement of transmission the strength of the motor impulses, corresponding to the better effect, is diminished.

Although patients with peripheral paralysis protect themselves against a painful sensation in touching the cornea, nevertheless the usual lid-closure (corneal reflex, p. 850) either fails entirely or occurs very incompletely. On the contrary, automatic winking (blepharoplegia) is retained (see p. 851).

The true corneal reflex must not be confused (although the mistake is common) with the ordinary eye closure excited reflexly by the visual appreciation of an approaching object even before the cornea has been touched. This latter is really an opticofacial reflex—*i. e.*, the optic nerve constitutes the sensory limb; and physiologically it is a most useful process, since it frequently protects the eye by closing the lid in time to prevent the injury, whereas the true corneal reflex would occur too late to be of any service. The optic reflex, like the genuine corneal reflex, is evidently either lost or diminished in peripheral facial paralysis. It is of some diagnostic importance in determining the condition of the facial in those cases where the true corneal reflex is absent as a result of anesthesia of the cornea (sensory trigeminus paralysis).

When a lesion is situated at (*e*), the palate reflex may be lost. This can be recognized by tickling the patient's pharynx with a brush, although in unilateral facial paralysis it is often very difficult to be sure of it, as the paralyzed half is passively moved by the healthy half of the palate.

Characteristic appearances of irritation are not infrequently observed in the partially paralyzed territory of old severe peripheral facial paralyses which have recovered completely. These consist either of contractures, which may lead to the erroneous supposition of a facial paresis of the opposite side, or of associated movements and of fibrillary contractions in the paretic and atrophic territories.

Hitzig assumes that these phenomena depend upon irritation in the facial nucleus as a result of degenerative processes proceeding from the periphery,

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which hypothesis coincides with Darkschewitsch's explanation. The latter has discovered that peripheral lesions of the motor nerves lead to changes in the nuclear cells. These irritative phenomena in the facial territory may persist through life.

The electric irritability is generally altered in nucleoperipheral facial paralyses. In mild cases there is a more or less diminished excitability and in severe cases a complete or partial reaction of degeneration, resulting in a degenerative atrophy of the paralyzed muscles (see pp. 812 and 816).

Severe peripheral facial paralyses commonly lead to vasomotor phenomena, as shown by the occurrence of coolness, cyanosis, and frequently some edema of the affected side. It has not yet been accurately determined whether this depends upon a connection between the peripheral facial and the vasomotor, or upon a lesion of the sympathetic fibers which are united to the facial branches in the periphery. Neither hypothesis is absolutely necessary, since the muscular action of the affected side is sufficiently impaired to produce some stasis (p. 795 *et seq.*).

The *diminished sensibility of the affected half of the face*, not infrequently observed in facial paralysis, has led to various hypotheses. Some authors, contrary to the general opinion, assume that the facial contains some sensory fibers. This is, however, unnecessary in order to explain the disturbances of sensibility, for certainly sensory trigeminus fibers do anastomose at the periphery with the facial, and these may quite well be injured by the same cause. The absolute immobility of the affected half of the face and the associated circulatory disturbances are alone sufficient to dull the sensibility. Such disturbances are most frequently discovered while the muscles are being tested by the electric current; for this procedure is found to be more painful upon the healthy side, especially in severe paralysis, than upon the injured side. The observation certainly suggests that the degeneration of the muscles may alone be responsible for a disturbance of the sensory nerve terminations. In many cases an hysterical cause may explain the difficulty.

The *pains* accompanying rheumatic facial paralysis in the affected half of the face are probably due to coincident involvement of the peripheral trigeminus fibers running with the facial.

Bulbar paralysis, which, as has been already mentioned, includes a partial nuclear facial paralysis, sometimes exhibits an *increased salivary secretion;* and this is peculiar in so much as there is actually more saliva secreted than normally, and not merely an excessive flow on account of imperfect closure of the lips. We may assume that this increased secretion falls under the heading of a so-called paralytic secretion. Physiologists have noted it in animals after they have cut the several nerves leading to the submaxillary glands. Such an assumption, however, hardly seems justified, in the first place because the fibers supplying the sublingual and submaxillary glands really come from the trigeminus, and in bulbar paralysis the latter nerve is one of the last to be involved, and in the second place because the paralytic secretion is rather a transitory condition, and even if continued is never very decided. The more rational explanation seems to the author to be that the swallowing mechanism is affected in bulbar paralysis, the saliva accumulates in the mouth, and its presence reflexly stimulates the secretion.

(*b*) Spasms of the Facial.

Spasmodic phenomena in the facial territory are not rare, but their description belongs to special pathology, so that here we need only mention their occurrence in the so-called "mimic facial spasm," tetanus, tetany, epilepsy, and chorea. The peculiar twitching of the lid depending upon fibrillary contractions of the facial, is worth mentioning from the diagnostic standpoint. It occurs in neurasthenics when they attempt to close the eyes at command. Such an attempt, as has

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been frequently noted, causes some degree of difficulty in many cases. This twitching is quite an important sign of neurasthenia. Irritative appearances in the territory of old facial paralyses have been mentioned in preceding paragraphs (p. 863 *et seq.*).

VIII. CRANIAL NERVE: AUDITORY (Acusticus).

(*a*) Paralysis of the Auditory Nerve.

Auditory paralyses occur in diseases of the internal ear, of the petrous bone, in affections of the base of the brain and of the medulla oblongata, and finally as a part of cerebral hemianesthesias.

A unilateral acoustic paralysis in hemianesthesia is never absolute, except in hysteric affections; there is much more apt to be only a moderate impairment of hearing. This may be accounted for by the fact that the central innervation of the acoustic is not entirely crossed, and coincides, so far as the author knows, with the fact that in unilateral lesions of the auditory center, situated in the temporal lobes, no instance of complete crossed deafness has as yet been observed.

In examining the auditory nerve, a procedure which should, of course, be carried out in as quiet a room as possible, the perception of sound by air conduction must be sharply distinguished from that by bone conduction. In testing the former, a ticking watch or vibrating tuning-fork is held near the external auditory canal, the maximum distance determined at which one or both can be heard, and the two sides then compared. In testing the latter, the perception of sound by bone conduction, the watch or tuning-fork is held upon the mastoid process, and any difference between the two sides determined from the patient's statements. Since neither watches nor tuning-forks emit sounds of definite intensity, Politzer[1] has constructed a sound-meter with which a sound of constant intensity may be produced by allowing a small hammer to fall upon a metallic bar. Politzer's sound-meter may be employed to test the perception not only of sounds transmitted through the air, but also of sounds conducted by bone, in which case the instrument is brought into contact with the cranial bones by means of a rod-shaped attachment.

Rinne's test is also of some importance. It consists in holding a vibrating tuning-fork to the mastoid process, until the patient can no longer appreciate any sound; then the tuning-fork (which is still vibrating) is quickly approached to the external ear. If the auditory transmitting apparatus is functionating well, the tuning-fork is heard again (positive result of Rinne's test); should this not be the case (negative result of Rinne's test) there must exist some disease of this apparatus. If the issue of the test is positive the transmitting apparatus is not necessarily perfect, because air conduction permits a better auditory impression than bone conduction, and slight affections of the sound-transmitting mechanism might escape notice.

To determine a disturbance of hearing independent of the otoscopic findings and Weber's and Schwabach's tests (see below), Rinne's test must be employed in a more exact quantitative manner by determining the degree of superiority

[1] Politzer, *Lehrb. der Ohrenheilkunde*, Stuttgart, 1887.

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of air-conduction over bone-conduction by measuring the time during which a vibrating tuning-fork is heard at the external auditory meatus after it has become inaudible upon the mastoid process. Under normal conditions this time for an A′ tuning-fork amounts to about 30 seconds. If Rinne's test gives a negative result, we may proceed in the opposite manner by determining the time during which the tuning-fork upon the mastoid process is heard after it has become inaudible when held in front of the external auditory meatus.

Zimmermann has recently objected to the principles involved in Rinne's test, believing that in placing the tuning-fork upon the bone we test the energy of movement of the handle of the fork, and that in holding the tuning-fork at the external auditory meatus we test the much greater energy of movement of the prongs. From this he concludes that the diagnostic importance of Rinne's test is doubtful and that the normal positive result cannot be employed as an argument for Helmholtz's theory, according to which the membrana tympani and the auditory ossicles are assumed to aid in the transmission of sound. According to Zimmermann's theory, (see p. 858 *et seq.*), the normal positive result of Rinne's test is due to the fact that the auditory ossicles may really be an accommodation mechanism for the acoustic organ, or, in other words, a sound-dulling rather than a sound-transmitting apparatus. These views of Zimmermann have been successfully combated by v. Bezold, who has suggested a new method of performing Rinne's test. Performed in this way, the test is free from objection and rehabilitates Helmholtz's theory. He provides the *a′* tuning-fork with a rounded handle and after the vibrations have become inaudible, both upon the mastoid process and at the external auditory meatus, he introduces this handle into the meatus so that an air-tight closure of the canal is effected. It then becomes manifest that the audibility of the tuning-fork, introduced in this manner, is increased by about 12 seconds over the ordinary perception by means of air conduction; the vibrations are consequently perceived by air conduction about 30 + 12 seconds longer than by bone-conduction (the positive normal result of Rinne's test carried out in this manner). In this test air conduction and bone conduction are both measured with the handle of the fork, so that the results may be absolutely and directly compared.

The so-called *Weber's test* has a similar significance for the differentiation of impaired hearing due to disturbances of the conducting apparatus from that dependent upon affections of the nervous portion of the acoustic organ. The handle of a vibrating tuning-fork is placed upon the middle of the vertex, and the patient is required to state upon which side he perceives the louder sound. In affections of the conducting apparatus the sound is usually more distinctly perceived upon the affected side, while the reverse is true in affections of the auditory nerve or of the labyrinth. The chief obstacle to Weber's test is the uncertainty of the localization of the auditory perception in one ear.

Schwabach's test consists in determining whether the vibrating tuning-fork is perceived by bone conduction for an abnormal period of time. If it be heard longer than normal, Schwabach supposes a disturbance of the conducting apparatus; if not so long as normal, an affection of the nervous mechanism. If the investigator be possessed of normal hearing this comparison is best made by removing the tuning-fork from the patient's mastoid process as soon as he declares it to be inaudible and placing it upon the mastoid process of the investigator; the result is then to be compared with that obtained by reversing the process.

The results of all these tests, however, should be utilized with caution, since the pitch and intensity of the sound employed may sometimes cause them to vary and even be the direct opposite of what the formulated rules would lead us to expect.

The usual method adopted in ear clinics for testing the auditory nerve is by

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means of determining which of a series of tuning-forks, comprising the entire tone scale, can be appreciated. This method has not yet been adopted in clinical medicine, because only gross disturbances can be utilized in the local diagnosis of cerebral diseases.

The acuteness of hearing should be tested by means of the whispered voice, as well as by means of the above-described instrumental test. In a quiet room the examiner notes at what distance from each ear (the other being closed) the whispered voice can be heard; experience has shown that such a method frequently furnishes quite different results from the instrumental test. The whispered voice is preferable to the loud voice, not only because the latter would be too intense in a closed space, but also because the individual sounds and words are more uniformly adapted for auditory perception when whispered than when loudly spoken.

(*b*) Irritative Phenomena of the Auditory Nerve.

Subjective sound perceptions result from various cerebral diseases, from galvanic stimulation of the brain, and especially from affections of the internal and middle ear. The most familiar are the ear noises heard in chronic sclerotic otitis media, the "crux" of the otologists. This acoustic phenomenon of irritation does not possess in cerebral disease any very important local diagnostic significance, because even the most exact otoscopic examinations cannot differentiate with certainty the subjective perceptions of peripheral from those of central origin. A peripheral origin is much more probable, on the one hand, on account of the great frequency of middle-ear diseases, and, on the other hand, on account of the generally applicable law that the terminal organs of sensory nerves are the most irritable parts of the sensory apparatus. Pathologic appearances of the drum argue for a peripheral origin of the ear noises. A normal drum, however, does not necessarily exclude such an origin.

Auditory Vertigo (*Menière's Disease*).—Since the vestibular branches of the acoustic nerve supply the semicircular canals, and since the latter are regarded as the organs of equilibrium, it is easy to understand that vertigo will result from affections of the acoustic nerve. Vertigo is in fact most usually observed accompanying affections of the labyrinth and of the middle ear. Cerebellar vertigo must be closely identified with auditory vertigo on account of the anatomic proximity of the auditory nerve to the cerebellum.

(*c*) Otoscopic Examinations.

Disease of the hearing apparatus is frequent, even in people who consider themselves perfectly well. Hence, under all circumstances in which a disturbance of hearing has been determined, it is important to make an otoscopic examination. In no other way can we differentiate between disturbance of hearing dependent upon disease of the auditory nerve and that depending upon the ear itself. This distinction, as was mentioned above, cannot always be determined by the Rinne test nor in some instances even by an otoscopic examination, so that a disturbance of hearing will not always assist much in the diagnosis of cerebral disease.

The reader is advised to consult the otoscopic pictures in Politzer's "*Atlas der Beleuchtungsbilder des Trommelfells*," Wien, Braumüller, 1896.

(*d*) Demonstration of Simulated Deafness.[1]

When total *deafness of both ears* is simulated a more prolonged observation in a hospital is usually all that is necessary to determine it.

[1] Partly taken from Siebermann, "Untersuchung auf Simulation von Schwerhörigkeit oder Taubheit," *Schweizerischer Medicinalkalender*, 1895, p. 76 f.

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The simulation of bilateral or unilateral impairment of hearing becomes evident when we test the patient's hearing (that of each ear) with his eyes closed, so that he cannot see the distance from which the sound comes. He will then express contradictory statements in regard to rapidly repeated tests.

Simulation of unilateral total deafness may be discovered in various ways:

1. The patient's normal ear is closed, and the examiner speaks directly in front of the supposedly deaf ear. If the patient claims not to hear this, either simulation or exaggeration exists, since under such conditions even the closed healthy ear would appreciate the conversational voice.

2. One end of a flexible rubber tube, about 4 feet long, is inserted into the external canal of the patient's ear, another similar tube into the other ear. Funnels are affixed to the other ends of each tube. The examiner, speaking in a whisper behind the patient, talks a tone moment into the one, and at another moment into the other funnel, alternating as quickly as possible, and the patient is required to repeat what is said. In consequence of the confusion and the fatigue which this process produces, if simulation exists the patient finally repeats what is spoken into the supposedly deaf ear. The examiner must be trained for the test. The best plan is to write down beforehand what is to be said in two columns, one marked L for the left ear, and the other R for the right ear. This procedure is to a certain extent analogous to the stereoscopic method of investigating simulated unilateral blindness (p. 825).

3. An A′ tuning-fork is set in vibration and placed vertically upon the middle of the skull; if even one ear is normal the patient must hear the tone. The test is then repeated with the normal ear closed, and if the patient says that he no longer hears the tone, it is evident that he is simulating, since by bone conduction the normal ear, even if it is closed, must hear the tuning-fork.

4. Many malingerers are exposed by the fact that they insist upon their inability to determine whether the tuning-fork vibrates or not, when the examiner places a large vibrating tuning-fork (A or C or A′) upon the skull in the neighborhood of the supposedly deaf ear, whereas the vibrations of such a type of tuning-fork can be felt even by the deaf.

Siebemann quite correctly calls attention to the danger of confusing exaggeration and complete simulation, and emphasizes the advisability of never placing any reliance upon the results of one method of examination.

In regard to the demonstration of the simulation of disturbances of hearing, the reader is referred to Burchardt's works, referred to upon p. 826.

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IX., X., XI. CRANIAL NERVES: GLOSSOPHARYNGEAL, VAGUS, SPINAL ACCESSORY (VAGUS GROUP).

Physiologic Introduction.

These three nerves are so intimately connected at their origin in the oblongata, and both during and after exit from the jugular foramen, and they possess so many anastomoses, that experimental physiology has not yet succeeded in isolating the functions of each. This is especially so in the case of the vagus and spinal accessory. Clinical investigations are made exceptionally difficult, because, as a result of the anatomic relations of these nerves, pathologic conditions frequently affect the three simultaneously, both centrally and peripherally.

The *peripheral glossopharyngeal nerve* supplies *motor fibers* to the muscles of the pharynx, to the pharyngeal constrictors, and to the stylopharyngeus muscle; and it participates with the facial and the accessory in the supply of the muscles of the soft palate. In addition the glossopharyngeal is the *secretory nerve* of the parotid gland. From the petrous ganglion it gives off these secretory fibers by means of the tympanic nerve (Jacobson's) to the otic ganglion, and from there by means of the auriculotemporal nerve of the third trigeminal branch to the parotid gland (Fig. 337, p. 861). The glossopharyngeal also contains *sensory fibers*, which are distributed to the back of the tongue, to the pharynx, and to the soft palate. These fibers are responsible for the sensation of taste—*i. e.*, for bitter and sweet—and they also prevent breathing during the act of swallowing.

The *peripheral vagus* includes both motor and sensory fibers. The *motor fibers*, with the help of the glossopharyngeal and accessory, supply the muscles of the pharynx, the soft palate, and the esophagus, so that the vagus has with the glossopharyngeal an important function in the act of swallowing. In addition it furnishes motor fibers to the larynx in its inferior or recurrent laryngeal branch, and there supplies all the laryngeal muscles, except the cricothyroid and the two epiglottis muscles (the thyro- and ary-epiglottis). The last three, although not innervated by the recurrent laryngeal nerve, are supplied by fibers derived from the vagus, through the superior laryngeal nerve. The vagus is furthermore the motor nerve of the stomach and of part of the intestines; it is also the inhibitory nerve of the heart, for, as is well known, a centrifugal irritation of one or both vagi slows the action of the heart and at the same time diminishes the cardiac power, whereas sectioning one or both vagi increases the cardiac rapidity. The vagus is further assumed to innervate the bronchial muscles and the vessels of the lungs. Sensory branches: It gives off a *small sensory branch* (auricular branch of the vagus) to the posterior wall of the external auditory canal. Thus the mucous membrane of the pharynx, larynx, trachea, and bronchi is supplied by the vagus, as well as the heart and probably also the stomach and intestines. The sensory nerves for the upper part of the larynx run in the so-called *superior laryngeal branch*, those for the lower part of the larynx in the *inferior* or *recurrent laryngeal*. The sensory fibers of the vagus exert an important regulatory control over the breathing; their best-known effect, perhaps, is the action of the superior laryngeal in centripetal stimulation—the respiration ceases in the condition of expiration. The sensory vagus branches to the lung itself influence breathing to the extent that distention or inflation of the lung excites expiration, while compression or collapse of the lung excites inspiration. The automatic regulation of breathing, as explained by Hering and Breuer, should be understood to mean merely that the sensory irritation of the pulmonary branches of the vagus endeavors to accomplish a median position of pulmonary distention, whereas the rhythm of breathing is independent of such regulation, but is much more an automatic function of the respiratory center. In reality the only influence that the centripetal vagus fibers have upon the rhythm is that sectioning the vagus slows the respiration, while weak centripetal vagus irritation increases it. The vagus is the most important sensory nerve for coughing, which can be induced by any one of its sensory branches (even including the auricular branch). The vagus branch coming from the heart, which can be demonstrated in animals,

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is called *Ludwig Cyon's depressor*. It is of some clinical interest, because its centripetal irritation produces a marked diminution of blood pressure without affecting the heart, probably from vascular dilatation.

The peculiar sensation of nausea and the reflex vomiting depending upon such sensation may start from the sensory branches of the vagus to the pharynx and stomach. Vomiting does not, however, depend upon intact vagi, for in animals the vomiting paroxysm can still be excited from the stomach, even after bilateral section of the vagus. Other tracts for the vomiting reflex probably must exist, although they are unknown. The vagus is supposed to have some *secretory influence*, but it has not yet been accurately demonstrated.

The functions of the *peripheral accessory nerve* are simple; they are purely motor. Its *internal branch* anastomoses with the pharyngeal branches of the vagus and the glossopharyngeal, thus sharing in the motor innervation of the soft palate, while its *external branch* supplies the sternocleidomastoid and trapezius muscles.

The determination of the central origin of fibers which are limited to individual functions is much more difficult than the study of the peripheral branching and functions of these three nerves. Two points are sure—that the taste fibers are derived from the glossopharyngeal nucleus, and that the innervation of the sternocleidomastoid is exclusively a function of the accessory nucleus. From reasons which have been detailed above, we are still undecided whether many functions executed in the peripheral tracts of the vagus and glossopharyngeal do not depend upon fibers which in reality are derived from the accessory nucleus, but which at the periphery have been attached to the two other nerves. For instance, the heart-accelerating fibers of the vagus, and especially the motor fibers for the larynx, are supposed by many authors to be derived from the accessory nucleus. Grabower has recently denied this relation of the accessory to the larynx. At all events the question is not settled. Nor is it yet possible to determine with certainty how far the accessory nucleus shares in the innervation of the glossopharyngeal and vagus fibers which supply the swallowing and palate musculature.

In regard to the anatomic relations of the nuclei of the three nerves of the vagus group see Figs. 343, 344.

Pathologic Relations.

The fibers of the three nerves of the vagus group may be affected either in their peripheral course or within the cranium—*i. e.*, the oblongata. The same peculiarities are true in regard to their central (supranuclear) innervation, as in the case of the nerves to the eye muscles, of the motor trigeminus, and of the superior branch of the facial, viz.: that their central innervation is not exclusively contralateral, but that the hemisphere of each side takes part in innervating the two nerves (see Fig. 318, p. 829). As a result a hemispheric lesion occasions very little or no disturbance in the nerves of the vagus group, because the other hemisphere then performs the function sufficiently well; and therefore without further discussion unilateral affections can for the most part be attributed to a lesion at the base of the brain or in the oblongata, provided, of course, that the trouble is situated within the skull. This is exactly the same condition as in unilateral paralysis of the eye muscles (see p. 832 *et seq.*) and motor paralysis of the trigeminus (p. 849). With a cerebral hemiplegia, such as is usually localized in the cerebrum, there is practically never any affection of the vocal cords, of the muscles of swallowing, nor of the sternocleidomastoid, whereas a paresis of the trapezius is apt to be noticed upon the hemi-

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plegic side, because the innervation of this muscle is for the most part entirely crossed. The clavicular portion of the trapezius, however, which aids in respiration, is bilaterally innervated and so exempt from this paresis. A bilateral paralysis of the nerves of the vagus group may, of course, arise in pseudobulbar fashion from bilateral hemispheric lesions (see 876).

Symptomatology of Lesions of the Three Nerves of the Vagus Group.—Disturbances of the motor innervation of the soft palate may occur in paralysis not only of the facial, but also of the glossopharyngeal and vagus, as well as of the nuclear and trunk portions of the accessory. In regard to the phenomena of unilateral or bilateral paralysis of the palate, compare the section upon the facial nerve (see pp. 850 and (reflex) 863). In a similar way the peripheral glossopharyngeal and vagus, and even the region of origin of the accessory, are to be considered also in connection with the other movements of swallowing. A patient's subjective complaints and the examiner's observation of the act of swallowing, will disclose any affection of that function. A unilateral vagus paralysis, such as is observed surgically, does not noticeably affect swallowing. Disturbances in the motor innervation of the larynx, proceeding from the vagus or the accessory, become evident either during phonation, or by the exhibition of deficient laryngeal closure or laryngeal stenosis (in posticus paralysis, see below). Imperfections in phonation depend upon a lesion of fibers running to the vocal-cord musculature in the inferior laryngeal nerve. Deficient closure of the glottis depends upon a paralysis of the glottis constrictors (crico-arytenoideus lateralis and interarytenoidei), as well as of the aryepiglottic muscle. The two former are supplied by the inferior laryngeal, and the latter by the superior laryngeal nerve. The consequence of deficient laryngeal closure is that patients readily choke in swallowing, and can no longer effectually cough or exert abdominal pressure. Traube has proved that paralysis of the glottis closure, with inability to cough and expectorate, is the most essential cause of the disastrous results (vagus phenomena) of bilateral section of the vagus in animals and of bilateral vagus paralysis in men. Whether or not paralysis of the bronchial muscles and of the pulmonary vessels plays any rôle in this is not yet accurately determined. In unilateral vagus paralysis phonation and glottis closure are not always so markedly affected as one would think, because a more pronounced movement of the healthy vocal cord will offset to a great extent the paralyzed cord. Still, the hoarse feeble voice of unilateral vocal paralysis is so very characteristic that such a diagnosis may often be ventured by the skilled, even before a laryngoscopic examination. A gradually developing paralysis of the recurrent nerve (it is quite immaterial whether this is of peripheral or of nuclear origin) almost always particularly involves the crico-arytenoideus posticus. This muscle widens the glottis. Hence the earliest symptoms of an increasing motor laryngeal paralysis is a narrowing of the glottis, because the constrictors overpower the weakened posticus. The corresponding vocal cord assumes a position of adduction. Should

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the paralysis be bilateral, there may result a serious obstacle to breathing with all the appearances of laryngeal stenotic dyspnea (see p. 86 *et seq.*), even necessitating tracheotomy. This condition is generally described briefly as "posticus paralysis." This remarkable selection of the crico-arytenoideus posticus in the beginning of recurrent paralysis has led to countless discussions. No satisfactory explanation has yet been given. One theory is that those neurons which preside over glottis widening suffer on account of a greater sensitiveness, but this seems to the author arbitrary and superfluous. A much simpler explanation is that, quantitatively, the musculature of the glottis constrictors overpowers the dilators very decidedly. This preponderance is in keeping with the fact that in phonation, coughing, exerting abdominal pressure, and, in short, in all forms of glottis closure, the constrictors accomplish very much more than the dilators. As a matter of fact, all laryngeal muscles, with the single exception of this "posticus," subserve the function of narrowing the glottis, and therefore it is not surprising that the crico-arytenoideus posticus is the first to suffer, and that in partial pharyngeal paralysis the constrictors overpower it. Evidently then, as the paralysis increases, the narrowing of the glottis recedes, while the vocal cord (or cords) from a position of adduction assume more and more the so-called cadaveric position. The exact diagnosis of a motor laryngeal paralysis can be accomplished only by means of the laryngoscope. (Compare the chapter upon Laryngoscopy and the illustrations of the larynx reproduced there, p. 696). A normal laryngoscope picture is characteristic of hysteric aphonia: the larynx resembles that of a person voluntarily whispering. There is no actual paralysis in the territory of the vagus and accessory, but merely a psychic inability to control the speech volition. Spasm of the glottis and the spastic form of hysteric aphonia may be mentioned here, as motor vagus accessory symptoms; but for their explanation the literature of special pathology should be consulted.

Compare p. 850 *et seq.* (trigeminus) concerning derangements of taste peculiar to lesion of the glossopharyngeal (especially for bitter and sweet). Affections of the sensory innervation of the larynx (superior and inferior laryngeal nerves) can be demonstrated by touching the mucous membrane of the larynx by means of a curved laryngeal sound under the control of a mirror, and noting the absence of sensitiveness, and finally the loss of the cough reflex.

The determination of disturbances in function of the cardiac and pulmonary branches of the vagus is very difficult, because all the changes which irritative and paralytic conditions of these nerves for the circulation and respiration produce can take place pathologically in other ways, especially in affections of the heart and of the lungs themselves, and also reflexly from almost any nerve territory. The establishment of less ambiguous symptoms, especially the determination of the phenomena described above in connection with the act of swallowing and the laryngeal innervation, as well as the establishment of lesions whose nature and position can be definitely attributed to the vagus, will gener-

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ally be sufficient to attribute certain respiratory and circulatory disorders to paralytic or irritative vagus phenomena. The cases of traumatic vagus paralysis are the easiest to understand.

The results of surgical or other accidents, such as cutting the vagus nerve during operations, or the results of injury to both vagi from the pressure of tumors, prove that a unilateral vagus paralysis need not influence the pulse very materially. At first it may be accelerated, but later on it will become equalized. A double vagus paralysis will, however, generally induce a permanent acceleration, up to 160 beats in a minute. Dilatation of the heart has not yet been observed to depend upon unilateral or bilateral vagus paralysis. Nor is there any foundation for the frequent assumption that irregularity of the pulse is to be attributed to paralyzing affections of the vagus. On the contrary, physiology teaches that irritation of the vagus may cause irregularity in the pulse.

The rule formulated by Gerhardt in connection with the diagnosis of the cause of *tachycardia* should be mentioned here: "Taken all in all, the majority of nervous tachycardias are to be attributed to vagus paralysis, those with very rapid pulse (above 200) to a combination of vagus paralysis with sympathetic irritation, a few rather milder forms to the latter alone." Martius adds: "This rule coincides with well-known physiologic facts. In unpoisoned animals an acceleration of the heart pulsation, 30 to 70 per cent. (not more, Aubert) can be brought about by stimulating the accelerating nerve of the sympathetic. After bilateral sectioning of the vagus, the acceleration of the pulse-frequency in mammals is not very great. According to v. Bezold it may rise to 120 to 180 beats. The following conclusions may be drawn: Acceleration up to about 120 beats (30 to 70 per cent.) depends upon irritation of the sympathetic; from 120 to 180 beats, upon paralysis of the vagus; more than that, upon a combined action of both causes."[1] Martius objects to the assumption of a vagus paralysis or of a sympathetic irritation to explain tachycardia. He assumes neither a vagus paralysis nor sympathetic irritation to interpret the characteristic clinical picture of paroxysmal tachycardia, but considers that it depends upon a temporary acute cardiac insufficiency with dilatation, in which the tachycardia is one of the secondary symptoms, probably for the sake of compensation. But this conception of Martius does not seem logical to the author. For in these cases of paroxysmal tachycardia evidence of cardiac insufficiency is so much in the background that one might better assume a sort of epileptic discharge in the territory of the accelerating nerve of the heart. Nothnagel has also made an attempt to formulate rules for diagnosticating the cause of tachycardia. He says: 1. "If a very decided acceleration of the pulse occurs in paroxysmal tachycardia, if the rhythm of beats is quite regular, and the cardiac impulse is very weak, if other symptoms are absent or are developed first as consequences of incomplete cardiac emptying, if, finally, the paralysis of other nerve tracts accompanying the vagus can be coincidentally confirmed, then one can assume a vagus paralysis as a cause in this special case. 2. If during the attacks of tachycardia the cardiac impulses are vigorous, if the peripheral arteries are well filled, and the tension is preserved (but this is not in any sense a necessity), if other marked evidence of irritation in vasomotor nerve tracts appears in paroxysms, then the assumption of a condition of irritation of the accelerating nerve (sympathetic) is justified." This position seems to the author arbitrary and untrustworthy.

The statements which are to be found in literature concerning the behavior of the breathing in vagus paralysis are of little use. Accord-

[1] Martius, *Tachycardia*, Stuttgart, Enke, 1895.

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ing to physiologic experiments it is fair to assume here, as with the heart, that only bilateral lesions can cause serious difficulties. Edinger's statement is worth noting that vagus paralysis may be associated with pulmonary distention and consequently with dyspnea. Vagus pneumonia (as we saw upon p. 871) depends essentially upon incomplete laryngeal closure, and is therefore merely an indirect pulmonary symptom of vagus paralysis.

As yet there is very little known about the effect of vagus paralysis upon disturbances of the functions of the stomach and intestines in man. This alone appears certain—a unilateral vagus paralysis does not noticeably influence these functions.

We now come to the mention of the symptomatology of the affections of the so-called external branch of the accessory, which supplies the sternocleidomastoid and trapezius muscles. A unilateral paralysis of the sternocleidomastoid causes a moderate twisting of the head toward the paralyzed side, associated with a slight elevation of the chin, due to the action of the antagonists. Turning the head to the healthy side, although performed less vigorously than normally, is not prevented, because this movement is accomplished not exclusively by the sternocleidomastoid but also by the deep muscles of the neck, especially the oblique capitis inferior, and the splenius of the other side. The clonic and tonic wry neck (*tic rotatoire, caput obstipum spasticum*) depends partly upon a unilateral irritation of the spinal accessory nerve leading to the sternocleidomastoid. Yet the unsatisfactory results following myotomies of the sternocleidomastoid alone, and the very much better results when the opposed splenius and the oblique capitis inferior have also been cut, show that the name "accessory cramp" for this condition is not absolutely justified. In reality the condition depends much more upon spasm of a central extensive area which supplies functionally similar muscles. The symptoms of unilateral paralysis of the trapezius vary according to whether the entire muscle or only separate portions of it are involved. If complete the affected shoulder hangs lower; the shoulder-blade is thrust out obliquely upwards and outwards; and the power to lift the arm is somewhat impaired, but not nearly so markedly as in serratus paralysis. If only the central portion (acromial) is paralyzed, the upper half of the median edge of the shoulder-blade is depressed outwards (*Mouvement de bascule*, Duchenne). According to Schlodtmann, if the origin of the accessory is affected, this central portion of the muscle should remain exempt because it is entirely or partly supplied by the cervical plexus. This point has not yet been definitely decided, nor has the extent to which the cervical nerves assist in the innervation of other parts of the trapezius. It is worth noting that in juvenile muscular atrophy the clavicular bundle of the trapezius, whose function is essentially respiratory, remains intact longest, so that Duchenne has described it as the ultimum moriens of the muscle.

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XII. CRANIAL NERVE: HYPOGLOSSUS.

The hypoglossus is the motor nerve of the tongue, and therefore aids in chewing, swallowing, and especially in speaking. Its function is tested by observing whether coarse movements of the tongue are performed equally well upon both sides, and by watching the patient while chewing and swallowing. With a unilateral hypoglossal paralysis or paresis the tongue, when protruded, deviates towards the paralyzed side because the healthy genioglossus muscle overpowers its paralyzed fellow.

Further evidence proving that the genioglossus muscle is at fault in unilateral hypoglossal paralysis is furnished by observing the tongue as it rests naturally upon the floor of the mouth. As a result of the preponderance of the unaffected muscle, the tip will, in this position, be deviated toward the healthy side; and, in addition, a more prominent arching of the dorsum of the tongue will be noted upon the paralyzed side. This curving shows that the pushing of the genioglossus forward has ceased.

In unilateral hypoglossal paralysis, we by no means always notice any pronounced affection either in chewing or swallowing. Even articulation may be carried out reasonably well, especially after some practice.

In old peripheral paralyses of the hypoglossus the affected half of the tongue is flaccid, thin, and wrinkled and often shows fibrillary contractions in the shape of peculiar peristaltic wavering. Electric stimulation of the lingual nerve (chorda tympani fibers) will oftentimes intensify this wavering so decidedly that an actual movement of the tongue occurs (pseudomotor action, Heidenhain). The tongue is frequently more coated upon the paralyzed than upon the healthy side.

With bilateral hypoglossal paralysis the disturbances of function are naturally very marked. The tongue then lies flaccid upon the floor of the mouth and can be protruded only very incompletely, if at all. Speech becomes incomprehensible, and mastication, chewing, and swallowing finally impossible. The patient cannot even swallow the saliva but must either spit it out frequently or constantly drool.

In a peripheral hypoglossal paralysis the muscles attached to the hyoid bone and supplied by the descending branch of the hypoglossus are also frequently involved in association (sternothyroid, thyrohyoid, sternohyoid, and the inferior belly of the omohyoid). The fibers for these muscles originate in the second and third cervical nerve roots. Part of them join the root of the hypoglossus nerve and later leave it again as the descending branch; part of them terminate further down in the latter branch. When in conjunction with a lingual paralysis these muscles are found to be affected, it is natural to conclude that the lesion is situated in the hypoglossal trunk below the anastomosis with the superior cervical nerves. The paralysis of the inferior hyoid muscle can be recognized by an atrophy of the musculature over the thyroid cartilage and by a more noticeable prominence of the latter. If the paralysis is unilateral, the larynx will be seen to be laterally dislocated during the act of swallowing. Under some circumstances such paralysis may be demonstrated by an electrical examination (motor points, see p. 801, Fig. 305).

The electric examination of the tongue itself should be carried out in the ordinary way (p. 798 *et seq.*). The motor point of the hypoglossus is situated just behind and above the horn of the hyoid bone.

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In many individuals, the nerve can be stimulated alone at this point by deep pressure of the fine electrode. The hypoglossus of each side is innervated by both hemispheres, and therefore unilateral cerebral lesions causing a hemiplegia give very little evidence of hypoglossal paralysis. The same conditions apply as in the behavior of the superior facial branch with a hemiplegia of central origin (see p. 853 *et seq.*). The crossed influence is, however, responsible for a more or less plain deviation of the tongue towards the paralyzed side in an ordinary hemiplegia, depending, as has already been said, upon a weakness of the genioglossus muscle upon the paralyzed side. This deviation of the tongue in hemiplegia ordinarily runs a parallel course with the facial paralysis, and for this reason was formerly attributed to the facial nerve. Such a supposition is, of course, incorrect, but the coincidence of central facial paralysis and hypoglossal paresis does depend upon the intimate proximity of the central tracts, or even of the cortical centers, of these two nerves. Ordinarily no particular difficulty in chewing, swallowing, or speaking results from this hemiplegic hypoglossus paresis; at most it has only a transitory effect on these functions.

II. THE CHARACTERISTICS OF MOTOR HEMIPLEGIA: PSEUDO-BULBAR SYMPTOMS.

In the preceding paragraphs (see the diagram Fig. 318, p. 829) we have emphasized the fact that most of the cranial nerves are supplied by both hemispheres, so that a unilateral hemispheric lesion producing a hemiplegia of the extremities does not cause any marked crossed paralysis of the cranial nerves. This rule applies particularly to the nerves of the eye muscles (except in the conjugate tract for the lateral movement), to the motor trigeminus, to the motor glossopharangyeal, to the vagus, and to most fibers of the accessory—*i. e.*, the vocal cord fibers and the fibers of the sternocleidomastoid. As was mentioned above, a unilateral hemispheric lesion has a slight crossed effect upon the upper branches of the facial, upon the hyoglossus (genioglossus) and upon the fibers of the trapezius, with the exception of the clavicular portion, which remains intact. The fibers of the inferior facial branch are, on the contrary, very decidedly affected, because crossed. Therefore the typical clinical picture of cerebral hemiplegia includes a hemiplegia of the extremities and of the muscles supplied by the lower branch of the facial, while those supplied by the other motor cranial nerves are either intact or slightly and partially paralyzed.

In a similar way the muscles of breathing and abdominal pressure, apparently innervated bilaterally, present merely a slight negligible weakness on the paralyzed side.

A bilateral defective innervation is, however, present in any unilateral hemispheric lesion, but escapes notice because it is very difficult to detect any objective evidence of a moderate degree of bilateral paralysis (this was alluded to in the case of the eye muscle upon p. 831 *et seq.*, and is quite as true for other muscle territories innervated bilaterally).

Wernicke and Mann undertook not long ago a more accurate analysis of motor hemiplegia in order to compare the degree of involvement of the individual muscles of the extremities. They demonstrated, what had been well known for a long time, that the leg is always affected less than the arm, and formulated the following rules in regard to the amount of involvement of the individual muscle groups of the arm and leg.

Arm[1]: The movements most affected, and in milder and less distinctive par-

[1] Wernicke, *Berlin. klin. Woch.*, 1889, p. 45, and *Lehrbuch der Gehirnkrankheiten*, 1881. Cassel, Fischer.

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alysis oftentimes the only ones, are extension in all joints (elbow, hand, and finger joints), supination of the hand, ab- and adduction and the opposition of the thumb, and spreading of the fingers. All other movements, especially those of flexion, are less affected. The ordinary position of the paralyzed arm is therefore flexed at all joints and slightly pronated.

Leg[1]*:* The muscles most decidedly paralyzed, and, in milder and less distinctive paralysis, the only ones affected are those shortening the leg in walking—*i. e.*, those which flex the leg and are thereby efficient at the first stage of the movement. The more important muscles for walking—those which elongate the leg and so push the body forward—are, on the contrary, either less paralyzed or in milder cases not at all affected. The ileopsoas, the gracilis, the sartorius, and

FIG. 338.—Left hemiplegia showing contractures and distortion of mouth (Dr. E. G. Cutler, Massachusetts General Hospital).

the dorsal flexors of the foot—*i. e.*, the tibialis anticus and extensor digitorum communis—are therefore conspicuously paralyzed. Although the long head of the biceps and the semitendinosis and semimembranosis can act as flexors of the leg, they are less paralyzed. This, however, does not argue against the above rule, because, on account of their extensor action upon the hip joint in walking, they functionate not as flexors of the leg, but as extensors of the thigh; in other words, as elongators of the leg. In the same way the gastrocnemius does not act as a flexor of the leg in walking, but as an extensor of the foot, therefore elongating the leg, and so, in accordance with the above rule, remains comparatively free in hemiplegias. It may be added that in hemiplegias, flexion of the thigh

[1] Mann, *Volkmanns Sammlung klin. Vorträge. Neue Folge*, No. 132, Leipzig, 1895, and *D. Zeits. f. Nervenheilkunde*, 1896, vol. x., parts 1 and 2, p. 1.

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is often found to be less affected than Mann's rules would lead us to think, because the quadriceps acting as an extensor aids the ileopsoas in this movement.

To explain this peculiarity in distribution of motor paralysis in hemiplegias, which applies both to cerebral and to spinal hemiplegias, we must assume that the seriously paralyzed muscles, like the muscles supplied by the inferior branch of the facial, depend for their innervation upon the opposite hemisphere, whereas the groups of muscles less affected by the hemiplegia, like those supplied by the upper branch of the facial and most motor cranial nerves, are supplied by both sides and therefore exhibit, perhaps a weakness, but never any decided paralysis. The researches of Pitres and Dignat[1] completely corroborate this assumption of bilateral innervation, for in cerebral hemiplegias they found a diminution in the power of the healthy leg up to about 50 per cent., in the healthy arm up to about 38 per cent. Therefore what we call hemiplegias are not hemiplegias at all, accurately speaking, but paraplegias with a preponderating crossed paralysis.[2]

In bilateral hemispheric lesions we observe very characteristic appearances upon the part of the motor cranial nerves, as a result of their bilateral innervation. Since each lesion affects fibers for both sides, a bilateral deficiency of innervation must result, which would remain latent, except that under these circumstances there is nothing to hide it. A glance at Fig. 318, p. 829 (supposing a coincident existence of lesions x and z), will make this clear. If these bilateral hemispheric areas involve especially the motor fibers, they may by summation cause a marked bilateral paralysis of the motor cranial nerve which would not be much paralyzed were the trouble unilateral. Since bulbar nerves (like the motor trigeminus, accessory, hypoglossus, and upper branch of the facial) are affected, the clinical picture will closely simulate the paralyses originating in the nuclear regions of the oblongata and the pons. The symptoms of this summation of hemiplegias are, therefore, spoken of as *pseudobulbar symptoms*. Pseudobulbar symptoms may be occasioned either by two hemispheric lesions coming on simultaneously, or by the addition of a fresh lesion of one hemisphere to an old hemiplegic lesion of the other side.

Similarly, in complete conformity with the theory of bilateral innervation, experience teaches that as soon as a hemiplegia of the other side is added to the first hemiplegia of patients who have retained but slight residual effects of such a hemiplegia, the former affected side promptly becomes more seriously paralyzed. This, together with the explanation furnished upon p. 831, could account for the fact that the bilaterally innervated muscle territories (in the arm the flexors, in the leg the extensors) overpower their antagonists and give rise to contractures in descending degeneration.

III. CEREBRAL DISTURBANCES OF SENSATION.

The sensory tract in the brain may be involved in a number of entirely different locations, as a clinical result of which there will be a more or less distinctly unilateral disturbance of sensation. A portion of the cerebral sensory tract, however, is so diffused that a circumscribed lesion producing marked sensory disturbances can be situated only in certain locations. These places are found exclusively in the compact portion of the sensory tract which is formed by the fillet. The sensory fibers of the fillet arise from the nucleus gracilis and nucleus cuneatus in the medulla, partly cross to the opposite side in the so-called decussation of the fillet, run through the tegmentum of the crus cerebri to the ventral nucleus of the optic thalamus, and finally reach the parietal lobe by passing through the

[1] Cited by Pierre Marie, *Leçons sur les maladies de la moelle*, 1892, p. 26.

[2] The rules proposed by Mann in regard to the behavior of the elongators and shorteners of the leg frequently apply in spinal cord lesions which partially interrupt the motor conduction upon both sides (incomplete crossed lesions, motor systemic diseases). Perhaps this peculiarity may be explained by assuming that, in addition to their bilateral innervation, the elongators possess other more favorable conditions of innervation—*e. g.*, possibly a greater number of fibers in their conduction tracts.

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posterior third of the internal capsule.[1] Hemianesthesia may consequently be observed as the result of lesions: in the posterior columns of the medulla; in the pons where the region of the fillet is affected; in the tegmentum of the crus, when the legion is situated between the red nucleus and the substantia nigra; in the subthalamic region and the adjacent posterior portion of the internal capsule; in the fibers of the corona radiata proceeding from the optic thalamus; and in extensive lesions of the parietal lobe, particularly when they involve the inferior parietal lobule and the central convolutions.

Cerebral hemianesthesia is most frequently dependent upon a lesion in the posterior portion of the internal capsule. Milder degrees of sensory disturbances may originate in the most varied locations in the brain, dependent upon the diffusion of that portion of the sensory tract which is not contained in the fillet, but little typically is known about them.

The Character of Cerebral Hemianesthesia Produced by Anatomic Lesions and Its Differentiation from Hysterical Hemianesthesia and Spinal Hemianesthesia.—The cerebral hemianesthesia due to anatomic causes is characterized by the fact that it involves the spinal sensory tracts together with the cutaneous area supplied by the trifacial; in some cases the optic tract may also be involved and, in rare instances, the acoustic tract. Involvement of the optic tract occurs when the lesion affects the sensory fibers in the region of the posterior portion of the internal capsule or those in the corona radiata of the optic thalamus, when it involves the optic radiation passing through the subthalamic region from the pulvinar and external geniculate body to the cerebral cortex, or when it implicates the primary optic centers themselves. The acoustic tract is affected when the lesion involves the internal geniculate body and posterior corpus quadrigeminum, or the fibers which connect these structures with the temporal lobe. Visual disturbances in cerebral hemianesthesia dependent upon anatomic causes are always hemiopic in character, since the visual center is cut off from the retinal halves corresponding to the side of the lesion. Hearing is simply impaired and never abolished upon the side opposite to the lesion, since each acoustic nerve is connected with both temporal lobes. Smell and taste are always intact, because the lesion is at some distance from the olfactory and gustatory fibers. These fibers from each side are connected with both hemispheres and do not run in a compact bundle, so that it will be readily understood that they could not easily be destroyed by a circumscribed lesion. Cerebral hemianesthesia is further characterized by the fact that all of the sensory powers may be involved—touch, pressure, pain, temperature, bone-sensation, the perception of the position and of passive motions of the extremities, and stereognostic sensation, although they are usually very incompletely affected. The perception of the position and of passive motions of the extremities and the stereognostic recognition of objects are most affected and may be completely abolished, while the sensations of touch, pain, and temperature are usually simply diminished. The disturbance is most marked at the distal ends of the extremities, possibly because this region is supplied by a comparatively larger number of crossed fibers from the opposite hemisphere, while the proximal portions of the extremities are presumably supplied by fibers from both hemispheres. If the cause of the hemianesthesia be situated in the pons or in the uppermost portion of the medulla, a so-called alternating hemianesthesia may be produced, since the sensor trigeminus is involved below its decussation and its sensation is abolished upon the side opposite to the anesthetic extremities. The hemianesthesia frequently extends somewhat beyond the median line of the body.

Since the cerebral hemianesthesia is crossed—*i. e.*, situated upon the side opposite to the lesion—it must be assumed that the sensations for one-half of the body

[1] The cerebral course of the sensory fibers from the anterolateral columns of the cord (see Fig. 362, page 921, and Fig. 355, page 907), which in physiologic and pathologic importance are second only to those of the posterior columns, is as yet unknown. Clinical experience would lead us to suppose that at least some of these fibers become associated with those of the fillet.

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are received by the opposite half of the brain. Since a triple decussation is improbable, we are led to suppose that the cerebral decussation (occurring chiefly in the decussation of the fillet) involves only those fibers which do not decussate at lower levels. This fact explains why the complicated conditions of spinal hemianesthesia (see p. 904 *et seq.*) are absent in hemianesthesia of a cerebral type.

Hysteric hemianesthesia differs from anatomic cerebral hemianesthesia in that the higher senses are usually involved, not only sight and hearing, as may be the case with a cerebral lesion, but also smell and taste. The visual disturbance is amblyopic rather than hemiopic, and consists of diminished sharpness of vision, together with narrowing and weakening of the visual field upon the hemianesthetic side. The auditory phenomena may consist of a complete unilateral deafness, such as does not occur in anatomic cerebral hemianesthesia. It must, nevertheless, be noted that the higher senses may not be involved in hysteric hemianesthesia. It is rather characteristic of this form of hemianesthesia that the most markedly disturbed sensation is that of pain.

Sensory Disturbances with Cortical Lesions.—Sensory disturbances with cortical lesions require particular consideration, since there is a conflict of views in reference to this subject. As the sensory terminals extend over a very large cortical area and are more diffused than the terminals of the motor tract, it is apparent that a localized cortical lesion can produce only a partial disturbance of sensation. These sensory disturbances are most marked in lesions situated in the so-called motor region and in lesions of the parietal cortex. These areas receive the termination of the thalamic radiation which conducts the fibers of the fillet to the cortex. It is likely that sensory fibers of different qualities terminate heterogeneously in both these areas, and that the location and extent of the pathologic lesion is indicated by the degree rather than by the nature of the sensory disturbance. The character of the sensory disturbances in lesions of the so-called motor area has a special practical interest, since such disturbances play an important, though still somewhat uncertain rôle in the localization of these lesions. In the first place it should be noted that sensory disturbances are not constantly observed with lesions of the motor area From what has previously been said, we are led to suppose that they are absent when the lesion is quite circumscribed. In large lesions of the motor area, however, the motor symptoms are usually accompanied by sensory disturbances. They are almost always most pronounced and sometimes present exclusively in the distal portions of the extremities, particularly in the terminal phalanges of the hand. This peculiarity, which has been previously referred to as characteristic of cerebral hemianesthesia in general (p. 879), is probably due to the fact that the proximal portions of the extremities receive sensory fibers from both hemispheres. It is well known that the analogous statement is true in reference to motor disturbances (see p. 878). A further characteristic of cortical disturbances of sensation is that they affect those sensory functions which are dependent upon the correlation of sensory impulses rather than upon the impulses themselves. The sensations for pain, touch, and temperature are usually but slightly if at all affected, while there are distinct disturbances of the perception of the position and of passive movements of the extremities, as well as impaired localization of the sensations of touch and pain. This correlation of sensory impulses is a complicated function of the cortex, and cortical lesions consequently may give rise to these peculiar symptoms, although the elementary sensations are fairly well preserved.

IV. VERTIGO.

Vertigo is a peculiar pathologic phenomenon which plays a great rôle in neurology and is characterized clinically by a feeling of uncertainty as to the position of the body. With this there is physiologically associated the discomfort attendant upon disturbed equilibrium; in severe cases there are also motor disturbances of equilibrium. Other physiologic sequelæ of vertigo are the apparent

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movement of objects,[1] due chiefly to the involvement of visual perceptions (*rotary vertigo*) and affections of other sensorial perceptions, particularly of the sensation of contact between the soles of the feet and the floor (oscillation of the floor). More remote sequelæ, produced by reflex action, are nausea and vomiting, a sense of faintness, palpitation, and even loss of consciousness.

The Pathogenesis and Clinical Significance of Vertigo.—The origin of vertigo, in many instances, is to be sought in the function of the semicircular canals of the labyrinth. At the present time it may be regarded as established that the function of these structures is to acquaint the individual with the position of his body in space. The sensory tract involved is the vestibular nerve, which innervates the semicircular canals; the other sensory nerves are not involved in this function. The perception of equilibrium is simply a special instance in which this function is employed, and the author does not believe we are justified in regarding the semicircular canals as the mechanism of a special "sense of equilibrium" or of a "static sense." It would seem more correct to designate them as an apparatus for the perception of space. It is equally erroneous to speak of the cerebellum as the organ of equilibrium, simply because it is the first central station for the impulses coming from the semicircular canals. Equilibration is but one of a number of functions of the cerebellum. The cerebellum serves rather for general orientation in space and for the adaptation of motor innervation to the position of the body; as the first central station it receives and conducts to higher levels the impulses from the vestibular nerve, and from the direct cerebellar and Gowers's tracts. The peculiar and wonderful arrangement of the mechanism for the perception of space need not be discussed in detail; it is enough simply to point out that the three semicircular canals are arranged in the three planes of space and surely are concerned with orientation, since every movement of the body—*i. e.*, the labyrinth—is analyzed into its three components, each of which exerts an exciting influence upon the nervous elements of one of the semicircular canals. The exciting influence is doubtless due chiefly to the displacement of the endolymph upon the walls of the labyrinth, which occurs with every movement as a result of the inertia of rest. But since we are conscious of our position even during rest, it must be assumed that static pressure effects may also act as the exciting influence which serves as a basis for the perception of space. The nervous impulses arising in this manner are first conducted to the cerebellum by that portion of the auditory nerve called the vestibular nerve. The apparent double function of the auditory nerve seems to justify Ewald's suggestion that it be named the nervus octavus; we should not elevate the vestibular nerve to the dignity of a special cranial nerve for the reason that the current terminology of these nerves is too firmly fixed in our literature. The nervous impulses received by the cerebellum are possibly metamorphosed and then conducted to the cerebrum.

From these physiologic facts it will be understood that a sensation of vertigo always arises when there is a contradiction between the nervous impulses from the semicircular canals and the position of the body (*i. e.*, the labyrinth) as indicated by the other sense-organs. This contradiction causes the symptoms of vertigo—uncertainty of judgment, together with the resulting consequences for the motor innervation of the body, particularly for the maintenance of equilibrium, concomitant discomforts, and the characteristic secondary reflexes. In this manner, the majority of the clinical symptoms of vertigo are easily explainable.

Vertigo occurs particularly in affections of the labyrinth which paralyze or irritate the end organs in the semicircular canals and thus lead to a pathologic distribution of the exciting influence and to a consequent faulty perception in reference to the position of the body. The classical example is Menière's disease, in which the violent pathologic excitation of the semicircular canals produces such marked vertigo that the patient cannot maintain the erect position. In some of these cases the sensorial perception of space may be suddenly and

[1] As we shall subsequently learn (p. 882 *et seq.*) this may also be the cause of the vertigo.

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completely abolished, and this may result in an interruption of some of the association-fibers so that unconsciousness is produced. The vertigo which is observed with innocent middle-ear affections is also to be referred to an associated involvement of the labyrinth, either in the form of circulatory disturbances or of variations in the intralabyrinthine pressure. All these cases are characterized as otogenic on account of the simultaneous disturbances of the acoustic function of the auditory nerve, disturbances which take the form of either impaired hearing or the presence of subjective auditory sounds. In these cases, conditions either of excitement or paralysis within the semicircular canals may produce the phenomena of vertigo if all the semicircular canals are not equally affected.

The vertiginous symptoms which give such a characteristic stamp to cerebellar disease are evidently due to the fact that the stimuli for the sensorial perception of space are correctly originated but not properly conducted, becoming blocked in the cerebellum. The so-called cerebellar ataxia, so frequently observed associated with vertigo in cerebellar disease, is made up of two components. One of these has nothing whatever to do with the sensorial perception of space, but is associated with that disturbance of muscle-tones which Luciani has demonstrated in cerebellar lesions. The other component is to be regarded as the motor effect of the vertigo—*i. e.*, the result of the defective correction of movements on account of faulty sensorial perceptions of space (see p. 756). Since the sensorial perceptions of space are prepared in the cerebellum, but do not become conscious sensations and concepts until they reach the cerebrum, it will readily be understood that affections of the cerebrum, as well as those of the cerebellum, may lead to vertigo. The frequent and marked clinical similarity between tumors of the frontal lobe and those of the cerebellum indicates that the frontal lobe contains centers which receive the sensorial perceptions of space by means of fibers radiating from the cerebellum and passing through the superior cerebellar peduncles and red nucleus.

The vertigo which occurs as a functional symptom of circulatory disturbances, cardiac diseases, arteriosclerosis, anemia, etc., is possibly due to abnormal excitation of the semicircular canals or of their co-ordinated centers. The same is true of neurasthenic vertigo.

The vertigo associated with paralysis of the ocular muscles is evidently due to a false projection of the retinal images, as a result of which disturbances of space-orientation might naturally give rise to vertigo. These disturbances of space-orientation are due not only to the occurrence of double images but also to the fact that these retinal images become abnormally displaced during ocular movement—*i. e.*, they do not conform with the motor impulses, so that the objects apparently seem to move. In unilateral ocular paralysis the vertigo may be controlled to a variable degree by having the patient concentrate his attention upon the image produced in the sound eye and disregard the faulty projection of the image in the paralyzed organ. Even in bilateral paralysis of the ocular muscles the vertigo may be eliminated if the patient can learn to disregard the visual sense in the formation of his conceptions of space. In all cases the vertigo produced by paralysis of the ocular muscles may be made to disappear by closing either one or both eyes.

A special variety of vertigo, partly also of ocular origin, is that which is experienced upon *rapid rotation of the body* or upon the sudden arrest of a passive movement involving the body, such as the stopping of a train. In the first instance the phenomenon is probably dependent upon the fact that a physiologic excitation is produced in the semicircular canals by the motion imparted to the body, which results in reflex stimuli to the ocular muscles so that the eyes follow external objects in spite of the movement of the body. When the particular motion is very rapid, the excitation of the semicircular canals becomes so strong that the reflex stimulation of the ocular muscles is excessive; since these ocular movements are unconsciously executed, the objects themselves apparently move and vertigo is consequently produced. The inability to fix the eyes permanently upon any object, together with the violence and rapid change in the excitation

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of the individual semicircular canals may also be a factor in producing the disturbance of space-orientation in this variety of vertigo. The vertigo which is experienced by many individuals upon the sudden stopping of a train is probably due to the fact that the unconscious reflex ocular movements, produced through the agency of the semicircular canals, are continued somewhat longer than the motion of the train, and the apparent motion of external objects results in vertigo as before.

Mountain vertigo, or the vertigo of elevation, is not a pure vertigo, but is partly due to a sensation of fear. The other factor is probably an ocular vertigo dependent upon a strong concept of falling, which by autosuggestion innervates the nervous mechanism of space-orientation to an abnormal degree.

Agoraphobia.—Vertigo produced by the fear of space is brought about by an analogous mechanism which is excited by autosuggestion.

Seasickness is probably nothing else than a violent vertigo with numerous irradiations (to the vomiting center, etc.), produced by pathologic excitation of the semicircular canals as a result of the oscillations of the ship. Ocular vertigo plays an important rôle in seasickness and originates in the labyrinth, just as is the case in the vertigo produced by rapid rotation of the body or upon the sudden stopping of a train. This is borne out by the fact that a large number of the phenomena of seasickness disappear when the eyes are closed. Since the symptoms by no means completely disappear, however, it is quite likely that a further cause is to be found in the inability of the mind and the movements of equilibration to follow the rapidly changing and violent excitations of the semicircular canals.

Galvanic vertigo, produced by the application of a galvanic current to the head, is doubtless dependent upon a direct and unequal excitation of the nerve-terminals of the semicircular canals, which does not correspond with the position of the body.

The existence of the so-called *gastric vertigo* of Trousseau (vertigo e stomacho laeso) in affections of the stomach is doubtful. The majority of the cases answering to Trousseau's description have been shown to be other varieties of vertigo (particularly aural vertigo), the origin of which were unknown in the time of this distinguished clinician. In many such cases the only symptom suggestive of gastric disease is the vomiting produced by the vertigo.

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V. CEREBRAL LOCALIZATION.

Without intending to enter minutely into the question of cerebral localization, which belongs more properly to the province of special diagnosis, the author wishes to submit the following diagrams to be utilized as a sort of guide in the problems of cortical and nuclear diagnosis:

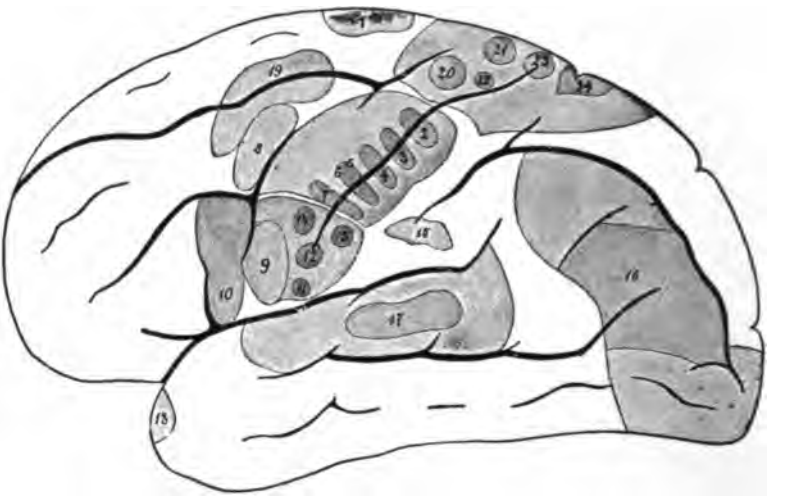

FIG. 339.—Cortical localization: 1, Trunk; 2, shoulder; 3, elbow; 4, wrist joint; 5, the three last fingers; 6, index fingers; 7, thumb; 8, "writing center" (see p. 894) (assumed by French writers); 9, larynx; 10, Broca's speech center (motor); 11, tongue; 12, mouth; 13, lower facial; 14, upper facial; 15, eye muscle; 16, vision; 17, hearing (Wernicke's sensory speech center); 18, taste; 19, conjugate movements of eyes and head; 20, movements of hip joint; 21, movements of knee joint; 22, movements of ankle joint; 23, movements of great toe; 24, movements of little toes (from Debove and Achard).

Compare p. 831 in regard to the dispute which is still going on concerning the central innervation of the conjugate movements of the eyes. Wernicke has assumed that the center for these movements is situated in the inferior parietal convolution, because he found but few projection fibers in this region. He supposes that the conjugate deviation of the eyes, which arises in case of lesions in this area, is due to lesion of an association tract, demonstrated by himself to exist between the visual area and the motor area.

Flechsig disputes this. He locates the "visual center" almost exclusively in the median surface of the hemispheres, especially in the region of the calcarine fissure. The visual disturbances so frequently met with clinically in lesions of this region (16) may be explained partly by the fact that such lesions often also involve the visual fibers situated deeper and connecting the central fibers of the optic tract to the visual center.

According to Flechsig, the center or area for smell in man has allotment both in the frontal and temporal portion of the brain. The former embraces the entire posterior portion of the base of the frontal lobes and the basal portions of the gyrus fornicatus, the latter embraces the uncinate gyrus and a part of the adjoining inner pole of the temporal lobe. Both portions are fundamentally connected with the island.

Flechsig, who has recently investigated the localization of the sense of taste, does not furnish any definite statements about its seat. 18 in the diagram is, therefore, placed provisionally.

Munk locates the region for stomatic sensibility in the territory of the central convolutions and the parietocentral lobule supplied with motor centers (see Flechsig, *Gehirn und Seele*, Leipsic, Veit & Co., 1896). That is to say, these areas contain, in addition to the motor centers, the central apparatus for sensory appreciations in so far as they do not belong to the "higher" senses (sight, hearing, etc.), which possess special centers. The area for corporeal feeling, according to Flechsig, also includes the lobus limbicus—*i. e.*, the gyrus fornicatus, gyrus uncinatus, and the gyrus hippocampus.

The white unshaded areas and the shaded area of the parieto-occipital brain, 16, according to Flechsig wrongly credited with the visual center, contain the Flechsig association centers. He differentiates three, viz., the frontal or anterior, the parieto-occipito-temporal or posterior great association center, and finally the Island of Reil. These areas are anatomically characterized by containing no projection fibers, or very few, and, on the contrary, very many association fibers. Lesions of the anterior association center should cause a loss of interest and a change of character; lesions of the posterior association center, a loss of collective representations, initiation, and mental capacity.

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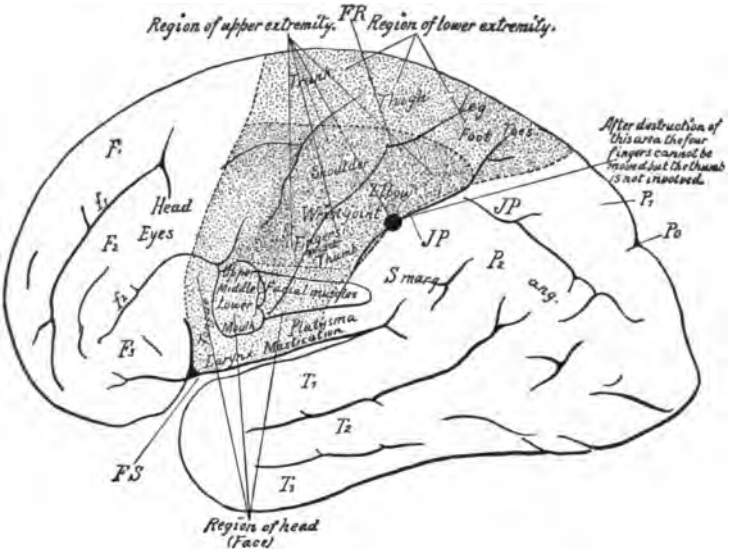

FIG. 340.—The lateral aspect of the human cerebral hemisphere. The motor areas according to Allen, Starr, Keen, Mills, Horsley, and v. Monakow, as arranged by the latter.

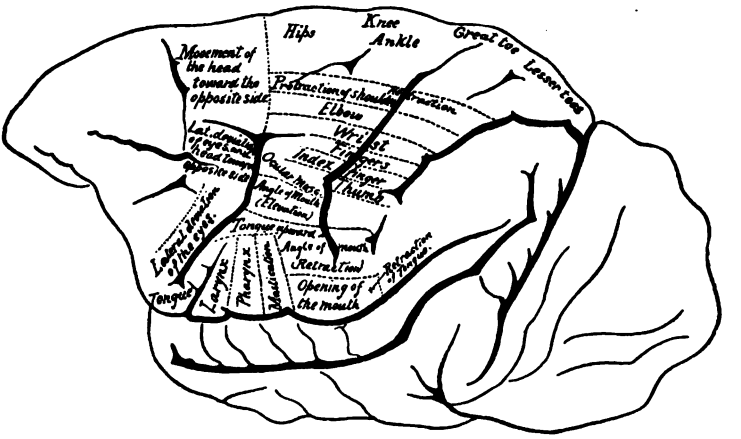

FIG. 341.—The motor areas in the ape, according to Beevor and Horsley.

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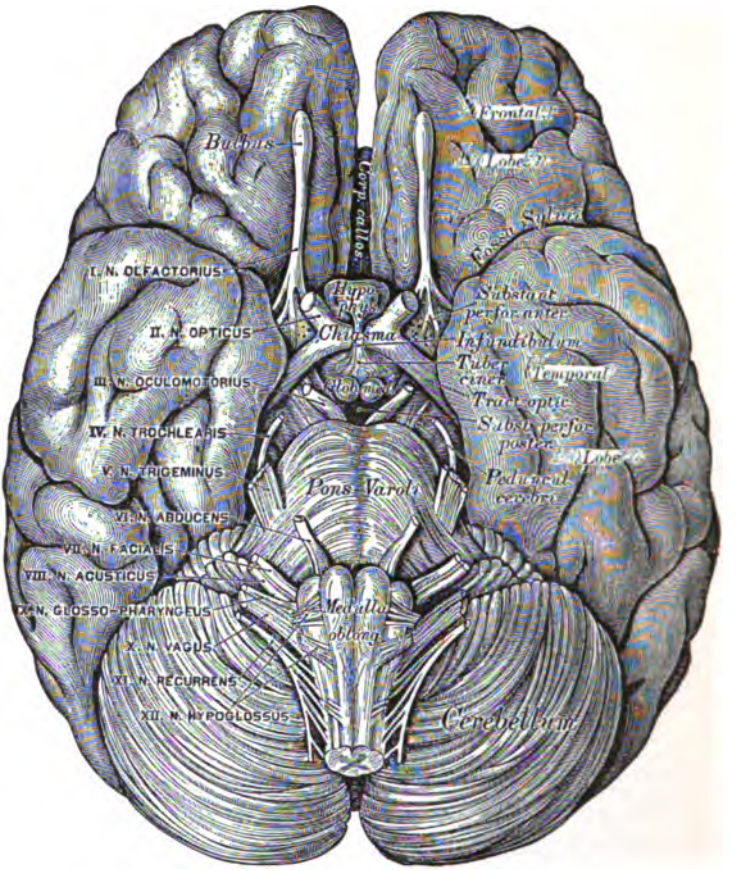

FIG. 342.—Base of the brain showing the origin of the cranial nerves (from Heitzmann).

(See Figs. on opposite page for the following legends):

FIG. 343.—Transparent surface view of the medulla oblongata from behind. Upon the right side the nerve nuclei are put in diagrammatically and numbered with Roman figures: *V*. Motor nucleus of the trigeminus; *V'* and *V''*, middle and lower sensory nuclei of the trigeminus. *VI*, abducens nucleus; *VII*, facial nucleus; *VIII*, posterior median acoustic nucleus; *VIII'*, anterior median acoustic nucleus; *VIII''*, anterior lateral acoustic nucleus; *IX*, glossopharyngeal nucleus; *X*, vagus nucleus; *XI*, accessory nucleus; *XII*, hypoglossal nucleus; 1, brachium pontis; 2, brachium conjunctivæ; 3, cerebellar peduncle; 4, eminentia teres; 5, striæ acousticæ; 6, ala cinerea. The large numerals represent the corresponding nerve roots (Erb).

FIG. 344.—Transparent lateral view of the right half of the medulla oblongata. To show the relations of the most important nuclei. The nuclei situated nearest are shaded darker (diagrammatic): *Py*, Pyramidal tract; *Py.Kr*, decussation of pyramidal tract; *O*, olive; *O.s.* superior olive; *V*, motor nucleus of trigeminus; *V'*, middle sensory nucleus of trigeminus; *V''*, lower sensory nucleus of trigeminus; *VI*, nucleus of abducens; *G.f.*, knee of the facial; *VII*, facial nucleus; *VIII*, posterior middle acoustic nucleus; *IX*, glossopharyngeal nucleus; *X*, vagus nucleus; *XI*, accessory nucleus; *XII*, hypoglossal nucleus; *Kz*, nucleus gracilis; *RV*, trigeminus root; *RVI*, abducens root; *RVII*, facial root (Erb).

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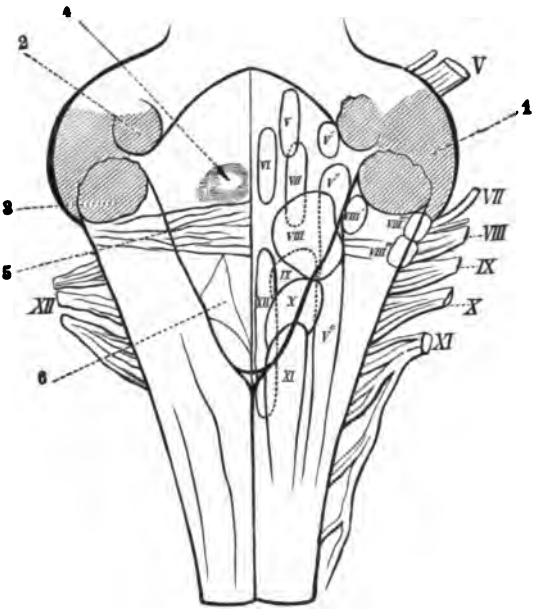

FIG. 343.

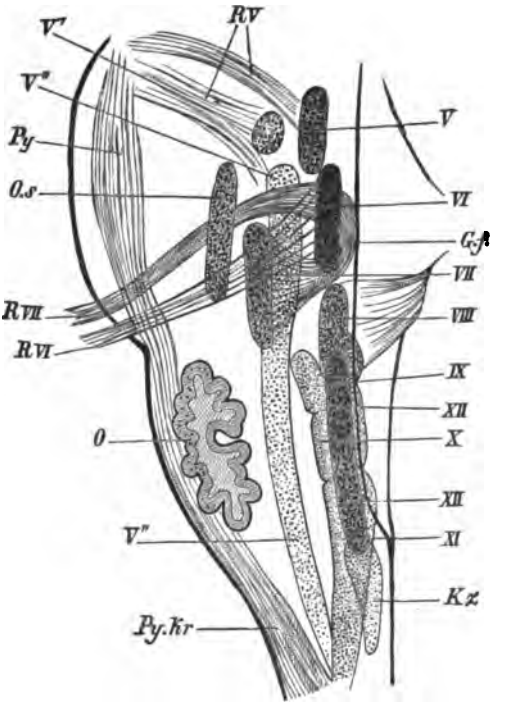

FIG. 344.

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VI. THE DISTURBANCES OF SPEECH.

1. THE CONCEPTION OF THE SPEECH TRACT.

Speech arises from the elaboration of motor speech conceptions in the so-called motor speech center situated in the left hemisphere. When the will is exerted, these conceptions are responsible for the co-ordinated impulses proceeding through the so-called central speech tract to the bilateral cortical centers for the muscles of speech and generating the spoken word.

The speech tract (Fig. 345) originates in the motor speech center, the so-called Broca's area (*a*, Fig. 345), runs to the cortical centers for the oral, laryngeal, and respiratory muscles, some of the fibers possibly passing through the corpus callosum, and finally enters the pyramidal tract to reach the nuclei for the muscles of speech. From the cortical centers to the nuclei of these muscles, the impulses travel through the same fibers that are utilized for the other functions of the muscles, so that an actual special tract does not exist after the impulse leaves the cortical centers.

2. THE DISTURBANCES OF SPEECH FROM LESIONS OF THE SPEECH AREA OR OF THE CONDUCTING FIBERS.

The innervation for the movements of speech may be interrupted by a lesion situated in any portion of its course. The clinical picture is subject to great variation, according to whether the lesion affects the speech center, the actual speech tract (the heavy line in Fig. 345), or simply the connection of the cortical centers of the muscles of speech with the nuclei of the latter (the light lines in Fig. 345). In the first instance, even though the lesion be circumscribed, the ability to form words will be more or less completely abolished. When the lesion affects the connection between the cortex and the nuclei of the muscles, however, the only result is a mutilation of the syllables, a disturbance of pronunciation; if the lesion be of moderate extent the disturbance is but slight, since owing to the commissural fibers the speech impulse is conducted downward from both hemispheres. As a result of the bilateral quality of this portion of speech innervation, the disturbance of pronunciation can be extreme only when the lesion is situated low down in the vicinity of the nuclei for the muscles, where the fibers of both hemispheres are simultaneously more or less involved.

The disturbances having as their cause some lesion in the speech center or in the actual speech tract of the left hemisphere are called aphasias; those due to some lesion between the cortex and the nuclei for the muscles of speech are called anarthrias.

Although in typical cases the clinical distinction between these two conditions is quite marked, the differentiation between them is not sharp, since lesions of the speech center and of the actual speech tract may give rise to symptoms resembling anarthria, providing that the lesion is neither large nor destructive (see Anarthria). Such a disturbance, which might be called a "central anarthria," may, nevertheless, be differentiated from a peripheral anarthria, and particularly from that localized in the vicinity of the nuclei, by the fact that in the latter the other functions of the muscles of speech (deglutition, mastication) are also involved.

The sharpest differentiation between aphasia and anarthria consequently consists of the fact that the former is a pure and exclusive disturbance of speech, while the latter is always accompanied by a disturbance of the other functions of the muscles of speech.

(*a*) Anarthria.

In anarthria there is an affection of individual fibers of the speech tract in the neighborhood of the nuclei. Hence the following peculiarity: The speech impulse is correctly formed in the speech area and correctly despatched to the periphery; but, as the result of partial interruption of conduction in the neighborhood of the nuclei, each of its ganglion cells no longer receives the measure of

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innervation essential to co-ordinated speech. The result is a disturbance in the co-ordination of the speech, analogous to the ataxia of the extremities in partial paralyses (see p. 753 *et seq.*), so that a patient with anarthria pronounces the word with approximately correct cadence of syllables and intonation, but with some of the letters of the word lacking or else incorrectly pronounced. Anarthria is, therefore, a disturbance of pronunciation, which is further characterized by the fact that the more or less marked paralysis of the muscles of speech also affects the other functions of these muscles.

It is evident that quite an analogous disturbance of speech will arise, when not the terminal fibers of the speech tract in the neighborhood of the nuclei, but the nuclei themselves or the peripheral speech nerves—*e. g.*, facial and hypoglossus—are affected. In such cases speech will be mutilated and pronunciation

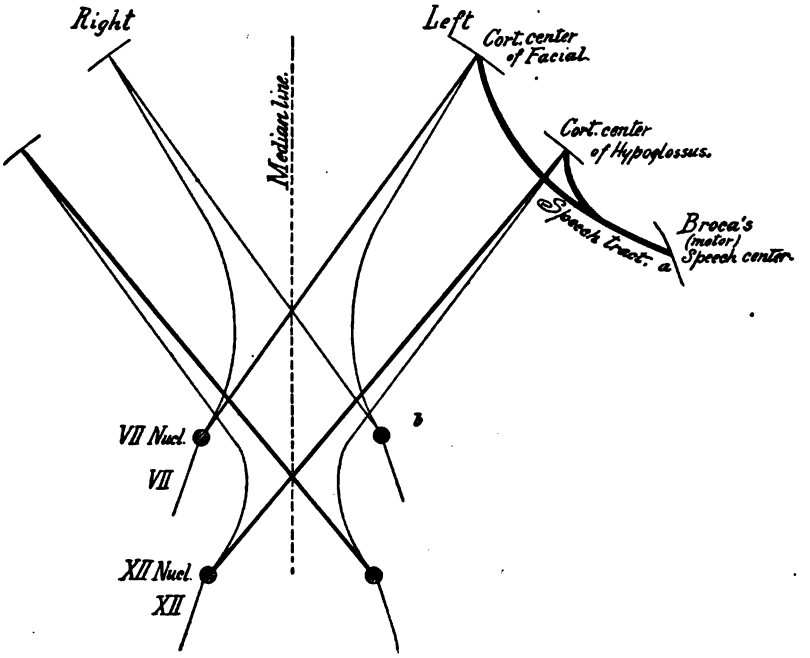

FIG. 345.—Diagram of the motor speech tract. For simplicity, of the nerves which subserve the function of speech, only the facial (*VII*) and hypoglossus (*XII*), are shown. For the center for spontaneous breathing, as well as that for the larynx, see Fig. 340; and for the motor trigeminus, especially for the part that presides over the movement of the jaws, see "Chewing," Fig. 340. Aside from this, there are left out in the figure the commissural fibers coursing in the corpus callosum, which unite the two lateral cortical centers.

will suffer. These nuclear and peripheral disturbances of speech parallel those of supranuclear anarthria, insomuch as they are associated with paralyses of other movements.

The clinical picture of anarthria may under some circumstances result even from an incomplete interruption of the speech center or of the actual speech tract. This can be explained only by assuming that the separate fibers or cells are to a certain extent individually diseased, because the fibers of the central speech tract are so compact that larger lesions would generally affect them *in toto*. Anatomic proof of the existence of pure anarthria depending upon such individual disease of separate fibers or cells has not yet been furnished. Anarthritic disturbances of pronunciation, which often persist for a long time after recovery from aphasia, may without doubt be explained by the fact that certain individual fibers or cells of the central speech apparatus, which is at first widely involved, later on

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recover, while certain others do not. In the same way manifold combinations of anarthria and aphasia, which have been sometimes observed, may perhaps be attributed to an unequal involvement of the central speech apparatus. As has previously been stated, the characteristic of all these cases of central "anarthria" is that the muscles perform all their movements except those necessary for the production of speech.

The simplest method of examining for anarthria is to have the patients pronounce the letters of the alphabet in their order, and then combinations of letters—*i. e.*, simpler and then more complicated words. Theoretically, it is evident that anarthria will occur very frequently in all lesions in the neighborhood of the nuclei of the speech muscles—*i. e.*, in hemorrhagic and softened areas and in tumors of the pons and of the oblongata, but especially in progressive bulbar paralysis.

(*b*) Aphasia (Agraphia; Alexia).

As contrasted with anarthria, we may define aphasia as embracing those disturbances of speech which arise from diffuse lesions of the actual speech tract or of the motor speech center itself. If we could represent the latter as merely a simple center, the process would be comparatively simple; a loss of speech would then depend upon a destruction of the center (Fig. 345, *a*), or upon a destruction of the speech tract (heavy line, Fig. 345).

The conditions of aphasia are, however, more complicated, because the speech

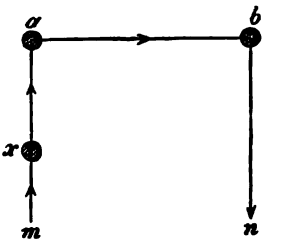

FIG. 346.—Primitive speech apparatus of the child for mechanical repetition of words: *a*, Sensory speech center; *b*, motor speech center (Broca's center); *x*, acoustic center, center of simple sound perception; *mx*, tract of the auditory nerve; *bn*, motor speech tract.

center is, in a broader sense of the word, not a simple motor center. We may consider the entire apparatus of central speech formation, which has been established psychophysiologically by the researches of Wernicke and Lichtheim, as proceeding centrally from the point *a* of Fig. 345, and this latter point as representing merely the motor terminus of the central speech mechanism, the so-called motor center of speech. Lesions of these portions of the speech apparatus, which have not yet been studied, can also give rise to disturbances properly designated as *aphasias*. To understand these different forms of aphasia, a knowledge of the physiologic mechanism of central speech formation is, therefore, required. A discussion, essentially following Wernicke and Lichtheim, will now be given, going somewhat more minutely into detail.

When a child learns to speak, the sound of the word heard is sent by way of the acoustic nerve to his left first temporal convolution in the cortex (sensory speech center, Fig. 346, *a*). The child then attempts to imitate this word. This latter procedure may be explained by assuming that, by means of association, the auditory images exert a conception of movement corresponding to the spoken words. This representation of movement, as Broca proved, is situated in the left inferior frontal convolution (motor or Broca's speech center, Fig. 339, 10). The child's primitive speech apparatus (Fig. 346) is formed in this way, and he thus repeats mechanically the word spoken beforehand. *a* represents the center for auditory images of the word heard—*i. e.*, the *sensory speech center* in the first temporal convolution. The auditory images are received here after they have properly

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stimulated the corresponding acoustic center (x), the center of simple hearing or sound perception. The association tract (ab) by which, by mechanica lbabbling of the child, the center b is incited by way of mab, runs, according to Wernicke, along the convolutions of the Island of Reil from behind forward. The arrow pointing upward to x represents the tract of the auditory nerve; the arrow pointing downward from b represents the motor speech tract represented in detail in Fig. 345.

The child's advance to that stage of development which is characterized by voluntary speech is attained through the association of centers (a) and (b) with ideas. The idea of an object is the aggregate of its partial representations. After the partial representations are acquired by experience, they are stored in the different sensory areas of the cerebral cortex, and are then associated as memories.

The conception of an object can, therefore, never be localized at a single point in the cerebral cortex—*e. g.*, the conception "bell" requires association tracts to quite different parts of the brain—to the acoustic, optic, and tactile centers. This is represented in the more complicated diagram (Fig. 347). The partial representations $c + c' + c''$ first produce the conception of bell.[1]

To simplify the speech scheme, we reduce the conception $c + c' + c''$ to a

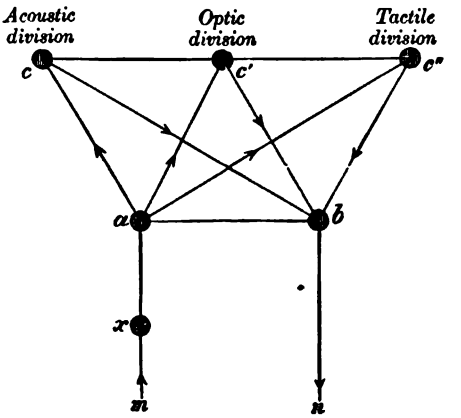

FIG. 347.—Apparatus for conscious speech and speech comprehension, with the relationship to the primitive speech center to conceptions, especially the sensory division of the latter. For simplicity merely, three divisions of the cortical sensory areas have been represented. The letters a, b, x, m, and n have the same meaning as in Fig. 345.

single point (c), using the diagram that Wernicke first made for voluntary conscious speech (Fig. 348). For the sake of simplicity, the acoustic center (x) is omitted here and in the following diagrams, because, as is shown upon p. 892, note, from a gross anatomic standpoint it practically coincides with the center a. The significance of the double arrows will be explained in what follows.

With the aid of this diagram we can readily explain many types of aphasia by supposing interruption of conduction at different places. We differentiate between motor and sensory aphasia according to whether the interruption affects the sensory centripetal (maC), or the motor centrifugal conducting portion

[1] To avoid misunderstanding, the author should add that the conception which a child has of a bell and associates with the word "bell" is a natural but not the complete conception of a "bell" which an adult possesses. A complete conception will only be developed gradually, in the course of mental development, by adding one partial representation to another. Thus, in small children, the conception of a "bell" may be exclusively the partial representation of the tone of a "bell" and perhaps of some of its metallic luster; then, in the course of development, are superadded the representations of the form of the "bell," of the sensation of cold by touching it, of its use, and of many other features to complete the conception.

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(*Cbn*). Aphasia arising from interruption of conduction in the line *ab* is spoken of as "*conduction*" or, perhaps better, "*associated aphasia.*" Disturbances between *ab*, on the one hand, and *C*, on the other, are called *transcortical*, those in *a* or *b* *cortical*, and those peripheral to *a* or *b* subcortical. These names have not been fittingly chosen, since the entire area of innervation (*abc*) is situated in the cerebral cortex,[1] and should, consequently, be designated as cortical. If the terms had not become so fixed, the author would suggest replacing them by he expressions transcentral, central, and subcentral, the word "central" naturally referring to the speech center.

To explain these different symptom-complexes, we must now call attention to the fact that, for correct speaking, the tract *Cab* must be intact, as well as the tract *Cbn*, although this does not directly appear in the diagram. If the tract *Cab* is interrupted at any point, word production is still possible by way of the tract *Cbn*; the fund of words is sufficient, but we observe a symptom called *paraphasia—i. e.*, a confusion of words—because the control of motor innervation by way of the path *Cab* is essential for correct speaking. Since this paraphasia may also be caused by lesions between *C* and *a*, it must be assumed that not only centripetal but also centrifugal conduction, requisite for the control mentioned above, is situated in the association fibers *aC*. Words are sounded inwardly.

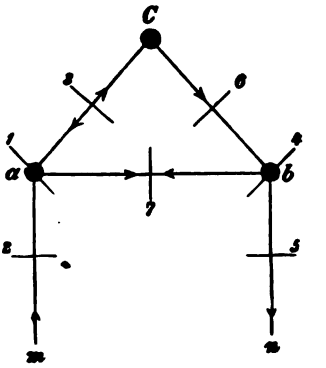

FIG. 348.—Simplification of the diagram of conscious speech (Fig. 346) by restriction of the conception to point *c* and the omission of the acoustic center *x*. The letters have the same meaning as in both preceding diagrams. The figures correspond to the list of aphasias in the text.

The double arrows in Fig. 348, between *a* and *C*, represent this idea; but it must be confessed that the innervation from *C* to *a* is not sufficient to speak anything by way of *Cab*, as otherwise no loss of speech would occur with a lesion between *b* and *C*, and experience proves that this is not the case.

The following main types of aphasic disturbances should now be easily understood. The numbers represent the point of interruption as indicated in the diagram (Fig. 346):

1. *Cortical sensory aphasia.* Comprehension of speech and repetition of speech are prevented. Paraphasia is present.

2. *Subcortical sensory aphasia*[2] (pure "word-deafness"). The same functions are interfered with as in 1, except that there is no paraphasia.

[1] This is demonstrated by the fact that the lesions producing aphasia are always situated in the cortex or in its immediate vicinity. At most the tract *am* (the heavy line in Fig. 345) describes but a slight curve in the white matter in the vicinity of the cortex.

[2] The anatomic findings in this form of aphasia have always shown, in contradiction to the term subcortical, foci of disease in Wernicke's sensory speech center. (Déjérine, Veraguth). For this reason the entire Wernicke-Lichtheim conception of the speech apparatus has been said, erroneously, to disagree with the facts. In the author's

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3. *Transcortical sensory aphasia.* Comprehension of speech is lost; repetition of speech, as contrasted with 1, is retained. Paraphasia is present.

4. *Cortical motor aphasia.* Spontaneous speech and capacity to repeat are lost. Comprehension of speech is preserved.

5. *Subcortical motor aphasia* (lesion of the speech tract). Clinical picture as in 4. Differentiation between 4 and 5 is discussed below.

6. *Transcortical motor aphasia.* Spontaneous speech is lost. Repetition and comprehension of speech are preserved.

7. *Conduction or association aphasia.* Spontaneous speech is paraphasic. Repetition by way of *aCb* is possible, but also paraphasic.

A differentiation between 4 and 5 can be determined by noticing the written speech. In cortical motor aphasia (4) writing is impossible; in subcortical (5) it is preserved. Further discussion of this point will be taken up later. Lichtheim has demonstrated that a patient with subcortical motor aphasia (5) can show by signs how many syllables there are in the name of an object held in front of him; whereas, with cortical motor aphasia (4), destruction of the center (*b*), this is impossible.

The varied disturbances of writing and reading which occur in aphasia show that the centers *a* and *b* have an almost reciprocal relation, expressed in Fig. 348 by the double arrows, and that the sensory representation and the motor together produce a complete so-called word conception (Wernicke) (see p. 895). In this collective word conception, and not merely in the individual representations (*a* and *b*), are to be found not only the letters, as we shall see, but also to a certain extent the number of the syllables, presupposing an internal speech. Therefore, in case there is an injury in *a* and *b* or between the two, the patient can no longer determine the number of syllables in a word corresponding to some conceived object. Thus far the author believes this phenomenon has been attributed only to lesions in *b*; that is, to cortical motor aphasia. However, if this explanation is correct, it must also occur in cortical sensory and in association aphasia.

In determining the characteristics of an aphasia, we must inquire into written speech—*i. e.*, the ability to write and to read—for this is closely connected with articulate speech.

To explain the psychical mechanism of reading and writing, one cannot do better than to start again with the development of this function in children.

The child learns to read and write by having the optical pictures imprinted upon a center (*a*, Fig. 349), and by learning at the same time to associate with them the corresponding auditory images. This association results from the formation of a tract (*a a*), in which *a*, as in Fig. 348, represents the sensory (acoustic) speech center. By means of such an association, printed or written letters acquire a certain significance to the child. In learning to write, a child forms an association between the optic center (*a*) and a motor center (β), and making use of β, learns mechanically to copy the letters. There first occurs an impression of sensory memory pictures (here by way of the optic), an almost simultaneous

opinion, the only conclusion to be drawn from these findings is that the path *am*, which in the diagram is represented as subcortical, in reality lies in the cortex itself—*i. e.*, that *a* and *x* in Fig. 346, from the gross anatomic standpoint, coincide, and that, from reasons which will be understood from what follows, the subcortical fibers in the cortex must be injured themselves in order to produce a sensory aphasia. The separation of *a* and *x* in the diagram should merely express the fact that sensory disturbances of speech and of hearing are independent of each other; provided that, from the gross anatomic standpoint, we identify the left sensory speech center and the left auditory center. This is explained by the fact that each auditory nerve is supplied by both hemispheres (p. 865). As a result of the arrangement, lesions of the left temporal lobe can be nullified, so far as the auditory function is concerned, by the activity of the right temporal lobe; and lesions of the white substance of the left temporal lobe need not cause sensory aphasia, because the sensory speech center is united with both auditory nerves, and the collective auditory stimulation of the sensory speech center cannot readily be eliminated, as would much more probably be the case if the lesion were situated in the left temporal cortex itself where bilateral subcortical fibers intermingle.

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association of these with acoustic memory pictures, and then a production of movement representations for writing the separate letters. Therefore, we assume that copying letters by a child occurs by way of the tract ($\mu a \beta \nu$).

Thus far we have included in the child's learning written speech only the mechanical copying of letters. Very soon, however, he learns to write letters when merely their auditory images have impressed him, either from dictation or from resounding spontaneously in his mind of his own accord. To explain the process of writing letters voluntarily or from dictation, we must introduce into our diagram still another centrifugal tract, which unites the diagram of writing with that of speaking. We have already in the tract $a\ a$ (Fig. 349) a centripetal connection of the writing mechanism with a sensory speech center; the line ba may represent the centrifugal connection between the motor speech center (b) and the psychical writing mechanism. Wernicke believes that he has proved that this connection runs from b to a, and not more directly from b to β. Writing letters

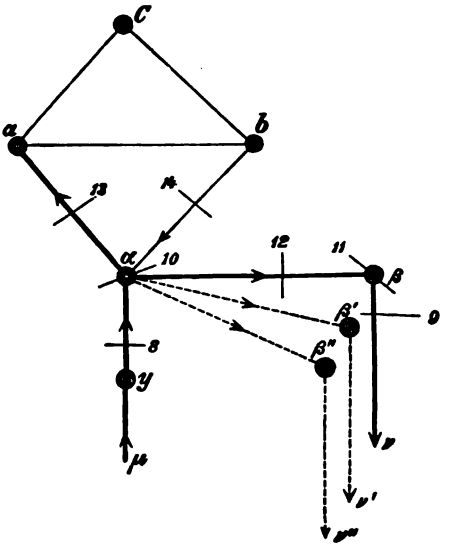

FIG. 349.—Diagram for the mechanism of written speech: a, Center for the optic representation of written or printed letters; γ, center of optic perception (cortical optic center); $\mu\gamma$, optic tract; a, sensory speech center; b, motor speech center; C, conception; β, β', β'', motor centers for writing movements (β for writing with the right hand; β' and β'' for writing with other parts of the body); $\beta\nu$, $\beta'\nu'$, $\beta'\nu''$, motor tracts for writing movements. The numbers 8 to 14 represent the locations of lesions in the so-called isolated alexias and agraphias (see p. 897 *et seq.*). For simplicity only, the heavy lines should be noticed at first in reading the text.

voluntarily or from dictation takes place, then, in this way: a is stimulated by the auditory images of the letter emerging from the mind, and excites in b the representation of movements of the spoken letter, then the optical picture in a, and from there finally the representation of movement of the written letter in β. Where do we find the points a and β in the brain? The center a is apparently to be found in the cortical visual area (Fig. 339, 16), according to pathologic findings probably chiefly in the left hemisphere; this seems quite comprehensible, if we take into consideration the connection between written speech and spoken speech. The point β has oftentimes been supposed to exist as an actual writing center; and Charcot located it in the frontal lobes, near the middle of the anterior central convolution (see Fig. 439, 8). Simple reflection shows that such an individual writing center does not exist. The movement of writing is, as a matter of fact, like any other movement, in case the form of the letter is known; one can write it with any part of the body—for example, the nose or the foot.

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We can go even so far as to place the supposed writing center in the brain of the horse when we describe the form of a letter by riding. As a matter of fact, we write by copying approximately the optical memory pictures of letters, using any sort of movements. Ordinarily, β represents the cortical center of the right hand; it could, however, just as well represent another motor center. The dotted tracts $a\beta'v'$, $a\beta''v''$, etc. (Fig. 349), are intended to express this multiplicity. Including only the essentials of Figs. 348 and 349, a diagram of the central speech mechanism, including reading and writing, may be represented as in Fig. 350.

The following explanation should be added to explain reading and writing entire words. Experience teaches that written speech is always disordered in aphasias if there is an injury anywhere upon the line *ab*, whether it be at *a* or at *b*, or between the two; for, as mentioned upon p. 893, *a* and *b* seem like a unity, which Wernicke has designated as the "substratum of the word conception." Now, it is ordinarily assumed that the word conception is produced from separate letters, that the tract *aab* is traveled by each letter, and that in writing entire words the conception will be dissected, so that an innervation impulse proceeds from *b* to *a* for each letter. Broadbent and Wernicke believe that they have proven beyond question the correctness of this assumption that reading and writing are always accomplished letter by letter, but the point is closely contested. In this connection these authors' original articles should be consulted. We need add only that the great speed with which a practised person runs over the words in reading, and the rapid writing of people who write a good deal, argue in these cases against writing and reading letter by letter.

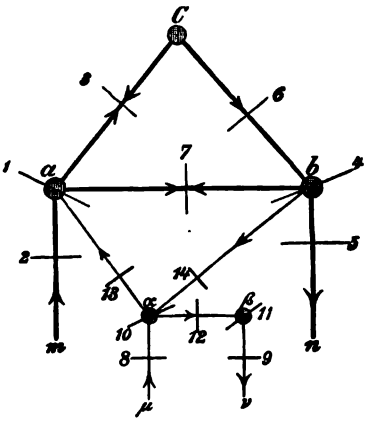

FIG. 350.—Diagram of all the central speech apparatus including reading and writing. The letters and figures have the same meaning as in the two preceding illustrations.

Even if the beginner and the unskilled, as is well known, always read by letters, it is conceivable and perhaps probable that there may be certain men in whose daily routine writing and reading play a large part and whose brains are therefore so especially accustomed to these processes that they no longer read and write by letters, but perceive the optical word picture as a whole, and in writing conceive it so. Illegible handwriting in which individual letters can scarcely be recognized is possibly always read in this fashion of grasping the word as a whole. This coincides with the experience that the illegible handwriting of people who are unaccustomed to write and who therefore write and read by letters, is much more difficult to decipher than the illegible handwriting of those who write without plain individual letters—*i. e.*, to a certain extent in hieroglyphics. Wherever writing and reading no longer have to do with letters but with hieroglyphics, the central mechanism for writing and reading in the brain naturally becomes to a certain degree independent. The center *a*, then, instead of being an optical center for letters, becomes really an optical center for words. It is conceivable that, as it is developed and becomes self-dependent, the centers *a* and *b*, as well as the association tracts (*aa*) and (*ba*), become less necessary for writing and reading, and the word center (*a*) is associated more and more directly with the conception (*C*). Quick writing is, therefore, very much facilitated. But this self-dependence of the writing apparatus never becomes complete, and even when at *a* not only letters but entire optical word pictures are stored, still in writing and reading the territory *aab* continues to be very important. This is proved by the fact that the aphasias 1, 4, and 7 always produce a decided disturbance of writing and reading. It does not seem absolutely essential that the territory *ab*,

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accredited by Wernicke with being the anatomic substratum of the word concept, should be limited to rendering spelling possible; but we can imagine that to a man who has gradually abandoned spelling in reading and writing, and who merely make suse of entire words, the centers *a* and *b* and their connecting tract are, nevertheless, essential for centripetal as well as for centrifugal impressions of the optical word picture. With this conception, it is clear that written speech will always be very decidedly affected with the aphasias situated at 1, 4, or 7. The greater or less development of independence of the optical word center should easily explain the variations in the grade of writing and reading disturbances in aphasias.

After what has been said, it is plain that written speech (including both the reading and writing of words) will always be affected if the word concept (*a* and *b*) is injured in any way—that is, if the lesion is situated at *a*, at *b*, or between *a* and *b*. This applies, on the one hand, just as much to reading aloud as to the comprehension of written speech; and, on the other hand, just as much to spontaneous writing as to writing from dictation. An interference in mechanical copying, in which one letter is pictured after the other, always depends upon a lesion in the territory of the writing arc (*μαβν*). It is worthy of note that, because a lesion between *a* and *b* destroys the word concept, it makes writing quite impossible, or occasions a very marked defect in it, but never leads to paragraphia. The latter is analogous to paraphasia, and produces only a confusion of words in writing. Paragraphia occurs only in transcortical sensory aphasia (see below).

The effects of the different kinds of aphasias on written speech (see p. 892) are as follows:[1]

1. *Cortical Sensory Aphasia.* Lost: voluntary writing to dictation, comprehension of writing, reading aloud. Retained: mechanical copying.

2. *Subcortical Sensory Aphasia*[2]. Written speech is quite unaffected.

3. *Transcortical Sensory Aphasia.* Lost: comprehension of writing. Retained: reading aloud without comprehension, and writing in all its forms. Paragraphia is, however, present in spontaneous writing—*i. e.*, the words are confused, just as in speaking.

4. *Cortical Motor Aphasia.* Lost: all forms of writing and reading, with the single exception of mechanical copying.

5. *Subcortical Motor Aphasia.* Lost: reading aloud. Retained: writing in all its forms, and the comprehension of writing.

6. *Transcortical Motor Aphasia.* Lost: spontaneous writing. Retained: writing to dictation, copying, reading aloud, and the comprehension of writing.

7. *Conduction or Association Aphasia.* Lost: all kinds of reading and of writing, with the exception of mechanical copying.

In these disturbances dependent upon aphasias, the writing and reading of individual letters is generally quite the same as the writing and reading of entire words. All aphasias cannot be tabulated under one of the above divisions, for reasons which will follow.

Besides disturbances in writing and reading dependent upon aphasias, there may also occur disturbances of written speech which are independent of the aphasias. This will be the case when a lesion occurs in the lower part of the diagram (Fig. 350)—*i. e.*, in the writing arc and in its connections with the central speech apparatus.

These disturbances, as contrasted with those depending upon aphasias, may be termed *isolated alexias* and *agraphias*. According to the diagram seven such disturbances are possible, for which a similar terminology may be employed as for the aphasias (Fig. 350).[3]

8. *Subcortical alexia* (injury between *u* and *a*). Reading of letters and words impossible; writing of letters and words, with the exception of copying, possible.

[1] The numbers correspond to those in Fig. 350 (see p. 891).
[2] In regard to the actual anatomic localization of the lesion, see p. 892, note.
[3] The numbers correspond to those in Fig. 350.

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9. *Subcortical agraphia* (injury between β and v). Reading of letters and words retained, but words can neither be written nor copied. (See below for the significance of this form.)

10. *Cortical alexia* (injury at a). Letters and words can neither be written nor read, and even copying is impossble.

11. *Cortical agraphia* (injury at β). Letters and words can be read but not written nor copied (as in 9). (See below for the significance of this form.)

12. *Conduction or association agraphia* (injury between a and β). Letters and words can be read but not written nor copied (again as in 9 and 11). (See below.)

13. *Transcortical alexia* (injury between a and a). Reading of letters and words impossible. Writing of letters and words, including copying, possible.

14. *Transcortical agraphia* (injury between b and a). Reading of letters and words possible. Writing of letters and words, except mechanical copying, impossible.

In the narrow sense of the word, lesions 9 and 11 do not actually come under the head of the agraphias, because the agraphias here depend only upon a paralysis of the arm.

Therefore only lesions 12 and 14 are to be considered as isolated, pure agraphias, and they can be distinguished one from the other by the retention of the power of copying in 14, and its loss in 12.

The three alexias, 8, 10, and 13, can be differentiated one from the other by the fact that in 13, as opposed to 8 and 10, copying is retained, and that in 8, as opposed to 10, spontaneous writing is retained.

Form 13, which we have called transcortical alexia, is the only well-known form of isolated alexia. It is also called word-blindness. The characteristic localization of the lesion in these cases is in the gyrus angularis (pli courbe). There is evidently an interruption in the association between the optical pictures of written characters stored in the occipital lobe and the sensory speech center in the temporal lobe. The term transcortical or, better, transcentral (in the sense indicated upon p. 891), is consequently justified.

The forms of isolated alexias and agraphias, considered here theoretically, have thus far rarely been observed as pure cases, but much more frequently there occur *mixed forms* arising from diffuse injuries to the cortical mechanism of speech writing or from the general injury of two or even more of the opposite convergent tracts in the neighborhood of the decussation angle. For this reason, and on account of incomplete interruptions of conduction, a local diagnosis of alexia and agraphia disturbances often becomes very difficult or even impossible.

It should also be noted that the significance of individual cases is often made very difficult in aphasias also by the occurrence of such mixed forms from lesions of the converging tracts, as well as by the occurrence of incomplete lesions of individual tracts and centers and of diffuse lesions. Hence, every case of aphasia cannot be tabulated under one or the other of the above types; at all events, not without much difficulty. There is still another reason why, practically, aphasias are often more difficult to explain than one would judge from our simple diagram. For in addition to the above-described type, where the disturbances depend upon injuries to tracts and destruction of centers, there is still another group of aphasias in which the difficulty is merely functional in nature and the tracts and centers in question are not actually destroyed or even injured. Such aphasias depend upon a very difficult or an imperfect innervation of the center and tracts upon the part of the patient. From the nature of the trouble, the clinical picture not only varies so conspicuously as to make a local diagnosis difficult, but also presents many other peculiarities. Under this head come the aphasias which are attributed to a disturbance of the memory, as the following case of Grashey will illustrate. This patient could give the name to an object held in front of him only so long as he was looking at it, and even then only while he was writing down its name and thus aiding his memory. These aphasias from disturbances of memory have been considered fundamentally different from the other aphasias, but the author cannot agree with this view. For what do we actually understand by a disturbance of memory? Apparently, on the one hand, only a difficulty in voluntarily recalling certain latent memory pictures—*i. e.*, a difficulty in associa-

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tions—and, on the other hand, an abnormally rapid loss of representations soon after their origin. Between these conditions and a complete destruction of the association fibers or of the representation centers there is only a difference in degree. Such destructions depend upon appreciable anatomic lesions, while disturbances in memory are to be attributed only to slight functional damage to the same centers and tracts.

By sticking to this definition of disturbances of memory, we can term these functional aphasias *memory aphasias* (in Grashey's sense), in contrast to aphasias from destructive lesions. But the former include not only Grashey's and similar cases, but, as will be easily understood, most of the transcortical and especially the complete transcortical aphasias. In a complete transcortical motor aphasia, which is the direct consequence of a gross anatomic lesion, all tracts which lead from *b* in Fig. 347 to the partial representations of the idea must be interrupted. But since these partial representations of the idea are spread out in the entire cortex, we must assume that the fibers *bc*, *bc''*, *bc'''*, etc., in reality extend in all directions from the motor speech center. A complete interruption of conduction of these collected tracts could then be imagined only if the center *b* were to a certain extent isolated, as by a ring-shaped lesion. However, in assuming a gross anatomic lesion, it is quite impossible that the center *b* could itself remain intact, so that symptoms of a cortical instead of a transcortical aphasia must result. It is, therefore, evident that complete transcortical motor aphasia and, for similar reasons, even a transcortical sensory aphasia cannot arise from direct gross anatomic destruction of all the transcortical tracts. A complete anatomic destruction of the latter is absolutely impossible without a lesion of the center *b* or of the centre *a*.[1] But from what has been said, it is easy to understand that these transcortical disturbances may result from those functional lesions defined above as disturbances of memory.

Such a functional disturbance may, of course, be diffused not only to the tracts of the speech mechanism, but also over the entire brain. In the former case merely a memory disturbance of speech will result; in the latter, a general mental weakness.

Most of the disturbances of speech called *indefinite* or *diffuse aphasias*, as contrasted with the definite, accompany functional lesions. They include aphasias which can be explained, not by a lesion at definite places in the scheme, but only by the assumption of a disturbance diffused over the territory of the speech apparatus. Such aphasias usually present an exceptionally variable character, and so cannot be attributed to the direct action of gross anatomic disturbances.

It should, however, be noted that in designating certain aphasic disturbances as *functional*, we do not mean that they cannot occur from the remote action of gross anatomic lesions. On the contrary, indefinite and transcortical aphasias, which, according to the above definition, must be considered as functional, generally exhibit lesions in the neighborhood of the speech centers. There is no actual destruction of brain substance in a gross mechanical sense, but the lesion indirectly causes a functional disturbance of the speech apparatus from *remote action*, or reaction at a distance. This remote action, playing so important a part in other parts of the cerebral pathology, is, as is well known, to be attributed partly to the results of circulatory disturbances, partly to inhibition.

Although most transcortical aphasias can thus be considered as functional, or, in the sense above, as of an amnesic nature (arising from weakness of memory or of association), such a grouping does not necessarily include those transcortical aphasias in which not all the transcortical tracts uniting the conceptual part of the brain with the speech center, but merely a certain number of transcortical tracts of separate function, are affected. To this class belong Freund's so-called optic aphasias, in which the patient cannot name objects after he has received their optical impression. Here we must assume an isolated lesion of the fibers running from the visual center to the motor speech centre. Such a lesion may

[1] As we shall see below, it must be acknowledged that transcortical aphasias may arise indirectly from gross anatomic lesions by so-called remote action.

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be anatomic as well as functional. Acoustic and tactile aphasias are analogously explained.

Finally, it may be mentioned that in aphasia the numbers are effected like other words only when they are spoken, not written. Numbers expressed in figures occupy a peculiar place in written speech, because no real word concept of them occurs either in writing or reading. The figure is a direct symbol of the notion of counting; to a certain extent, a hieroglyphic. Its optical representation probably may be considered as directly associated with the conception (Fig. 350). Hence, even when the word concept in aphasia is destroyed, the patient can write figures quite well and can understand written figures.

For the explanation of *anarthria* as a result of incomplete aphasia—*i. e.*, of incomplete lesions of the central speech apparatus—p. 888 *et seq.* may be consulted. In another type of incomplete aphasia the difficulty is limited to certain definite words. These are forms in which ordinarily no definite location in the speech mechanism is possible, and which usually come under the head of the functional or diffuse disturbances (p. 898).

(*c*) Other Speech Disturbances from Paralytic Phenomena.

Anarthria and aphasia are the best known and the best studied of the disturbances of speech resulting from paralytic phenomena. There are, however, a number of other speech disturbances to be regarded as paralytic phenomena which are not yet exactly explained and, in fact, cannot be definitely localized.

The disturbance of speech in *progressive paralysis* which is characterized by syllable-stumbling belongs to this class. From its peculiarities it belongs rather to the anarthrias than to the aphasias, and is ordinarily so classed; but it is questionable whether such a classification is correct. The well-known anatomic localization of progressive paralysis in the cerebral cortex makes it most probable that the speech disturbance is also cortical. Such a conception of its origin would suggest a closer relation to the aphasias, despite its external resemblance to the anarthrias. As a matter of fact, such a disturbance of speech may perfectly well be conceived to arise from minute lesions in the area of the speech center, by which speech co-ordination is destroyed, although a complete paralysis of the center does not result. Perhaps the disturbances of speech in intoxicated persons may be similarly explained. Without doubt the *hysteric speech disturbances*—hysteric aphonia (improperly called hysteric vocal-cord paralysis) and hysteric mutism—should be localized in the cortex, and, therefore, despite clinical differences, are closely related to aphasia. *Congenital dumbness* is nothing more than motor aphasia, and deaf-mutism is sensory aphasia plus motor aphasia plus deafness. The monotonous and imperfect speech which deaf-mutes can be taught may be considered as a form of speech arising, with sensory aphasia, by arduous and unusual paths, in which, instead of the entire word concept $a + b$, there is only available a motor word representation. The different disturbances of speech of those who are seriously ill (the vibrating, tremulous, slowed, and abnormally faint speech) have not as yet been definitely localized. They may be central as well as peripheral. The characteristic scanning speech of *multiple sclerosis* and the speech defect of *Friedreich's ataxia* do not yet possess a definite significance. The former, perhaps, may be regarded as a sort of spastic gait of the speech. As a result of the increased muscle and tendon reflexes, which may frequently be demonstrated in the muscles of speech in this disease (jaw-clonus, etc.), the speech movements are mechanically hindered by the occurrence of spasms of the muscles, which the patient instinctively tries to overcome by slowing his speech and accentuating its movements. The monotone and the lack of the ability to modulate the voice may also be explained by such spastic phenomena. Ordinarily the speech does not become tremulous, as do the other spastic movements, although a tremor may sometimes be observed as a result of quivering contractions of the vocal cords. This is probably due to the fact that the muscles of speech run in all the directions of space, and that tremors can be produced only

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by the alternate activity of antagonistic muscles, the lines of whose action are situated in the same plane.

3. AFFECTIONS OF SPEECH FROM IRRITATION PHENOMENA.

These irritation or spasmodic phenomena in the territory of the speech apparatus are considered as exceptional, and have been much less studied than the speech disturbances thus far described.

They include the commonest varieties of stuttering (labiochorea and guttural tetanic stuttering), which names characterize them sufficiently. They also include the affections of speech in chorea. It is not yet clear how these disturbances arise, nor is it known from what point the impulses spring, although they probably have a cortical localization. For, as the speech muscles are not utilized exclusively for speech and are not innervated exclusively by the speech center, it is easy to understand that choreic movements of the speech muscles might arise from other parts of the cortex and still affect the speech.

4. PLAN FOR TESTING THE FUNCTIONS OF SPEECH.

The following procedure for examining patients with speech disturbances has been devised from what has been said above:

1. Disturbances of pronunciation of letters and then of simpler and more complicated words. Anarthritic disturbances of bulbar paralysis; speech disturbances of progressive paralysis; of multiple sclerosis; stuttering, etc. For the differential diagnosis between pure anarthria and what is called on p. 889 "central anarthria," it must be noticed whether disturbances other than those of speech (swallowing, masticating) occur in the region of the speech muscles.

2. In the actual aphasic disturbances the following functions should always be examined

(*a*) TEST FOR WORDS.

1. Voluntary speech
2. Repetition.
3. Reading aloud.
4. Voluntary writing (figures to be tested especially).
5. Writing from dictation (figures to be tested especially)
6. Copying.
7. Speech comprehension.
8. Script comprehension (figures to be tested especially).
9. Counting syllables (see p. 893 *et seq.*).

One should note whether paraphasic disturbances—that is, confusion of words—occur in speaking or writing.

(*b*) TEST FOR LETTERS

1. Speaking the letters of the alphabet spontaneously with special attention to the quality of the pronunciation.
2. Repetition of letters.
3. Reading letters aloud.
4. Writing the alphabet spontaneously.
5. Writing letters from dictation.
6. Copying letters.
7. Recognition of spoken letters—*i. e.*, association of the letters pronounced with the pictures of letters (picking out the corresponding printed letters).
8. Recognition of written letters—*i. e.*, association of the letter pictures with the spoken letters (where the letters cannot be named, suggestive questions should be used in the test).

Since paralysis of the right arm is very commonly associated with aphasia, the patient's ability to write with the left hand should be tested (many aphasias produce mirror-writing). If the patient cannot write with the left hand, be

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should attempt to construct the words with letters such as are used in the ordinary game of anagrams.

If every case of aphasia is examined according to this plan, it is easy either to localize the aphasia at one or more definite points of the speech mechanism, or to determine that it is caused by an indefinite—*i. e.*, a functional or diffuse—injury of the speech mechanism.

VII. DISTURBANCES RELATED TO APHASIA: ASYMBOLIA; AMIMIA; APRAXIA; AMUSIA; PSYCHIC DEAFNESS; PSYCHIC BLINDNESS.

Asymbolia is a condition of the mind in which the ability to make one's self understood by signs and gestures (as well as by speech) is impaired or lost. A differentiation may be made between active or motor and passive or sensory asymbolia, dependent upon whether pantomimic speech or the ability to understand it is affected.

Amimia is a disturbance or loss of the power of imitation, whether it be simply the mimicry associated with speech or the mimicry expressing psychic processes.

Apraxia is the inability to employ objects in the proper way, the patient taking a spoon or fork in his hand, for example, and being unable to use it correctly.

The disturbances just described are also characterized by the fact that they are not associated with actual motor paralysis—*i. e.*, with motor disturbances of the affected muscles for other muscular functions. The intimate relation between the functions above mentioned and the function of speech sufficiently explains the fact that these disturbances are found almost exclusively with aphasias, and that their localization is practically the same as that of aphasia.

The loss of the ability to produce or comprehend music or musical sounds has been designated as **amusia**. From the definition, it will be seen that amusia may be either motor or sensory. Motor amusia is related to motor aphasia; it has a similar localization and is sometimes associated with it. Sensory amusia is related to sensory aphasia, with which it is not infrequently combined. Its presence is indicative of a lesion in the left temporal lobe. A special form of amusia, the loss of the ability to recognize the musical significance of notes or to sing or play from notes, has been designated as "note-blindness," and another form, in which the power to comprehend music has been abolished, has been rather inappropriately called "tone-deafness."

Psychic deafness is a condition in which not only words, but all sounds, though heard and perceived as such, awaken no intelligent conception. This disturbance evidently includes sensory aphasia, and the relationship of the two conditions is shown by the fact that sensory aphasia or word-deafness is sometimes combined with psychic deafness or passes into the latter condition.

Psychic blindness requires a more detailed description. By this term is meant that peculiar condition in which objects are seen but not recognized. In other words, in spite of maintained optic perception, the association of optic impressions is no longer possible. It holds the same relation to blindness as does psychic deafness to deafness. Psychic blindness can be produced only by a lesion in the transcortical tracts of the visual apparatus—*i. e.*, by a lesion in the association fibers of the visual center (see p. 891)—and is associated with lesions in the occipital lobes.

As the retinal impression of one eye, on account of the semidecussation of the optic nerve, affects both hemispheres, it is probable that the visual representations, upon whose associations with other representations depends the recognition of objects, are, unlike speech or word representation, localized in both hemispheres. This will explain the fact that psychic blindness is never observed in a unilateral lesion of the brain. If one hemisphere is completely preserved, visual perception and representation are not only pictured but also associated. The following figure explains diagrammatically the occurrence of psychic blindness from bilateral lesions of the occipital brain. It is supposed to represent a horizontal section

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of the brain: *a* the left, *b* the right retina; *c* the left, *d* the right visual center; *ad* and *bd* the fibers of the right, *ac* and *bc* those of the left optical tract; *ec* and *fd* represent diagrammatically examples of association fibers between the optic centers and other parts of the brain upon the same side. These are, of course, not single, but run in countless directions to all regions of the cortex not associated with vision. Now, according to the above, psychic blindness may arise from lesions of the bilateral association tracts (*ec* and *fd*)—for example, from lesions *g* and *h*; but it is highly improbable that in both posterior lobes only the association tracts should be affected in this way. As a matter of fact, heretofore it has been found that the fibers of the optic tract or even the visual center itself are injured coincidently with the association tracts upon the one side by a large lesion like *i*; whereas upon the other side a smaller lesion (*h*) may affect only the association tracts and not include the visual tracts or even the visual center.

A patient with such a bilateral lesion (*i* and *h*) presents clinically homonymous

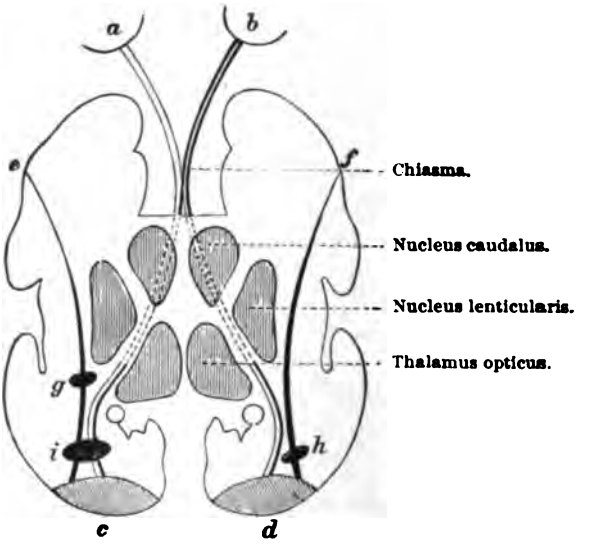

FIG. 351.—Diagram to explain the simultaneous occurrence of psychic blindness and hemiopia from a bilateral lesion in the occipital lobe. Horizontal cross-section of brain: *a*, Left retina; *b*, right retina; *c*, left sight center; *d*, right sight center (see Fig. 339); *ad* and *bd*, fibers of the right; *ac* and *bc*, fibers of the left optic tract; *g*, *h*, and *i*, lesions.

hemianopsia—*i. e.*, he is absolutely blind to objects whose pictures fall upon the left retina. He perceives objects with the right retinal half, but he cannot recognize them, because the association of the right visual center with the remainder of the brain is interrupted. It need scarcely be emphasized that all the associations of optical representations need not be destroyed in every case of psychic blindness, but that in many cases only certain of them fail, while others are retained. Thus, it is conceivable that a patient can associate the optical representation of a rose with the word representation of a rose, but not with the representation of the smell of the rose. Such examples of partial psychic blindness are undoubtedly not so very rare in the psychoses. But we ordinarily speak of psychic blindness in the narrow sense of the word, or of a total psychic blindness, only when so many associations of the optical representations are affected that the conception of the latter (see p. 890 *et seq.*) can no longer be elicited—*i. e.*, the object can no longer be recognized. An interference with the association of the optical representation with the speech mechanism, which is a kind of partial

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psychic blindness, has been studied as a partial transcortical aphasia upon p. 898, under the title of Optic Aphasia.

Complete psychic blindness frequently presents the clinical picture of mental confusion or insanity. Patients do not recognize objects at all, and name them in a most peculiar fashion. In certain characteristics they may behave like the blind, stumbling, for example, over objects which they may perhaps see but do not recognize. The condition is, therefore, often not very easy to diagnose, and in examining such a case the following plan may be adopted:

1. Examination of the perception in order to differentiate from a simple visual disturbance or from blindness. To be tested are: refraction, visual field (determination of hemiopia), acuteness of vision (this may be attempted with corrected refraction). Testing the visual acuteness in psychic blindness naturally presents very serious obstacles, as the ordinary test objects, such as letters, etc., cannot be recognized. We are therefore usually obliged to make use of some artificial device, such as requiring the patient to name the number of black points upon a sheet of paper.

2. Examinations to determine whether visual representations exist. In this connection only the patients' statements about optical memory, etc., can furnish any information.

3. Examination of association of visual impressions. Recognition of objects by statement of the name or by demonstration of their use, reading (aloud and with comprehension), copying, drawing, voluntary reactions to optical irritation, power of orientation.

4. Examination of the association of visual representations. Drawing from memory, writing spontaneously, writing from dictation.

5. In differentiating psychic blindness from general confusion or insanity, it is necessary to determine whether the other senses—viz., those of hearing, smell, taste, as well as the sensory skin sensation—are properly associated, and to observe whether the patients deport themselves sensibly so far as their behavior toward visual sensations is concerned.

The diagnosis of actual psychic blindness is frequently very difficult, and is often impossible when complicated with marked diminution in vision, because conditions occur in all forms of diminished visual acuteness which have this in common with real psychic blindness, that the significance of what is seen is disturbed merely because the visual impressions are not sharp enough.

Psychic blindness is not to be confused with cortical blindness. The latter affection is an actual blindness, a loss of the visual power, produced by lesions of the cortex, and, owing to the hemiopic distribution of the visual function in both hemispheres, must be bilateral.

VIII. SPINAL HEMIPLEGIA.

Unilateral lesions of the spinal cord produce a symptom-complex which is known as "spinal hemiplegia," in the broadest sense of the word. This hemiplegia varies in character according to the location and extent of the lesion.

The motor disturbances appear in the extremity upon the same side as the lesion and in only the lower, or both the lower and the upper, extremities, according to the level of the lesion. The motor paralysis dependent upon an interruption of the long tracts, particulary of the pyramidal tract, is spastic in character, as in a complete transverse lesion of the cord, is associated with increased tendon reflexes, and is not accompanied by degenerative atrophy of the paralyzed muscles. If the lesion have a considerable vertical dimension, this spastic paralysis may be associated with a degenerative atrophic relaxed paralysis due to involvement of the nuclei in the anterior cornua. With an extensive vertical lesion in the cervical enlargement, for example, the spastic paralysis will be bounded above by a degenerative relaxed paralysis of the upper extremity; if the extensive vertical lesion be situated in the lumbar cord, the paralysis of the muscles supplied by the injured segments will be atrophic and relaxed. Should the lesion involve the entire length of the lumbar swelling, the spastic paralysis of the leg will be

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entirely replaced by a relaxed degenerative paralysis. In reference to the spastic components of the paralysis dependent upon the involvement of the long tracts, it may be said that the laws given for cerebral hemiplegia on p. 876 *et seq.* in reference to the more or less marked involvement of the individual muscle groups also obtain in spinal hemiplegia.

The vasomotor tracts are involved upon the side of the lesion, so that the extremity at first seems warmer than its fellow of the opposite side. This difference subsequently becomes equalized by the decreased heat production in the paralyzed muscles, and also by the disappearance of the vasomotor palsy as the result of the vicarious action of deeper centers. The paralyzed side may then even be colder to the touch than its opposite fellow.

The sensory disturbances in unilateral spinal lesions are of special interest and somewhat more complicated. We have just seen that the motor symptoms, corresponding to the uncrossed exit of the motor tracts from the spinal cord, occur only upon the same side as the lesion; but the sensory disturbances are partly bilateral, although most of them are present only upon the side opposite to the lesion. The observation of the sensory phenomena in such cases has resulted in the establishment of the three following types:[1]

Type 1 (Fig. 352). Upon the same side as the lesion: Cutaneous hyperesthesia of the skin to touch, bounded above by a zone of cutaneous anesthesia for all sensory qualities; above the latter area there is sometimes still another narrow zone of hyperalgesia to touch. Disturbances of sensation of the deep viscera (disturbance of the perception of passive changes of the position of the extremities and of bone sensation).

Upon the opposite side: Cutaneous analgesia and thermanesthesia, sometimes (though more rarely than upon the same side as the lesion) bounded above by a zone of cutaneous hyperalgesia to touch. Intact sensation to touch.

Type 2 (Fig. 353). As in Type 1, except that the analgesic and thermanesthetic regions upon the side opposite to the lesion also exhibit hyperesthesia or anesthesia to touch.

Type 3 (Fig. 354). As in Type 1, but, with the exception of the zones at the level of the lesion, there is no hyperalgesia to touch upon the side of the lesion; on both sides below the lesion or below the small zones of hyperalgesia there is hyperesthesia to touch (Gowers).

These different symptoms may be explained by the following assumptions: The majority of the fibers conducting the sensations of pain and temperature decussate immediately upon their entrance into the spinal cord and pass upward to the brain upon the other side of the median line. About half of the fibers conducting the sensation of touch decussate immediately and half remain upon the same side. The fibers for the perception of passive movements of the extremities and for bone sensation run upward in the spinal cord without decussation. The uncrossed fibers probably pass upward in the posterior columns, while the crossed fibers, after their decussation, run upward in the anterolateral columns. The decussation of the fibers in the latter group, if it occur at all (see above), takes place in the gray matter, since an impulse from a collateral in the posterior horn of one side is taken up by an offshoot of the anterolateral column of the other side (see Fig. 362). It should be noted that, as the decussation never affects the entire fibers of the posterior roots but always only their collaterals,[2] we might indicate the relation between the crossed and uncrossed sensory fibers diagrammatically by representing the posterior root as consisting of a crossed and an uncrossed portion. This conception is of importance for the explanation of the hyperalgesia upon the side of the lesion.

[1] See Mann, *Zeits. f. Nervenkrankh.*, 1896, vol. x., and *Gowers's Handbook of Nervous Diseases.*

[2] As is well known, every fiber of the posterior root immediately upon entering the spinal cord divides into an ascending and a descending branch, both of which give off transverse ramifications known as collaterals. These collaterals run approximately transversely in the cord, and send on the sensory impulses through their terminal arborizations.

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The most important of these assumptions are diagrammatically indicated in the accompanying illustration (Fig. 355). In order to render it more compre-

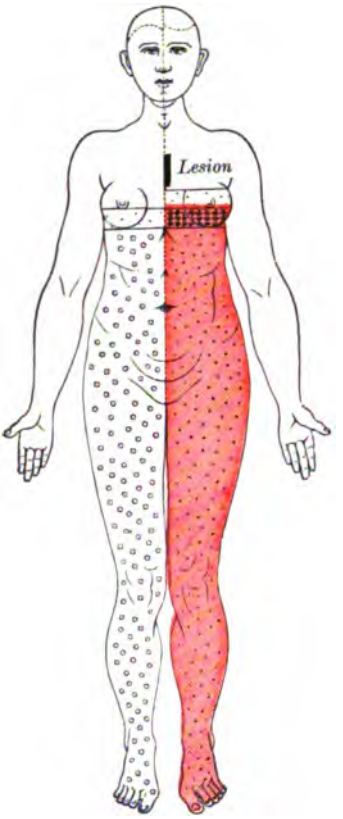

FIG. 352.

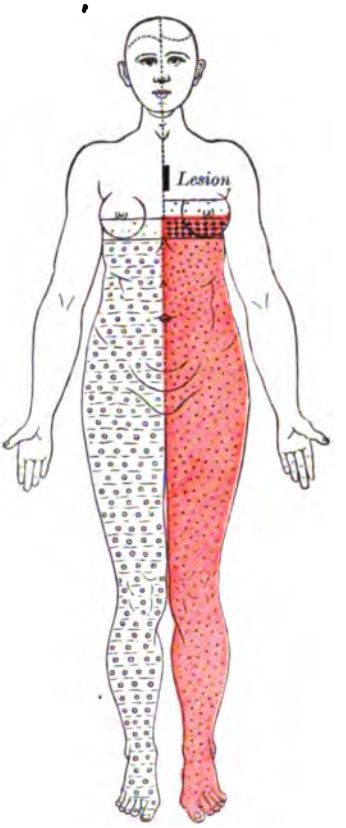

FIG. 353.

FIG. 352.—Spinal hemiplegia. Lesion upon the right side of the patient. Type 1:

Motor and vasomotor paralysis. Varying degree of disturbance of sensation of the deep viscera (perception of position), including the bones.

Analgesia and thermanesthesia of the skin. Intact sensation to touch.

Cutaneous hyperalgesia to touch (also in the red shaded area).

Complete cutaneous anesthesia.

Upon the white side there is neither motor nor vasomotor paralysis.

FIG. 353.—Spinal hemiplegia. Lesion upon the right side of the patient. Type 2:

Motor and vasomotor paralysis. Varying degree of disturbance of sensation of the deep viscera (perception of position), including the bones.

Cutaneous hyperalgesia to touch (also in the red shaded area).

Complete anesthesia.

Diminished sensation to touch and abolished sensations of pain and temperature.

Upon the white side there is neither motor nor vasomotor paralysis.

hensible, the sensory fibers for the perception of passive movements of the extremities and for bone sensation, as well as the motor and vasomotor fibers,

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have been omitted. By the use of this diagram the differences between the sensory symptom-complexes of unilateral lesions may be readily understood.

The red lines represent the fibers for the conduction of the sensations of pain and temperature; although these sensations are not transmitted by the same fibers, they both pursue a similar course. The black lines represent the fibers for the conduction of the sensation of touch. It will be observed that each of these

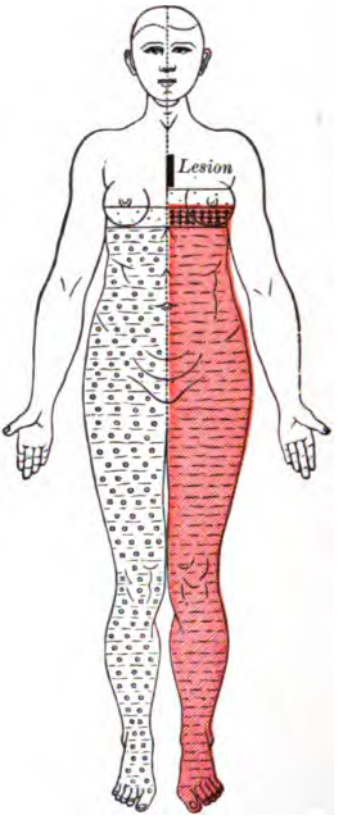

FIG. 354.—Spinal hemiplegia. Lesion upon the right side of the patient. Type 3: Motor and vasomotor paralysis. Varying degree of disturbance of sensation of the deep viscera (perception of position), including the bones.

Hyperalgesia to touch.

Complete anesthesia.

Diminished sensation to touch and abolished sensations of pain and temperature.

Diminished sensation to touch (also in the red shaded area).
Upon the white side there is neither motor nor vasomotor paralysis.

fibers, after its entrance into the spinal cord, divides into a crossed and a direct portion. The direct portions of the (red) fibers for the conduction of the impulses of pain and temperature are indicated by light lines, in order to give the impression that they are few in number and that they play an unimportant rôle in comparison with the much greater number of crossed fibers. The direct and the crossed portions of the (black) fibers for the sensation of touch are, on the contrary, indicated by exactly similar lines. For the sake of clearness, no attention

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has been paid to the topographic arrangement of the fibers in the cross-section of the spinal cord, nor to the composition of the crossed tracts from two neurons. The shaded area represents the substance of the spinal cord which is involved by a unilateral lesion.

With the aid of this figure the sensory phenomena of Type 1 (Fig. 352) may now be explained. The fact that the perceptions of passive motion of the extremities and of bone sensation will be disturbed upon the side of the lesion clearly follows from the fact that these fibers (not indicated in Fig. 355) pass upward uncrossed in the spinal cord. We can also understand that a complete transverse section of one-half of the cord will be followed by an almost complete absence of the sensations of pain and temperature upon the opposite side below the lesion (due to involvement of the heavy red lines in Fig. 355). The direct fibers for these sensations (indicated by the light red lines) are so few in number that they are unable to produce symptoms upon the same side as the lesion, and are equally powerless to prevent a marked disturbance of the sensations for pain and temperature upon the opposite side. The figure also explains

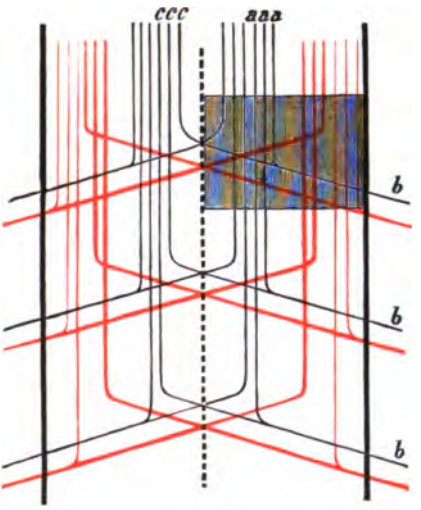

FIG. 355.—Diagram of the sensory fibers for the explanation of the phenomena of spinal hemiplegia.

why in many cases there is no diminution of the sensation to touch below the lesion, with the exception of the zone corresponding to the involved segment, since the equal division of these fibers into crossed and uncrossed tracts is evidently sufficient to maintain the normal sensory conditions. Many theories have been advanced to explain the hyperalgesia to touch which is usually observed upon the same side and below the lesion. The author believes that this symptom can be rendered intelligible by assuming that the crossed and direct tracts for tactile sensation are mutually complementary ramifications of the sensory roots. When the direct tracts *bbb aaa* are interrupted, the crossed tracts *bbb ccc* receive a nervous impulse of double intensity, and this intensity is sufficient to cause the impulse to radiate into the dendritic network of the gray matter and excite the tract for the sensation of pain. Upon the opposite side the crossed fibers for tactile sensation are involved, and this would be followed by hyperalgesia from analogous reasons were it not that the increased intensity of tactile sensation cannot be converted into pain on account of the interruption of the tract for its transmission. The anesthetic zone observed upon the side of the lesion is explained simply by the involvement of the spinal segment by the

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lesion, since all of the sensory root fibers are interrupted. The hyperalgesic zone, sometimes observed above this anesthetic zone upon the same side as the lesion, and occasionally upon the opposite side, can hardly be explained otherwise than by the supposition that the roots in the vicinity of the lesion may easily be rendered hyperirritable by inflammatory hyperemia, possibly from the meninges.

The involvement of tactile sensation in the hemianesthesia opposite to the lesion in Type 2 (Fig. 353), is explained by presuming that in these cases the crossed fibers for tactile sensation preponderate greatly over the direct. Such individual peculiarities are frequently encountered in neuropathology.

The cases of Type 3 (Fig. 354), in which, instead of hyperalgesia to touch on the same side below the lesion, we have hyperesthesia on both sides below the lesion, are explained by the supposition that the excitability of the tract for tactile sensation is so diminished that the loss of one-half of its innervation results in a decrease of sensibility, and, since the fibers run to both sides, this symptom is bilateral. Hyperalgesia is not produced below and upon the same side as the lesion on account of the diminution of the sensory impulses.

It should also be noted that, according to the condition of irritability of the sensory tracts and according to the remote effects of the unilateral lesion upon the opposite side, the symptom complex of spinal hemiplegia is subject to still further variation.

IX. PATHOLOGIC GAITS AND POSTURES.

In many diseases, and especially in nervous diseases, the way patients stand and walk shows something very characteristic, permitting a conclusion not only in regard to the type of the functional disturbance, but frequently even in regard to the anatomic nature of the disease. The following are the best-known pathologic gaits:

(*a*) **The Paraparetic or Paraplegic Gait.**—In paresis of both lower extremities. Both legs are brought forward slowly, dragging and trailing upon the floor.

(*b*) **The Hemiparetic or Hemiplegic Gait.**—In unilateral paralysis of the legs or hemiplegia. The affected leg drags after the other, or is first circumducted by a twisting movement of the pelvis and so brought forward. An explanation of this gait is to be found upon p. 877 *et seq.* The predominance of the paralysis of the flexors is considered responsible for the hemiplegic disturbances of motility.

(*c*) **The Atactic Gait.**—Characterized by the incoördinate atactic nature of the movements. Sometimes the foot feels uncertainly for the ground, sometimes it is thrown outward at random or stamps, and again is lifted high from the ground, making the gait resemble that of a fowl. (See Ataxia, p.752 *et seq.*)

(*d*) **The Spastic Gait.**—In spastic paresis of the lower extremities (spastic spinal paralysis, multiple sclerosis, etc.), the legs, being very slightly flexible, are put forward very stiffly. In putting the foot down the tendon reflexes (especially the Achilles-tendon reflex) are sometimes excited and the gait becomes characteristically jumping. The difficulty in the spastic gait depends at one time more upon the stiffness of the knee-joints, at another more upon the knees being pressed together by the forcible action of the adductors.

(*e*) **The Spastic Paretic Gait.**—Mixture of *a* and *c*.

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(*f*) **The Gait of Hip-joint Disease.**—This is characterized by a rigidity of the hip joint, so that the pelvis is largely responsible for any forward motion of the limb. Under some conditions the gait in hysteric coxalgia may be identical.

(*g*) The **gait in sciatica** may resemble that in hip-joint disease. The patient favors the affected leg by fixing it to the pelvis, almost as regularly as in hip-joint disease. In so-called sciatic scoliosis the vertebral column is curved while the patient is walking or standing. There are usually two characteristic curves; the lower curve is convex, the upper concave toward the affected side. The trunk is, therefore, as a rule, bent toward the healthy side (heterologous sciatic scoliosis), although the curves may be reversed (homologous sciatic scoliosis).

Attempts have been made to explain these differences in sciatic scoliosis, but it seems quite plain that no explanation is possible for them all. Albert described heterologous sciatic scoliosis, but did not venture an explanation. Lorenz assumes that this form of scoliosis is brought about simply by shifting the point of gravity to the healthy leg. Kocher explains it by an associated neuralgic affection of the sensory nerves supplying the territory of those muscles which hold the trunk straight. When the contraction of these muscles results in pain it is no longer attempted. The spinal column bends, therefore, its direction depending upon what nerves are thus affected.

(*h*) **The Choreic Gait.**—In chorea. (See Chronic Movements, p. 750.)

(*i*) **The Staggering Gait.**—In affections which are associated with vertigo and disturbances of equilibrium (drunkenness, cerebellar tumor, paralyses of the eye muscles, diseases of the internal or middle ear, lead encephalopathy).

(*k*) **The Gait of Propulsion and Retropulsion.**—In affections with stiffness and weakness of the muscles, especially in *paralysis agitans*. This gait is peculiar in that patients once started forward or backward are not able to stop quickly, but must go on a little farther in the direction in which they are headed, because they cannot correct quickly the point of gravity which impels them in that direction. It is, however, not really specific for paralysis agitans, as is sometimes thought, for one often notices the same peculiarity in pedestrians who have been tired by a long tramp.

The attitude or mode of standing is very characteristic in many of the affections cited above. In *hip-joint disease* and in *unilateral leg paralysis* the patient supports himself entirely upon the healthy leg.

In *sciatica* the scoliotic appearances described above are very prominent. In *paralysis agitans* the position of the trunk, bent over forward, with slightly flexed knees and elbow joints, is highly characteristic. (See the picture in *Strümpell's Lehrbuch der Pathologie.*) Romberg's symptom consists of more or less noticeable swaying in patients who stand with closed eyes (in severe cases even with open eyes). It occurs in anesthesia of the lower extremities, also in ataxia with or without anesthesia (especially in tabes), in affections of the cerebellum, and in

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some other affections which lead to a staggering gait. The sign depends upon a disturbance of the equilibrium.

X. SPECIAL POINTS IN REFERENCE TO THE EXAMINATION OF THE SPINAL NERVOUS SYSTEM.

1. PLANS FOR THE EXAMINATION OF MUSCLE ATROPHIES AND PERIPHERAL MOTOR PARALYSIS.[1]

Upper Extremity.

(For motor points compare p. 802 *et seq.*)

SHOULDER-BLADE MOVEMENTS.

1. *Elevation of the Shoulder Blade.*
   - Middle portion of the trapezius (spinal accessory).
   - Rhomboids (branch from V. cervical).
   - Levator anguli scapulæ (II. to III. cervical and branch from V. cervical).
   - Superior portion of the pectoralis major (thoracic anterior from V. and VI. cervical).
2. *Depression of the Shoulder Blade.*
   - Pectoralis minor (thoracic anterior).
   - Inferior portion of the latissimus dorsi (subscapular).
   - Inferior portion of the pectoralis major (thoracic anterior).
3. *Adduction of the Shoulder Blade.*
   - Inferior portion of the trapezius (spinal accessory).
   - Superior portion of the latissimus dorsi (subscapular).
   - Rhomboids. (Branch from V. cervical).
4. *Abduction of the Shoulder Blade.*
   - Superior third of the pectoralis major (thoracic anterior).
   - Serratus anticus major (thoracic longus from sixth, seventh, and eighth cervical nerve).

SHOULDER-JOINT MOVEMENTS.

1. *Elevation of the Upper Arm.*
   - (*a*) To the side :
     - Up to the horizontal : deltoid (circumflex).
     - Up to the vertical : deltoid and serratus anticus major (thoracic longus).
       - With straining, the upper part of the trapezius in addition (spinal accessory).
   - (*b*) Forward :
     - Anterior portion of the deltoid (circumflex).
     - Coracobrachialis (musculocutaneous).
     - Biceps (musculocutaneous).
       - In elevation to the vertical, the serratus anticus major also aids.
   - (*c*) Backward :
     - Posterior portion of the deltoid (circumflex).

[1] The anatomic statements in these schemes have been collected from Scheube and Duchenne, and then compared with Gegenbauer's Anatomy (4th edition, 1890). For the sake of simplicity, the origin of a nerve from its motor root is stated in the plan only where the nerve is mentioned for the first time. In this way it is easy enough to find the root origin of a certain nerve for a certain muscle by searching for the name of this nerve in the first place it is mentioned in the scheme. For those nerves whose origin is not stated in the scheme, the reader should consult the diagrams of the extremity plexuses, pp. 935, 936, and Kocher's plates of the spinal motor segmental innervation. Further clinical experience is necessary to clear up individual points still under contention.

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2. *Depression of the Upper Arm.*
   The united adductors of the upper arm.
3. *Adduction of the Upper Arm.*
   Pectoralis major (thoracic anterior from V. and VI. cervical).
   Latissimus dorsi and teres major (subscapular).
   Infraspinatus (suprascapular from V. and VI. cervical).
   Teres minor (circumflex).
4. *Inward Rotation of the Upper Arm.*
   Subscapularis (subscapular).
   Teres major (subscapular).
5. *Outward Rotation of the Upper Arm.*
   Infraspinatus (suprascapular).
   Teres minor (circumflex).

Elbow-joint Movements.

1. *Flexion of the Forearm.*
   Biceps [flexor and supinator] (musculocutaneous).
   Brachialis anticus (musculocutaneous).
   Supinator longus [supinates or pronates according to the position; it is, however, chiefly a flexor in the median position] (musculospiral).
2. *Extension of the Forearm.*
   Triceps (musculospiral).
3. *Supination of the Forearm.*
   Supinator brevis (musculospiral).
   Supinator longus (see flexion).
4. *Pronation of the Forearm.*
   Pronator quadratus (median).
   Pronator teres [pronation and flexion] (median).
   Supinator longus [in an extreme position of supination] (musculospiral).

Wrist Movements.

1. *Flexion of the Hand.*
   Flexor carpi radialis [flexion to the radial side] (median).
   Flexor carpi ulnaris [flexion to the ulnar side] (ulnar).
   Palmaris longus (ulnar).
2. *Extension of the Hand.*
   Extensor carpi radialis longior and brevior [extension to the radial side] (musculospiral).
   Extensor carpi ulnaris [extension to the ulnar side] (musculospiral).
3. *Abduction (Radial Flexion) of the Hand.*
   Flexor carpi radialis and extensor carpi radialis longior and brevior (median and musculospiral).
4. *Adduction (Ulnar Flexion) of the Hand.*
   Extensor carpi ulnaris and flexor carpi ulnaris (musculospiral and ulnar).

Finger Movements.

1. *Flexion of the Fingers.*
   Flexor digitorum sublimis [flexing the second phalanx] (median).
   Flexor digitorum profundus [flexing the finger from the distal phalanx] (median and ulnar. The former supplies the radial sides; the latter, the ulnar sides of the several fingers).
   The interossei and lumbricales (flexing the proximal phalanx and extending the two distal phalanges). Nerve supply principally the ulnar; in the innervation of the lumbricales, the ulnar nerve is aided by the median nerve to the extent that the latter is distributed to the two radial and a portion of the next lumbrical while the ulnar supplies the rest.

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2. *Extension of the Fingers.*
   Extensor digitorum communis, extensor indicis, extensor minimi digiti [extending the proximal phalanx] (musculospiral).
   Interossei and lumbricales [extending the two distal phalanges] (ulnar and median; see above).
3. *Adduction of the Fingers.*
   Palmar interossei [flexing the proximal phalanx simultaneously] (ulnar).
4. *Abduction of the Fingers.*
   Dorsal interossei [flexing the proximal phalanx simultaneously] (ulnar).

Thumb Movements.

1. *Flexion of the Thumb.*
   Flexor longus pollicis [flexing the distal phalanx] (median).
   Flexor brevis pollicis [flexing the proximal phalanx] (median).
2. *Extension of the Thumb.*
   Extensor brevis pollicis (musculospiral).
   Extensor longus pollicis (musculospiral).
3. *Abduction of the Thumb.*
   Abductor longus pollicis (musculospiral).
   Abductor brevis pollicis [more opponens than abductor] (median).
4. *Adduction of the Thumb.*
   Adductor pollicis (ulnar).
5. *Opposition of the Thumb.*
   Opponens pollicis (median).
   Abductor brevis pollicis [more opponens than abductor] (median).

Little-finger Movements.

1. *Flexion of the Little Finger.*
   Flexor digitorum communis profundus and sublimis (median and ulnar).
   Flexor brevis minimi digiti (ulnar).
2. *Extension of the Little Finger.*
   Extensor minimi digiti proprius (musculospiral).
3. *Abduction of the Little Finger.*
   Abductor minimi digiti (ulnar).
4. *Opposition of the Little Finger.*
   Opponens minimi digiti (ulnar).

Lower Extremities.

(For motor points, see p. 804 *et seq.*)

Hip-joint Movements.

1. *Elevation of the Thigh.*
   Ileopsoas [outward rotation simultaneously] (lumbar plexus).
   Rectus femoris, Sartorius } (crural from the I. to IV. lumbar).
2. *Depression of the Thigh.*
   Gluteus maximus [outward rotation simultaneously] (gluteal inferior from ischiadic plexus).
   Biceps, Semitendinosus, Semimembranosus } (ischiadic, IV. lumbar to III. sacral) [at the same time flexing the leg, but only with extension of the thigh, not in walking—Wernicke-Mann (see p. 877)].
3. *Inward Rotation of the Thigh.*
   Gluteus medius et minimus (gluteal superior from ischiadic plexus).

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4. *Outward Rotation of the Thigh.*
   - Quadratus femoris } (sciatic).
   - Obturator internus and Gemelli }
   - Obturator externus (obturator from II. to IV. lumbar).
   - Pyriformis (ischiadic plexus).
   - Ileopsoas (lumbar plexus).
   - Gluteus maximus (gluteal inferior from ischiadic plexus).
5. *Adduction of the Thigh.*
   - Adductors [simultaneous outward rotation] (obturator).
   - Pectineus [simultaneous flexion] (crural and obturator).
   - Gracilis (obturator).
6. *Abduction of the Thigh.*
   - Gluteus medius et minimus (gluteal superior).

Knee-joint Movements.

1. *Flexion of the Leg.*
   - Sartorius [simultaneous inward rotation of the flexed leg] (crural). } Principal flexor (shortener of the leg) in walking—Wernicke-Mann (see p. 877).
   - Gracilis [simultaneous inward rotation] (obturator). }
   - Semitendinosus } [simultaneous inward rotation] (sciatic). } Not as flexion of the leg, but as extensor of the thigh in walking (elongator of the leg)—Wernicke-Mann (see p. 877).
   - Semimembranosus }
   - Biceps [simultaneous outward rotation] (sciatic). }
   - Popliteus [simultaneous inward rotation] (internal popliteal from sciatic).
2. *Extension of the Leg.*
   - Quadriceps (anterior crural).
3. *Inward Rotation of the Leg.*
   - Popliteus (internal popliteal).
   - Sartorius (anterior crural).
   - Gracilis (obturator).
   - Semitendinosus } (sciatic).
   - Semimembranosus }
4. *Outward Rotation of the Leg.*
   - Biceps (sciatic).

Foot-joint Movements (Sciatic).

1. *Dorsal Flexion of the Foot.*
   - Tibialis anticus [innervating at the same time the inner foot surface] (anterior tibial nerve from sciatic).
   - Extensor digitorum communis longus (simultaneous abduction).
2. *Extension (plantar flexion) of the Foot.*
   - Gastrocnemii } (internal popliteal from sciatic).
   - Soleus }
   - Peroneus longus [simultaneous abductor and elevator of the outer foot surface] (musculocutaneous nerve from sciatic).
3. *Adduction of the Foot.*
   - Tibialis posticus [simultaneous raising of the inner foot surfaces and plantar flexion of the foot] (posterior tibial nerve).
   - Tibialis anticus [simultaneous dorsal flexion of the foot and raising of the inner foot surfaces] (anterior tibial nerve).

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4. *Abduction of the Foot.*
   Peroneus longus [simultaneous plantar flexion with elevation of the outer foot surfaces] (musculocutaneous).
   Peroneus brevis [pure (?) abductor with elevation of the outer foot surfaces] (musculocutaneous).
   Extensor digitorum communis longus (anterior tibial).
5. *Elevation of the Inner Foot Surfaces.*
   Tibialis anticus [simultaneous dorsal flexion and adduction] (anterior tibial).
   Tibialis posticus [simultaneous adduction and plantar flexion] (internal popliteal).
6. *Elevation of the Outer Foot Surfaces.*
   Peroneus longus and peroneus brevis (musculocutaneous).
   Peroneus tertius (anterior tibial).

Toe Movements (Sciatic).

1. *Flexion of the Toes.*
   Flexor digitorum communis longus and brevis [second and third phalanges] (tibial).
   Interossei and lumbricales [first phalanx] (tibial).
2. *Extension of the Toes.*
   Extensor digitorum communis longus and brevis (anterior tibial).
3. *Adduction of the Toes.*
   Interossei plantares (tibial).
4. *Abduction of the Toes.*
   Interossei dorsales (tibial).

Great-toe Movements (Sciatic).

1. *Flexion of the Great Toe.*
   Flexor hallucis longus [second phalanx] (tibial).
   Flexor hallucis brevis [first phalanx] (tibial).
2. *Extension of the Great Toe.*
   Extensor hallucis longus [second phalanx] (anterior tibial).
   Extensor hallucis brevis [first phalanx] (anterior tibial).
3. *Adduction of the Great Toe.*
   Adductor hallucis.
   Inner belly of the flexor hallucis brevis } (tibial).
4. *Abduction of the Great Toe.*
   Abductor hallucis
   Outer belly of the flexus hallucis brevis } (tibial).

Little-toe Movements (Tibial).

1. *Flexion of the Little Toe.*
   Flexor digiti quinti (tibial).
2. *Abduction of the Little Toe.*
   Abductor digiti quinti (tibial).
3. *Opposition of the Little Toe.*
   Opponens digiti quinti (tibial).

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2. THE PERIPHERAL DISTRIBUTION OF THE SENSORY CUTANEOUS NERVES.

For the localization of peripheral sensory disturbances consult Figs. 356–361.

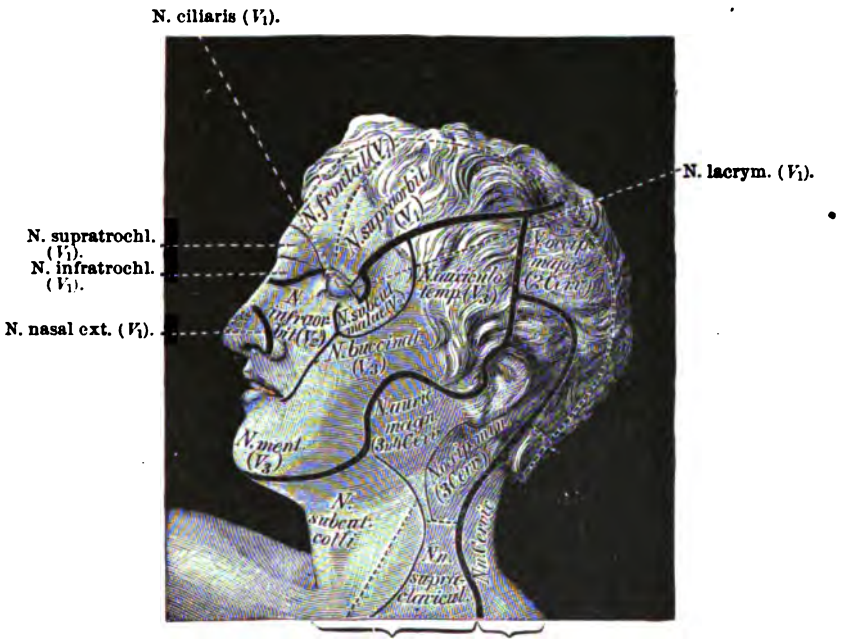

FIG. 356.—Cutaneous nerves of the head. The back of the ear, and the skin of the back wall of the external auditory meatus are supplied by the auricularis vagi.

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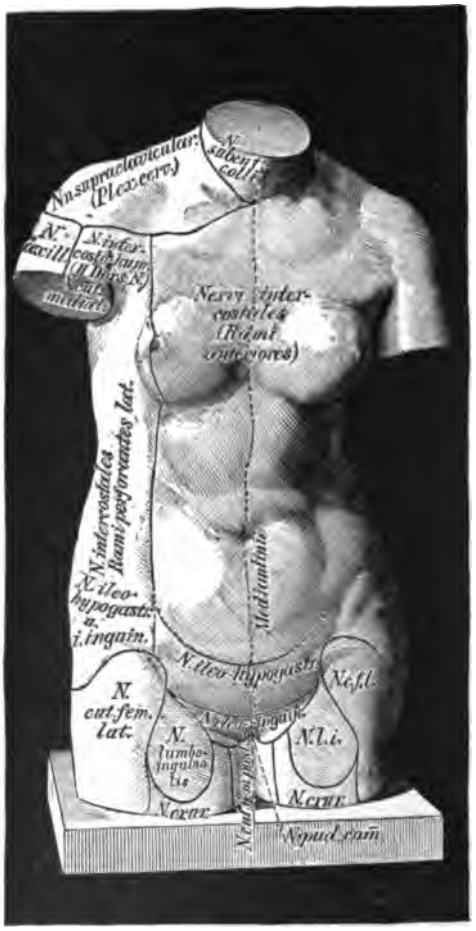

FIG. 357.—Cutaneous nerves of the anterior surface of the trunk (see also Fig. 364.).

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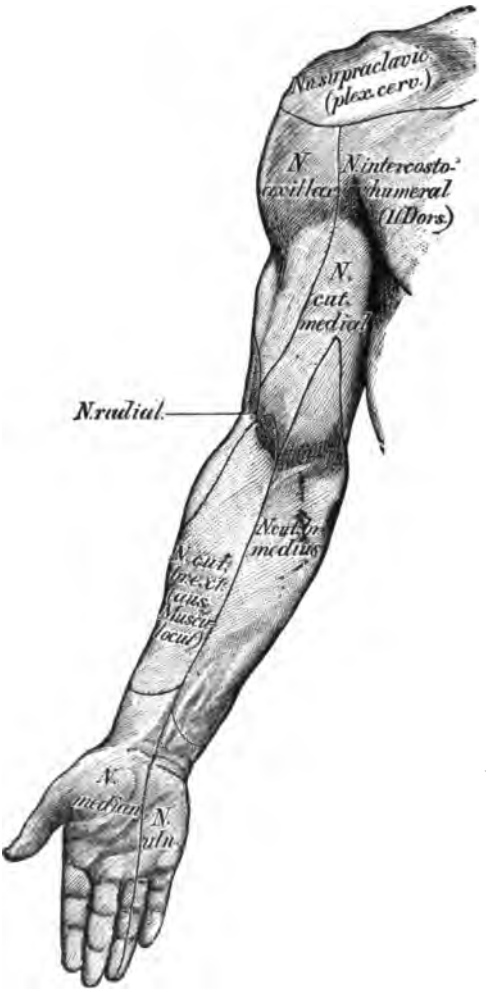

Fig. 358.—Cutaneous nerves of the flexor surface of the upper extremity (see also Fig. 364).

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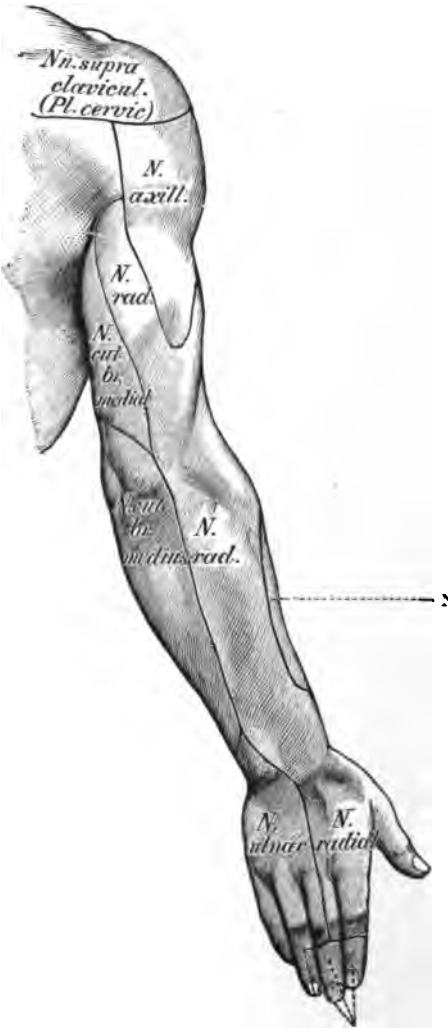

FIG. 359.—Cutaneous nerves of the extensor surface of the upper extremity (see also Fig. 364).

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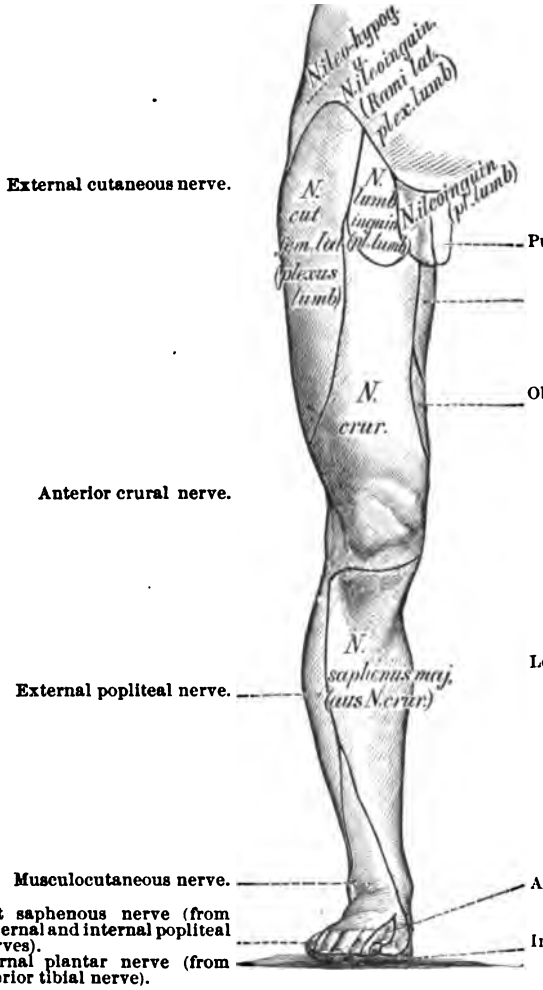

FIG. 360.—Cutaneous nerves of the anterior surface of the lower extremity (see also Fig. 364).

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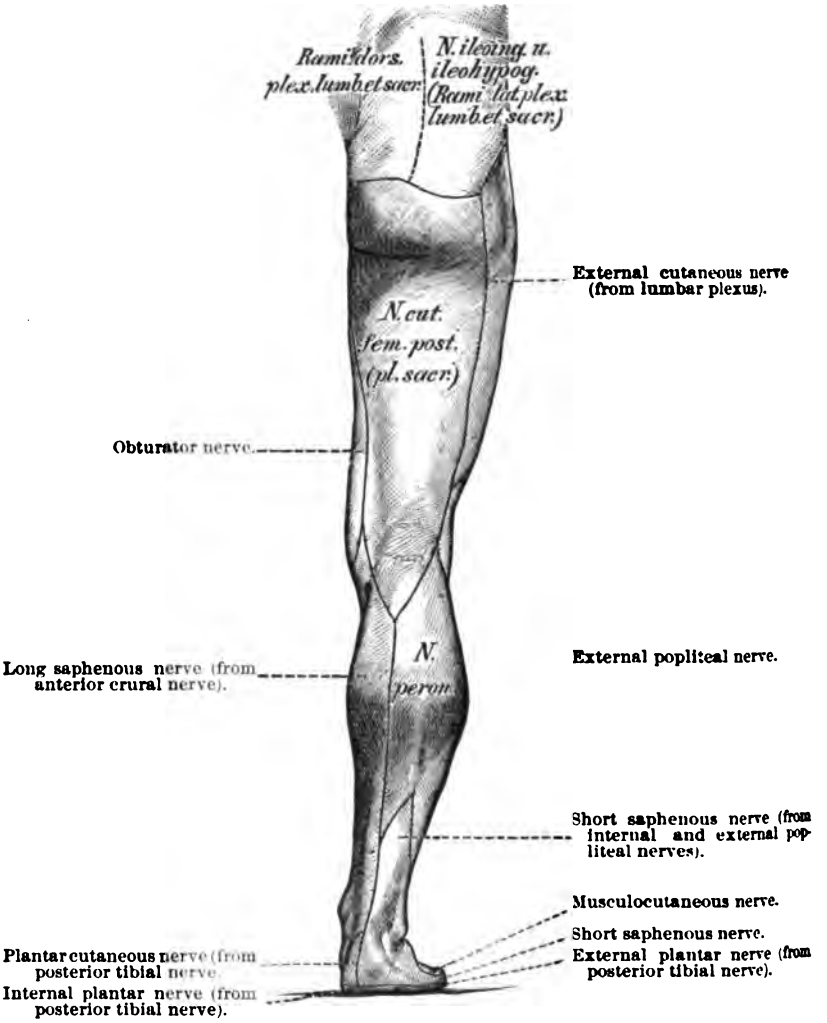

FIG. 361.—Cutaneous nerves of the posterior surface of the lower extremity (see also Fig. 364).

3. SPINAL LOCALIZATIONS.

(*a*) Cross-section Localization of the Spinal Cord.

In regard to this point the reader is referred to the text-books upon Spinal Anatomy, which discuss the anatomic significance and the physiologic functions of the individual areas of the cross-section. We must be content here with inserting the two plates taken from Edinger and Obersteiner for orientation of the sensory tracts.

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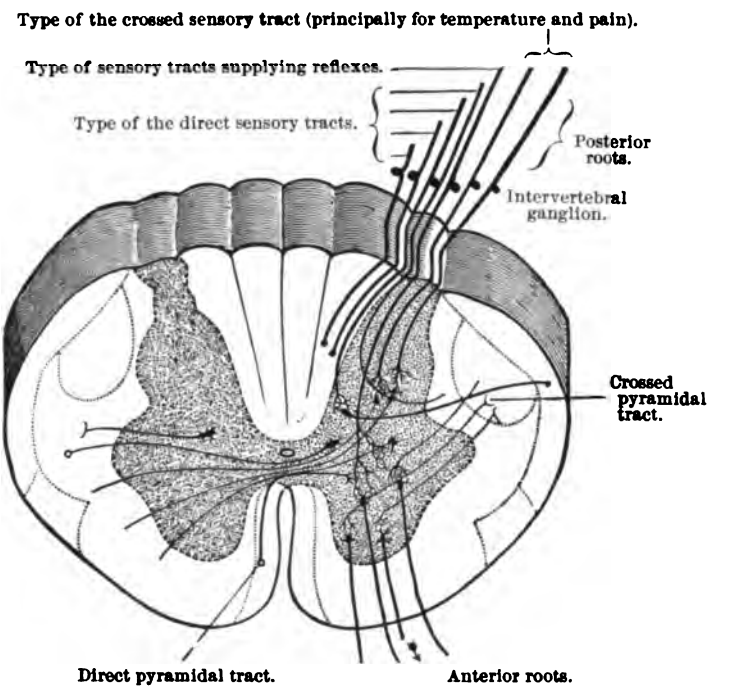
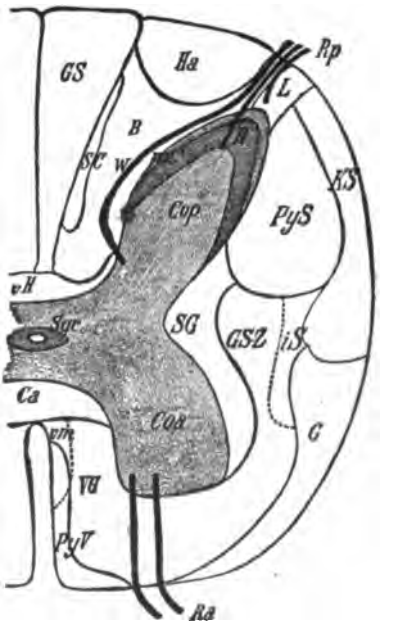


FIG. 362.—Crossed section of the cord (Edinger). The interpretation for the posterior roots not according to Edinger.



B, Burdach's column; *Ca*, anterior commissure; *Coa*, anterior cornu; *Cop*, posterior cornu; *GS*, column of Goll; *GSZ*, mixed lateral tract; *Ha*, postero-external field; *iS*, intermediate lateral tract; *KS*, direct cerebellar tract; *L*, Lissauer's marginal zone; *PyS*, lateral pyramidal tract in marginal zone; *PyV*, anterior pyramidal tract; *R*, substantia gelatinosa Rolandi; *Ra*, anterior roots; *SC*, Schultze's comma; *SG*, lateral boundary zone; *Sgc*, substantia gelatinosa centralis; *VG*, anterior ground bundle; *vH*, central substance behind the columns; *vm*, fasciculus subcomarginalis; *Rp*, and *W*, posterior roots (after Obersteiner).

FIG. 363.—Cross-section of the cervical cord.

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(*b*) **Segmental Localization of the Spinal Cord.—Longitudinal Localization.**

RECENT VIEWS.

Segmental Localization of Cutaneous Sensibility.—The behavior of the cutaneous sensibility in lesions of separate sensory roots—*i. e.*, of separate segments of the spinal cord—has been very minutely studied recently by Sherrington,[1] Thoburn,[2] and Kocher.[3] Sherrington has shown experimentally that individual sensory roots—*i. e.*, segments of the dorsal cord—supply a circular girdle-shaped area of the skin upon the trunk, and that the individual segments overlap each other in both directions. Hence the upper boundary of the disturbance of sensibility, in cross-lesions of the spinal cord, does not follow the descending direction of the ribs, but presents a girdle-shaped line perpendicular to the body axis. (See Fig. 364.) On account of the overlapping of the separate zones of sensibility, we find that in cross-sections of the spinal cord a zone of relatively disturbed sensibility can always be distinguished above, between the boundary of the absolute loss of sensibility and the higher zone of hyperesthesia. This zone of relatively disturbed sensibility corresponds to the area which is supplied also by the segment lying above the lesion, and which is deprived of the lower part of its double innervation. As is shown by the figure, the rules for the segmentary arrangements of disturbance of sensibility do not hold good for the extremities or the neck and head. However, if the arms are horizontally abducted, these rules still apply (Fig. 364).

In almost complete conformity with the experiments of Sherrington and with the clinical observations of Thoburn, Kocher, from minute investigation of traumatic lesions of the spinal cord, has represented in Fig. 364 the cutaneous areas corresponding to the separate spinal cord segments—*i. e.*, the sensory roots. We should supplement this figure by including the overlapping of the zones, as determined by Sherrington. In Fig. 364 the boundary between each segment and the one next below corresponds, according to Kocher, to the upper limits of the absolute disturbance of sensibility which he found in individual cases of transverse lesions of the spinal cord—*i. e.*, to the lower limits of the innervation area of the upper segment. Therefore all these boundaries should be understood as representing the lower limits of the upper zone—*e. g.*, the boundary between the seventh and eighth dorsal zones corresponds to the lower boundary of the seventh zone—*i. e.*, to a level to which the seventh zone still sends its prolongations in lesions of the eighth segment, as a result of the double innervation; but the upper boundary of the eighth innervation area should be sought somewhat farther upward. The upper sensibility girdle must therefore be understood to overlap the lower, like shingles on a roof, although in the figure only the uncovered parts are represented.

[1] *Philosophical Transactions of the Royal Society*, vol. clxxxiv., London, 1893.

[2] *A Contribution to the Surgery of the Spinal Cord*, London, 1889, Griffin & Co.; again, Brain, 1893 and 1894.

[3] "Die Verletzungen der Wirbelsäule, zugleich als Beitrag zur Physiologie des menschlichen Rückenmarks," *Mittheilung aus den Grenzgebieten der Medicin und Chirurgie*, vol. i., part 4.

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The relation of the plane of the sensibility zones of the skin—*i. e.*, of the anesthesia in lesions of the sensory roots or *focal* lesions of the cord—to the plane of the corresponding spinal cord segments, spinal roots, and vertebræ is of the greatest importance. Fig. 364 and the

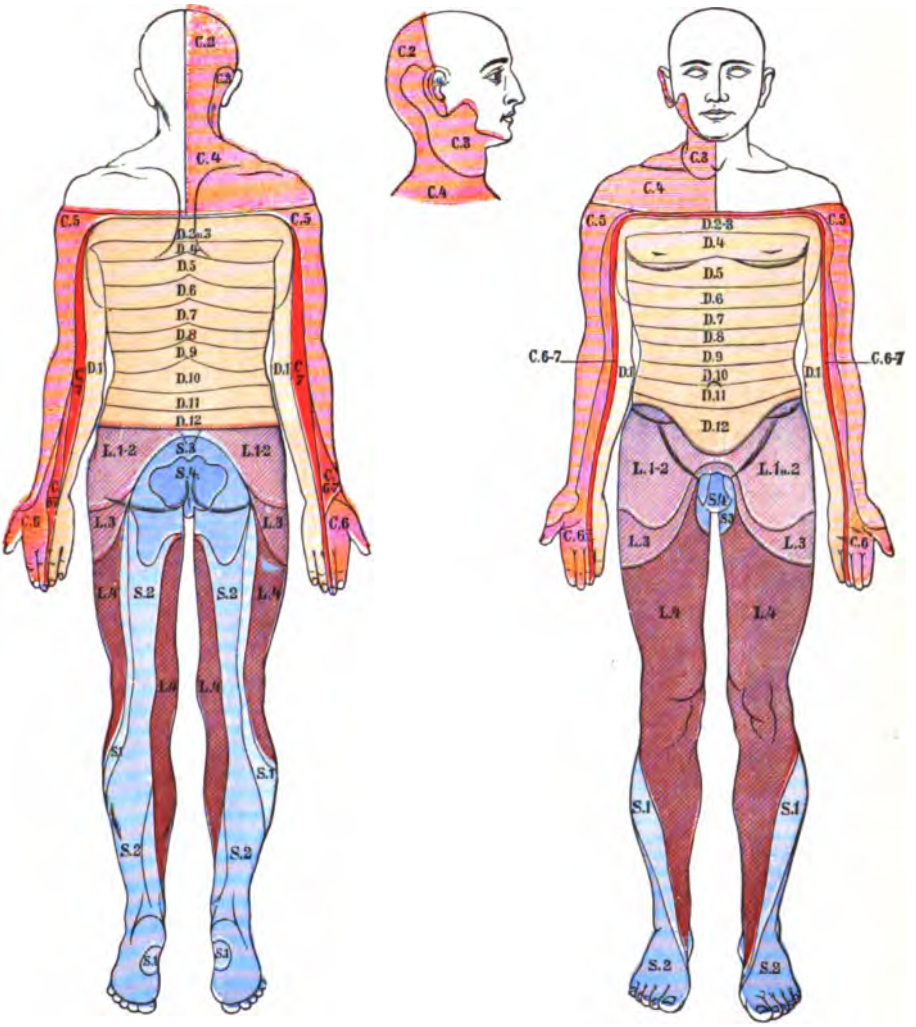

FIG. 364.—The sensory innervation of the body by the spinal segments according to Kocher.
Red: Cervical segments.
Brown: Dorsal "
Violet: Lumbar "
Blue: Sacral "
} The intensity of the color depends upon the level of the segment.
C_2, D_2, L_2, S_2, etc.=Second cervical, dorsal, lumbar, sacral segment, etc.

following pages demonstrate plainly enough that the injured spinal cord segment, the place of exit of the corresponding sensory root, or the vertebra corresponding numerically to the injured segment, and, finally, the cutaneous sensibility border do not lie in a horizontal plane, but that the

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cutaneous sensibility is pushed considerably downward in relation to the vertebra or the exit of the nerve, and this again in relation to the segment of the cord. In the operative treatment of certain spinal cord affections, a neglect of this peculiarity has occasioned many unfortunate results. The plainest rules for use in such conditions have been recently formulated by Kocher. They will be given later.

One reason for such caudal dislocation of the limits of cutaneous sensibility, as compared with the affected segment, depends upon the fact that the spinal cord is so much shorter than the vertebral column that the nerve roots within the vertebral canal must take a descending course. As a result (see Fig. 365) each segment up to the fourth or fifth upper dorsal vertebra is situated at the level of the next higher vertebra. Thus, the first dorsal root arises from its segment in the spinal cord behind the seventh cervical vertebra, the sixth dorsal root behind the fifth dorsal vertebra, etc. From the fourth or fifth vertebra downward the segments lie still higher in relation to the corresponding vertebra, so that the eighth dorsal segment lies behind the upper part of the seventh dorsal vertebra; the ninth segment, behind the cartilage between the seventh and the eighth vertebra; the tenth segment, behind the lower part of the eighth; the eleventh, behind the ninth; the twelfth, behind the tenth vertebra. Thus, in the upper half of the dorsal column the difference of level between the segment and the corresponding vertebra is equal to the height of one vertebra; whereas in the lower dorsal column it approaches more and more the height of two vertebræ.

This variation of level between the segment and the corresponding vertebra naturally shows at the same time the difference in height between the segment and the exit of the corresponding roots.

Now, the upper boundaries of the absolute sensibility disturbance which correspond to a lesion of a certain nerve root—*i. e.*, of its corresponding segment—are again lower than the point of exit of the nerve root, because the intercostal nerves take a still further descending course to reach the skin, and because the unaffected root which lies above overlaps about one finger's breadth the area supplied by the affected root. In consequence of this in lesions of a sensory root, or in transverse lesions of the dorsal cord, the limitations of sensibility (upper border of absolute anesthesia) are in the upper dorsal region about three, in the lower dorsal from four to five, vertebral heights below the points of exit of the affected roots—*i. e.*, the uppermost segments affected. Since the spinal cord segments in the upper dorsal column are situated about one vertebral height, and in the lower dorsal two vertebral heights, above the exit of the corresponding nerve root, the affected segment in cross-lesions of the upper part of the dorsal cord is situated about four (3+1), and of the lower part of the dorsal cord from six to seven, vertebral heights above the upper boundaries of absolute anesthesia.

These relations are represented diagrammatically in Fig. 365.

This diagram corresponds with the rules empirically established by

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Kocher—viz., that in cross-lesions of the dorsal cord—*i. e.*, in lesions of its sensory roots—the upper boundaries of absolute sensibility disturbance correspond to the deepest (nearest the end of the spine), and to the most anterior point (for the upper ribs) of the intercostal space in which the intercostal nerve belonging to the affected root runs. From this level the limitations of sensibility run horizontally backward—*i. e.*, perpendicular to the vertebral column, and not obliquely like the ribs. For those roots whose intercostal spaces do not reach the sternum, we must determine the boundaries anteriorly, corresponding to the supply of the abdominal wall, by following the intercostal

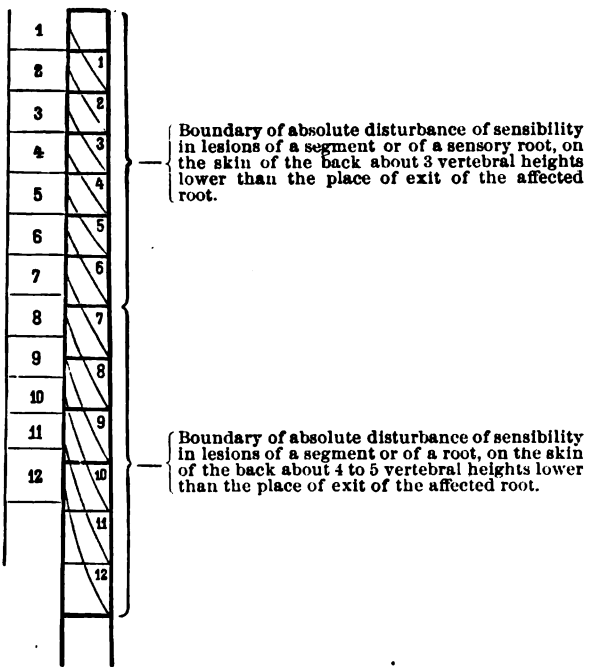

FIG. 365.—Diagram of the level of the dorsal segments in relation to the dorsal vertebræ and to the corresponding boundaries of the zones of insensibility on the back. Drawn from Kocher's rules. The oblique lines represent the emerging roots.

nerve downward in an outward convex curve—*e. g.*, the boundaries corresponding to the twelfth intercostal nerve will reach down to the symphysis. In reality, Kocher's rules hold good even here, because the borders run practically horizontally backward from the deepest point of the intercostal space.

From what has been said about the variations in levels between the exit of the root and the corresponding segment, it is clear that one and the same upper boundary of anesthesia may signify lesions at different planes, depending upon whether the spinal cord or a root is involved. This point is of some importance in operative cases, as it adds an element of uncertainty in determining the appropriate point for an

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attempted incision. Of course, additional surgical evidence, such as a fracture of a vertebra, dislocation, or spondylitis, frequently indicates the proper place for the incision. When in doubt, it is advisable to begin the incision midway between the vertebra and its corresponding segment, and from there to enlarge the field of operation according to the findings.

There is still another difficulty in local diagnosis that is worth mentioning. In incomplete spinal cord lesions, which involve the central parts of the section of the spinal cord more than the periphery, the disturbance of sensibility begins considerably deeper than we should expect from the above rules, because some of the sensory fibers of the central parts of the white substance take their exit lower down than those from the periphery. Such a preponderance of involvement of

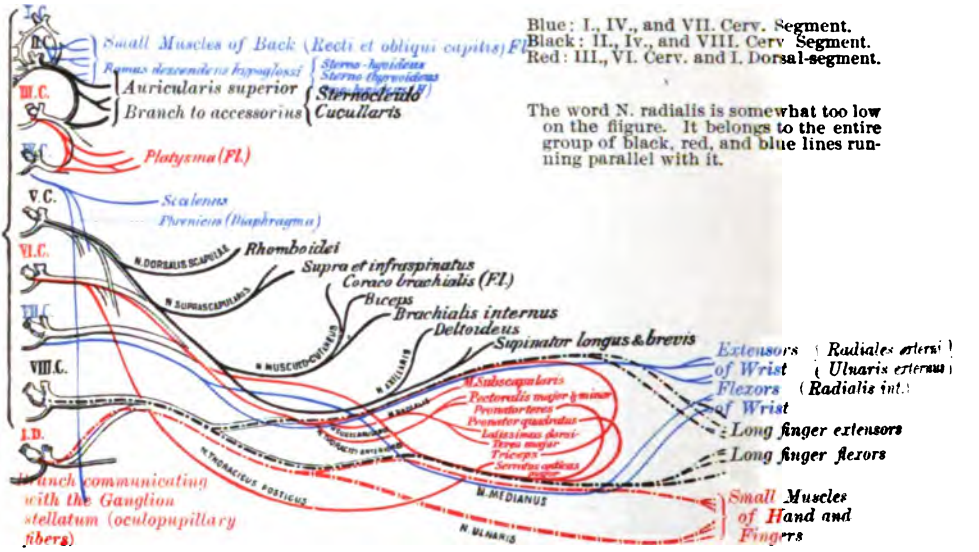

FIG. 366.—Spinal motor nerves from cervical plexus (Kocher).

the central parts of the spinal cord is observed not only in lesions which actually proceed from the interior of the cord (central myelitis and hemorrhage), but even in traumatic and spondylitic pressure lesions, probably because the central portions are more susceptible to injury than the periphery. So that it scarcely needs to be emphasized that the rules for localization are to be applied strictly only when we are convinced that the transverse lesion affects the entire cross-section equally. This is practically very difficult. Among other aids, the condition of the reflexes may be utilized to decide the question. The more complete the involvement of the spinal cord cross-section the more decidedly the reflexes are affected. (See p. 784 *et seq.*)

Segmental Localization of Motility.—Kocher has represented the motor segmental localization in the two plates Figs. 366, 367). They

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are based upon his own observations and upon the well-known atlas of Flower[1] and the works of Risien Russel[2] and Thoburn.[3] The names of the great nerve trunks composed of the fibers from the different roots are printed in black capital letters, while the names of the individual branches are printed in italics and agree in color with the corresponding segments—*i. e.*, the corresponding motor roots.

According to Kocher, these plates illustrate the following facts about the segmental motor innervation of the dorsal cord: I.–XII.

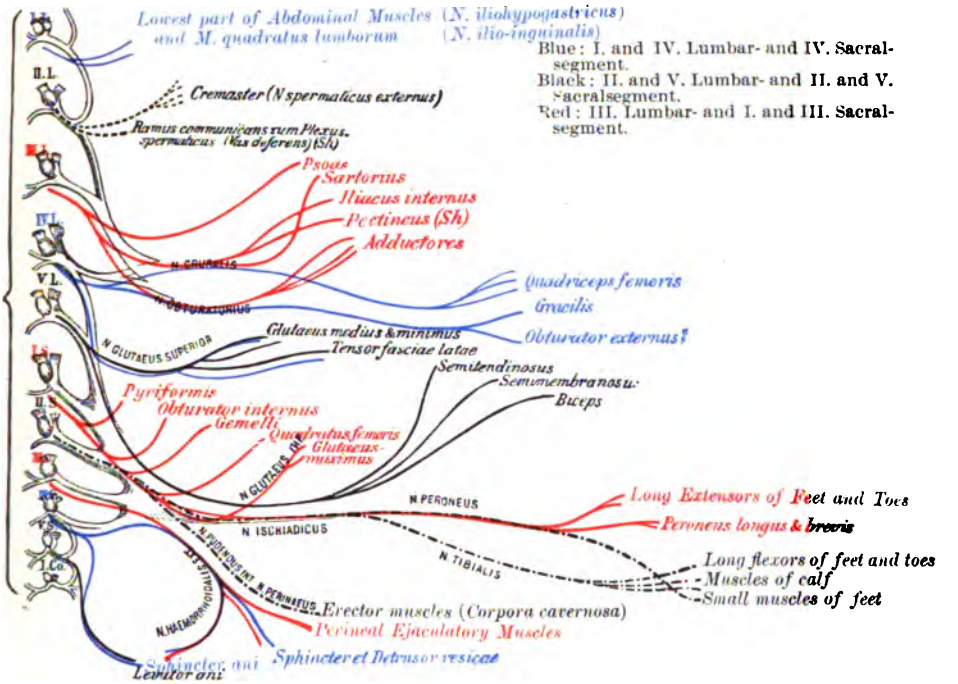

FIG. 367.—Spinal motor nerves from lumbar and sacral plexus (Kocher).

dorsal segments supply the intercostal muscles; VII.–XII. dorsal segments supply the abdominal muscles; I.–IV. dorsal segments furnish the sympathetic nerves to the head, neck, heart, and lungs; V.–IX. dorsal segments furnish the sympathetic nerves to the intestinal canal and to the abdominal glands (superior splanchnic nerve); X.–XII. dorsal segments furnish sympathetic nerves to the testicles, bladder, and rectum (inferior splanchnic nerve, internal spermatic plexus, and inferior mesenteric plexus).

Segmentary Localization of the Reflexes.—By the diagrams

[1] *Atlas schématique de système nerveux*; translated by Duprat and Déjérine.

[2] "Experimental Investigation of the Nerve Roots of the Lumbosacral Plexus," etc., *Proc. of the Royal Society*, vol. liv.; and "Experimental Investigations of the Nerve Roots of the Brachial Plexus of the Dog," Pathological Laboratory of University College, 1892.

[3] *Loc. cit.*

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of the motor and sensory segmental innervation of the spinal cord (Figs. 364, 366, and 367, etc.), the reflexes may be localized at the appropriate segments. Their value for the local diagnosis of the level of a cross-lesion is evident. The shortest reflex tracts in the spinal cord must be contained in that portion situated between the entrance of the sensory and the motor roots which conduct the reflex in question. If one knows the segment which perceives the sensory irritation exciting the reflex and the segment which sends out the motor fibers inner-

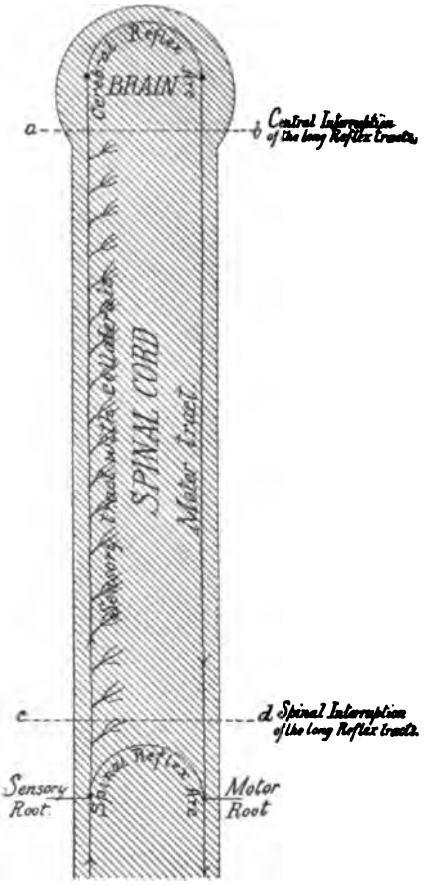

FIG. 368.—Diagram of a cerebronuclear (cutaneous) reflex to illustrate the connection between short (segmental) and long reflex tracts.

In this diagram only the cerebral reflex arc is represented completely. But the numerous collaterals (supplied with terminal twigs) show that, in addition to the shortest (segmental) circuit and to the cerebral arc, numerous spinal lateral circuits must be taken into account. This shows how incorrect it is to speak of reflex centers.

This figure should explain at the same time the doctrine of reflex stasis in transverse lesions of the cord which was discussed upon p. 783. It explains why beneath the lesion a reflex stasis—*i. e.*, increase, and, later, abnormality of the reflexes, and, finally, pathologic reflexes—would result from a transverse lesion (*c d*); whereas, if the lesion is at *a b* in the brain, there will be no reason for any reflex stasis, because of the countless paths of escape for the sensory irritation. Therefore, a destruction of the cerebral arc is much more apt to enfeeble the reflexes. For the sake of simplicity we have omitted any representation of the bilateral course of the tracts.

vating the reflex movement, then between the two must be situated the shortest intraspinal reflex tract. This was formerly spoken of as the "reflex center." There is, of course, no question of any actual reflex center, for we have learned that there is no actual transformation of the sensory reflex impulses in one group of ganglion cells directly to the motor limb of the so-called reflex arc. At most, such an idea can assist our conception only as a diagram does. In reality, the reflex tracts comprise numerous ganglion cells intercalated one after the other,

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as well as manifold lateral connections. (Compare p. 779 *et seq.* for the modern idea of the origin of the reflexes.) The shortest path of a reflex lies between the entrance of the sensory and the exit of the motor root of the reflex arc; but this does not mean that in normal cases the reflex always proceeds only by this shortest path. On the contrary, we have already seen upon p. 783 *et seq.* that an upper reflex arc reaching to the brain must ordinarily be innervated at the same time, and that the shortest path can be exclusively affected only in interruptions of spinal cord conduction, as a result of reflex stasis. Fig. 368 may recall this conception to the reader. It has been more minutely explained upon p. 783. Without any further explanation, it is evident that only for the tendon reflexes does the interference in the reflex arc normally occur exclusively by the shortest way (between the entrance of the sensory and the exit of the motor roots). Clinically these shortest reflex paths are important, for, as shown in Fig. 368, in a complete cross-lesion of their segment of the spinal cord the reflexes in question must be lost, because naturally all longitudinal accompanying circuits are interrupted as well. On the contrary, we must emphasize the fact that if the shortest path is anatomically intact, the reflex in question may be either preserved or lost, depending upon whether the reflex normally proceeds by the shortest path or by a longer path, whether the latter is free or interrupted, and whether the reflexes are diminished by the indirect action of an area lying farther above, which causes inhibition or circulatory disturbances, or (p. 779), conversely, reflex stasis. In other words, preservation of a certain reflex shows that the segment of the spinal cord uniting its motor and sensory nerve roots must still possess, at least, partial conductivity; whereas the loss of such a reflex suggests an interruption of the corresponding segment of the spinal cord, although it does not in any way prove such interruption with certainty.

Older Views.

In concluding this presentation of the segmental localization of the spinal cord, derived from the most recent sources, it seems necessary to include in the following pages the most essential of the older views upon this topic. The frequent contradictions which exist between individual views prove how unsettled this question still remains, and show that accurate clinical and pathologico-anatomic examinations will not only supply numerous corrections, but will extend our knowledge as well. These findings, especially where they concern the reflexes and their relation to the segments, must be critically examined for light upon the genesis of the reflexes (see pp. 779 and 783). Thus far the comparison of the conformity and non-conformity of individual views has been the only method of judging the accuracy of the data. In the discussion certain experiments performed upon animals are not included, because they are less suitable for criticism. In this connection the observations made by Ferrier and Yeo upon apes by irritating the motor-nerve roots are especially valuable, particularly because many other experiments, especially the well-known researches of Horsley and Beevor upon the cerebral cortex, show the most intimate analogy between the nervous system of apes and that of man. Even the gross anatomy of the plexuses of the extremities must be taken into account, and therefore finds a place in the following:

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CLINICAL DATA.

Localization of the Functions in the Different Spinal-cord Segments (from Starr-Edinger-Bruns).[1]

Segments.	Motor roots for—	"Reflex centers"[2] for—	Sensory roots (cutaneous innervation) for—
I. Cervical nerve.	Small muscles of the neck, Sternocleidomastoid and trapezius.		
II.–III. Cervical nerves.	Sternocleidomastoid, Trapezius, Scaleni and neck muscles (complexus, splenius, longus colli).	. . . . .	Neck and occiput.
IV. Cervical nerve.	Complexus, Splenius, longus colli, Levator scapulæ, Diaphragm, Supra- and infra-spinati, Deltoid, Biceps and coracobrachialis, Supinator longus, Rhomboidei.	Dilatation of the pupils from a sensory stimulation of the neck (IV.–VII. Cervical nerve).	Neck, Upper-shoulder region, Outside of the arm to the second rib.
V. Cervical nerve.	Diaphragm, Deltoid, Biceps, brachialis anticus, Coracobrachialis, Sup. longus and brevis, Pect. major (pars clavicul.), Serratus magnus, Rhomboidei, Teres minor, Latissimus dorsi.	Scapular reflex (V. Cervical nerve to I. Dorsal nerve). Tendon reflexes of the muscles and tendons about the elbow-joint (V.–VI. Cervical nerve).	Posterior aspect of the shoulder and arm. Outside of the arm and forearm.
VI. Cervical nerve.	Biceps, Brachialis anticus, Pectoralis major (pars clavicul.), Serratus anticus major, Triceps, Extensors of the hand and fingers, Pronators.	Extensor reflexes of the arm and forearm.	Outside of the forearm.
VII. Cervical nerve.	Long head of the triceps, Extensors of the hand and finger, Flexors and pronators of the hand, Pectoralis major (pars cost.), Subscapularis, Latissimus dorsi, Teres major.	Flexor reflexes.	Radial area of the hand and part of the median area.

[1] Starr collected the older data from the clinical examinations of localized spinal-cord lesions, which are recorded as late as 1888.

[2] This expression (see p. 928 *et seq.* and Fig. 368) is incorrect, and signifies here merely the shortest reflex arc, and therefore that segment of the cord which includes the sensory and motor roots of the reflex in question.

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Localization of the Functions, etc. (Continued).

Segments.	Motor roots for—	"Reflex centers" for—	Sensory roots (cutaneous innervation) for—
VIII. Cervical nerve.	Flexors of the hand and fingers, Small muscles of the hand.	Dilator of the pupil and smooth muscles of the orbit, with I. Dorsal nerve.	Median area, Ulnar area.
I. Dorsal nerve.	Extensors of the thumb, Small muscles of hand, Muscles of ball of thumb and little finger.	Dilator of the pupil and smooth muscles of the orbit in junction with VIII. Cervical nerve.	Ulnar area.
II.–XII. Dorsal nerve.	Back muscles, Abdominal muscles.	Abdominal reflexes in the IV.–XI. dorsal segments; according to Dinkler, in the IX.–XII. Dorsal segments.	Skin of the breast, back, abdomen, and upper gluteal region.
I. Lumbar nerve.	Abdominal muscles, Psoas, Sartorius.	Cremaster reflex.	Skin of the pubic region, anterior surface of the scrotum.
II. Lumbar nerve.	Psoas, Sartorius, Flexors of the knee (Remak?) Quadriceps femoris.	Cremaster reflex. Patellar tendon reflex (II. – IV. Lumbar nerve).	Outside of the hip region.
III. Lumbar nerve.	Quadriceps femoris, Psoas and pectineus, Inward rotators of the thigh, Abductors of the thigh.	Patellar tendon reflex (II. – IV. Lumbar nerve).	Anterior and inner side of the hip region.
IV. Lumbar nerve.	Adductors and abductors of the thigh, Tibialis anticus femoris, Peroneus longus, Flexors of the knees (Ferrier?).	Patellar tendon reflex (II. – IV. Lumbar nerve). Gluteal reflex (IV.–V. Lumb. nerve).	Inner side of the hip and leg as far as the ankle. Inner side of the foot.
V. Lumbar nerve.	External rotators of the hip, Flexors of the knees (Ferrier?), Flexors of the foot, Extensors of the toes, Peronei.	Gluteal reflex (IV.–V. Lumb. nerve).	Posterior side of the hip, of the thigh, and external part of the foot.
I.–II. Sacral nerves.	Flexors of toes and foot, Small muscles of feet, Peronei.	Plantar reflex. Bladder and rectal center (Sarbo).	Posterior side of the thigh, outside of the leg and foot.
III.–IV. Sacral nerves.	Perineal muscles.	Achilles tendon reflex. Bladder and rectal center.	Posterior side of the scrotum, Perineum, anus, sacral region.

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W. Thoburn[1] has obtained similar results to Kocher.

BRACHIAL PLEXUS (Motor Innervation).

Cervical nerves:	
IV.	Supra- and infraspinatus, teres minor (?).
V.	Biceps, brachialis anticus, deltoid, supinator longus, supinator brevis.
VI.	Subscapularis, pronatores, teres major, latissimus dorsi, pectoralis major, triceps, serratus major.
VII.	Extensors of the wrist.
VIII.	Flexors of the wrist.
Dorsal nerve:	
I.	Small muscles of the hand.

LUMBOSACRAL PLEXUS.

	Motor distribution.	Sensory distribution.
Lumbar nerves:		
I.	(?)	The iliohypogastric and ilio-inguinal nerves.
II.	(?)	External (?) and upper region of the thigh.
III.	Sartorius, Adductors, Flexors of the thigh.	Anterior surface of the thigh, below the region supplied by the II. Lumbar nerve.
IV.	Extensors of the knee, Abductors of the thigh.	Anterior and inner surface of the thigh.
V.	Flexors of the leg.	Posterior side of the thigh, except the territory supplied by the sacral roots.
Sacral nerves:		
I. }	Calf muscles, Gluteal muscles, Perineal muscles, Dorsal flexors of the ankle-joint, Small muscles of the foot.	Narrow strip on the posterior surface of the thigh and leg, sole of foot, A part of the dorsum of the foot.
II. }		
III. }	Nervi erigentes, Perineal muscles.	Perineum, external genitals.
IV.	Bladder and rectum.	Posterior surface of the thigh.

Dinkler's[2] observations, made in Erb's Clinic, upon the localization of the abdominal reflexes, should be mentioned. He found that the abdominal reflexes have their shortest circuit in the lowest part of the dorsal cord, that the middle and lower abdominal reflexes (p. 777) belong to the X., XI., and XII. intercostal nerves and their corresponding spinal segments, while the upper abdominal reflex is limited to the tract of the IX. and possibly, also, of the VIII. dorsal nerves. This tallies with the segment localization of the corresponding cutaneous areas (see Fig. 364, p. 923). Still, these statements apply only to the shortest circuit of the reflex arc and do not exclude the longer reflex arcs reaching further up through the affected sections.

For the local diagnosis of lesions of the cervical cord the reader should consult Kraus's article (from Kahler's Clinic), *Zeits. f. klin. Med.*, 1891, vol. xviii., p. 343. In conformity with Fraulein Klumpke's experiments on animals, he found that in man the sympathetic oculopupillary fibers, whose paralysis causes myosis and retraction of the globe with sympathetic ptosis (Paralysis of Müller's Smooth Orbital Muscle, see p. 833), leave the spinal cord with the motor root of the first dorsal nerve. These are the fibers which connect the principal trunk of the sympathetic with the so-called ciliospinal center of the spinal cord. The latter was localized by Budge between the sixth cervical and the second dorsal segments. Kraus's statements coincide with L. Jakobsohn's[3] anatomicopathologic

[1] *A Contribution to the Surgery of the Spinal Cord*, London, 1889.
[2] *Deutsch. Zeits. f. Nervenheilk.*, 1892, vol. ii., p. 325.
[3] *Zeits. f. klin. Med.*, 1899, vol. xxxvii.

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Gowers gives the following table:[1]

Motor functions.	Nerves.	Motor functions.	Nerves.	Sensory functions.	Reflex.
Sternomastoid, upper neck muscles, upper part of trapezius.	*C* 1	Small rotators of head,	1		1
	2	Depressors of hyoid.	2	Scalp,	2
	3	Levator anguli scapulæ.	3	Neck and upper part of chest.	3
	4	Diaphragm.	4	Shoulder.	4
	5		5	Arm, outer side.	5
Lower neck muscles, middle part of trapezius.		Serratus, Flexors of elbow, Supinators. (Shoulder muscles.)	6	Radial side of forearm and hand, thumb.	6
	6		7	Arm, inner side, ulnar side of forearm and hand, tips of fingers.	7
	7	Extensors of wrist and fingers.	8		Scapular.
		Extensors of elbow,	1		
		Flexors of wrist and fingers,	2	Front of thorax.	8
		Pronators.	3		
	8		4		1
			5		2
			6		3
		Muscles of hand.	7	Ensiform area.	4
	D 1				5 Epigastric.
	2		8	Abdomen (Umbilicus 10).	6
	3		9		7
	4		10		8
	5	Intercostal muscles.	11		9
Lower part of trapezius and dorsal muscles.	6		12		10 Abdominal.
	7			Buttock, upper part.	11
	8				12
	9		1		1
	10	Abdominal muscles.		Groin and scrotum.	Cremaster.
	11		2		2
	12			Outer side.	
			3		3 Knee-jerk.
	L 1			Thigh Front.	
			4		4
	2	Cremaster,		Inner side.	5 Gluteal. Foot-clonus.
	3	Flexors of hip.	5	Leg, inner side.	
Lumbar muscles.		Extensors of knee, Adductors of hip,	1	Buttock, lower part.	
	4	Extensors and abductors of hip.	2	Back of thigh.	
Peroneus I. Flexors of ankle, extensors of ankle.	5			Leg and foot (except inner part.)	1
		Flexors of knee.	3		2 Plantar.
	S 1		4	Perineum and anus.	3
	2	Small muscles of foot.	5	Skin of coccyx and anus.	4
	3	Perineal and anal muscles.			5
	4				
	5				
	Co.		*Co.*		*Co.*

[1] *Handbook of Nervous Diseases*, vol. 1, p. 222.

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findings in man. This author found the lateral horn corresponding to the first dorsal segment diseased in a lesion of the oculopupillary sympathetic fibers.

EXPERIMENTAL DATA.

Ferrier and Yeo,[1] in irritation experiments upon apes, obtained the following results in reference to the assignment of individual muscles to the motor-nerve roots:

MOTOR ROOTS OF THE BRACHIAL PLEXUS (after Ferrier and Yeo).

IV. *Cervical Nerve.*—Deltoid, rhomboids, supra- and infraspinatus, teres minor, brachialis anticus, supinator longus, extensors of the hands and fingers, diaphragm.

V. *Cervical Nerve.*—Deltoid (clavicular portion), biceps, brachialis anticus, serratus anticus major, supinator longus, extensors of the hands and fingers.

VI.—*Cervical Nerve.*—Latissimus dorsi, pectoralis major, serratus magnus, pronators, flexors of the wrist (?), triceps.

VII. *Cervical Nerve.*—Teres major, latissimus dorsi, subscapularis, pectoralis major, flexors of the hand and fingers (median), triceps.

VIII. *Cervical Nerve.*—Long flexors, ulnaris internus, small muscles of hand, extensors of the hand and fingers, long head of the triceps, pectoralis major (?).

I. *Dorsal Nerve.*—Small muscles of the hand.

MOTOR ROOTS OF THE LUMBOSACRAL PLEXUS (after Ferrier and Yeo).

III. *Lumbar Nerve.*—Psoas, sartorius, adductors, extensor cruris.

IV. *Lumbar Nerve.*—Extensors of the thigh, extensor cruris, peroneus longus, adductors.

V. *Lumbar Nerve.*—Flexors and extensors of the toes, tibial muscles, calf muscles, peroneal muscles, external rotators of the thigh, flexors of the leg (biceps, semitendinosus, etc.).

I. *Sacral Nerve.*—Calf muscles, flexors of the leg, flexor hallucis longus, small muscles of the foot.

II. *Sacral Nerve.*—Small muscles of the foot.

Dastre and Morat found in experimenting upon animals that most of the vasomotor and sweat-secretory fibers for the face first leave the cord with the second to sixth dorsal nerves.

Additional experimental results worth mentioning are those obtained by Nawrocki and Skabitschewsky upon the origin of the motor and sensory fibers for the bladder of the cat. They found that the motor nerves of the bladder leave the spinal cord by two paths—an upper and a lower. The upper leads to the vesical plexus from the fourth and fifth anterior lumbar roots; the lower from the second and third anterior sacral roots. The sensory fibers are partly sympathetic, partly cerebrospinal, in origin; the former are contained exclusively in the hypogastric nerve, the latter in the four upper posterior sacral roots. (See, also, L. B. Müller's data, on p. 944, *f.*)

Rossolimo found in dogs that the center for the *anal reflex* (p. 778) was situated in the region of the third and fourth sacral nerves.

PURELY ANATOMIC DATA.

Herringham[2] obtained the following results in regard to motor innervation from purely anatomic researches upon the course of the individual motor-nerve roots through the brachial plexus:

V. *Cervical Root.*—Biceps, brachialis anticus, subscapularis, deltoid.

VI. *Cervical Root.*—Pectoralis major, biceps, brachialis anticus, pronator

[1] *Brain*, 1882, vol. iv., p. 226. [2] *Proc. Royal Soc.*, 1886, No. 243, p. 225.

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teres, radialis internus, thenar muscles, subscapularis, teres major, deltoid, supinator longus and brevis, radialis externus, longus and brevis.

VII. *Cervical Root.*—Pectoralis major and minor, coracobrachialis, flexor sublimus, latissimus dorsi, triceps, radialis externus, longus and brevis.

VIII. *Cervical Root.*—Pectoralis major and minor, flexor sublimus, latissimus dorsi, triceps.

I. *Dorsal Root.*—Pectoralis major and minor.

At all events, we have anatomic examinations to thank for the knowledge that the *phrenic nerve*, to a large extent, derives its fibers from the *fourth motor*

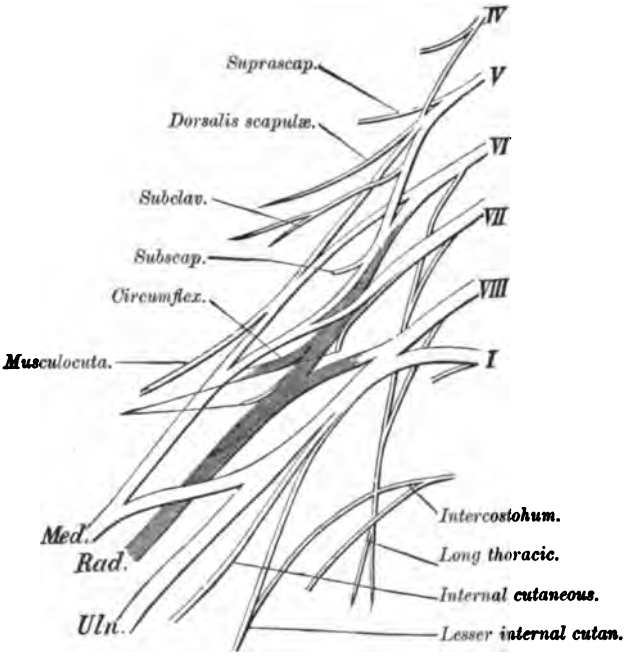

FIG. 369.—Plexus brachialis: *IV.*, *V.*, *VI.*, *VII.*, *VIII.*, *I.*, Anterior branches of the five lowest cervical nerves, and of the *I.* dorsal nerve. Of the branches extending anteriorly, only the subclavius nerve is represented (after Gegenbaur).

cervical nerve root, and sometimes a part from the third and fifth; whereas, the *occipitalis magnus* derives its fibers from the second, the *occipitalis minor* from the first, second, and third, and the *auricularis magnus* from the third and fourth *sensory* cervical nerve roots.

The annexed figures (369 and 370) and Gegenbaur's *Lehrbuch der Anatomie* may be consulted for a general idea of the origin of the plexuses for the extremities, and their subdivisions into the peripheral nerves.

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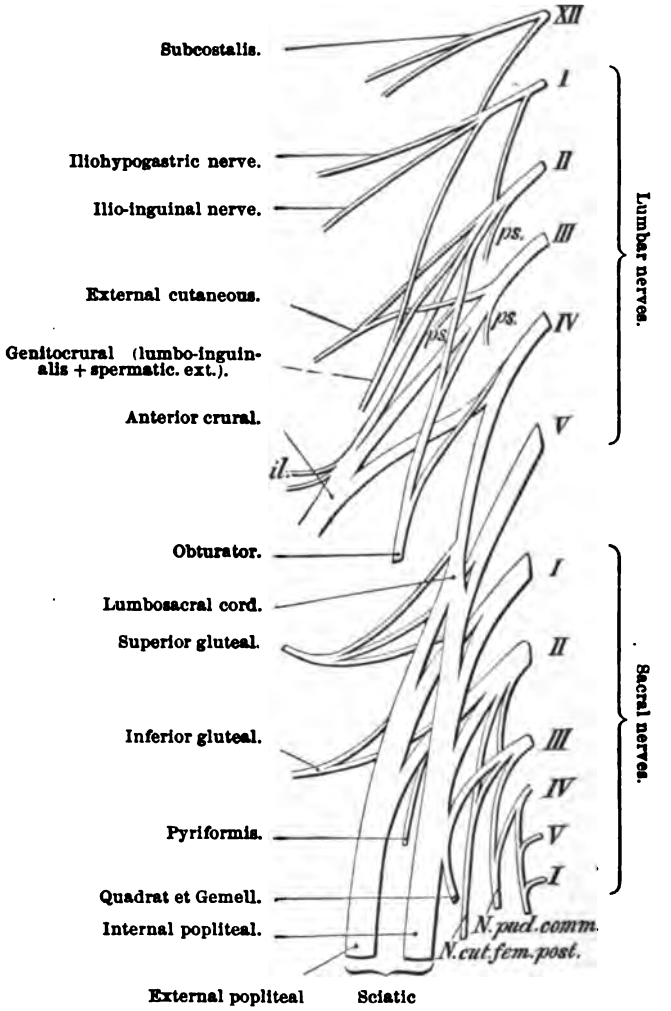

FIG. 370.—Lumbosacral plexus: *ps*, Branches to the psoas muscle; *il*, branches to the iliac muscle (after Gegenbaur).

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(c) Topography of the Lumbosacral Cord, of the Conus Terminalis, and of the Cauda Equina.

In order to make a differential diagnosis and determine the definite localization of affections of the cauda equina and of the lumbar and sacral cord, it is very essential to possess an exact knowledge of the topographic relations of this region and especially of the situation of the points of origin of the lumbar and sacral nerves from the cord, as compared with the vertebræ or the points of exit of the nerve roots from the spinal-cord canal. The following plates will serve for orientation.

The limits of the conus terminalis have not yet been very sharply defined.

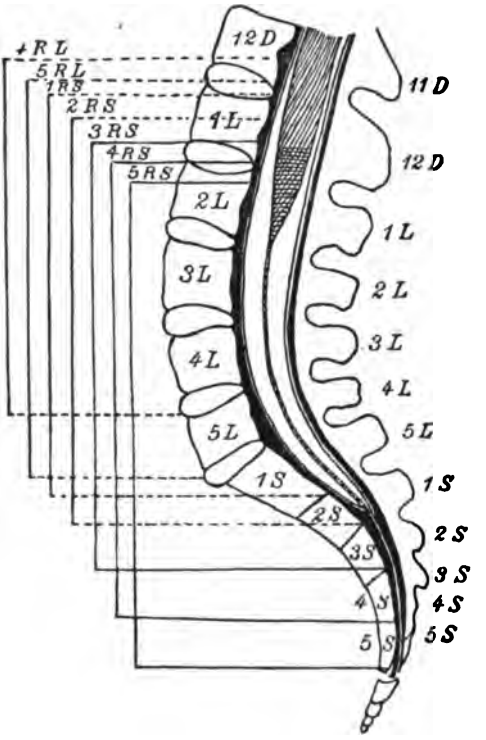

FIG. 371.—The lower end of the vertebral column and its topographic relation to the lumbar and sacral cord, and to the origins and exits of the lumbar and sacral nerves. The double-lined portion represents the conus terminalis, according to Raymond's definition (see the text), to which is attached the filium terminale. Between it and the wall of the vertebral canal (the part in white) the cauda equina passes. The upper horizontal lines indicate the heights at which the nerve roots spring from the spinal cord; and the lower horizontal lines connected with the upper ones by means of vertical lines indicate the exits of the corresponding roots from the vertical column. The vertical lines thus represent the lengths of the separate roots of the cauda equina (after Raymond [1]).

Raymond puts its upper borders sufficiently high to include the centers, or the medullary origins of the last three sacral nerves. Such delimitation conforms both with the requirements of descriptive anatomy—*i. e.*, with the boundaries of the actual wedge-shaped terminal portion of the spinal cord—and with those of clinical experience—*i. e.*, the clinical picture of a lesion of the second lumbar vertebra.[2] In lesions of the conus terminalis, both motility and sensibility of

[1] Raymond, "Sur les affections de la queue de cheval," *Nouvelle inconographie de la Salpetrière*, 1895, Nos. 1 and 2.

[2] A Schiff (from Schrötter's Clinic), "A case of Hematomyelia of the Conus Terminalis," *Zeits. f. klin. Med.*, 1896, vol. xxx., p. 87.

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the lower extremities are practically intact, but the functions of the bladder and rectum are disturbed, and there is an anesthesia of the perineum, the inferior

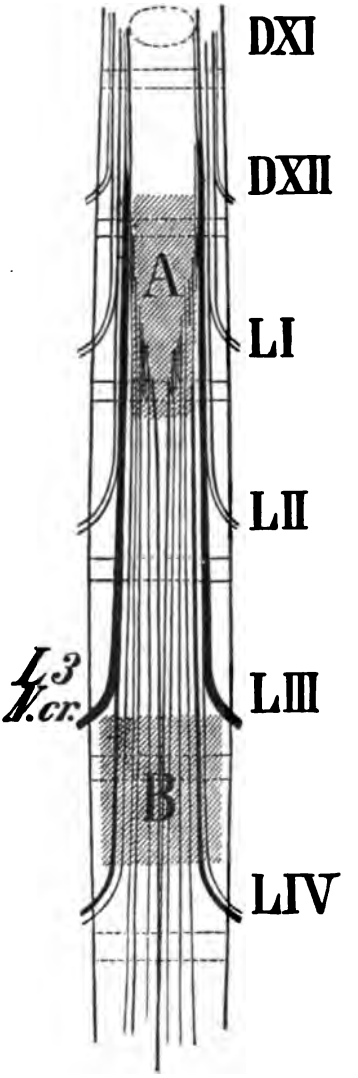

FIG. 372.—Topography of the cauda equina, showing its relation to the vertebral column and to the lower end of the spinal cord (⅔ natural size). Schultze gives the following explanation: "The lower end of the lumbar enlargement is found at the level of the first lumbar vertebra; the third lumbar nerve (heavy black line), with its crural and obturator fibers, arises at different levels, as indicated, in the upper part of the lumbar enlargement, according to Gerlach between the spinal processes of the eleventh and twelfth dorsal vertebræ, a height which, according to the original investigations of Schiefferdecker, corresponds to the lower part of the body of the twelfth dorsal vertebra" (after Schultze and Schiefferdecker [1]). The position of the conus terminalis is higher here than in Fig. 371.

gluteal region, and of a zone upon the back of the thigh, which is supplied by the posterior femoral cutaneous nerve. This does not entirely coincide with the

[1] Schultze, "Zur Differentialdiagnose der Verletzungen der Cauda equina und der Lendenanschwellung," *D. Zeitschr. f. Nervenheilk.*, 1894, vol. v., p. 247.

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topography of sensibility disturbance pictured upon p. 923. A lesion of the lower part of the cauda equina will, of course, produce a similar clinical picture.

Fig. 372 shows, according to Schultze, that still other clinical pictures can be produced by lesions of the cauda equina, as well as by lesions of the lowest part of the spinal cord. It is quite plain from the drawing that the lesion A, at the most inferior portion of the spinal cord, and the lesion B, at the height of the third lumbar vertebra, which injures the cauda equina, will produce the same motor and sensory disturbances of the sciatic region and will not involve the crural and obturator region (II.–IV.). A hyperesthesia of the crural region, or a very slight initiatory involvement of it, may occur in either case, because the lesion A (represented by shading) affects the spinal cord itself above the origin of the crural fibers, whereas the lesion B involves the same fibers laterally and lower down at the height of the third lumbar vertebra.

4. THE VESICAL AND RECTAL FUNCTIONS.

The Mechanism of the Vesical and Rectal Functions under Physiologic and Pathologic Conditions.

It is difficult to understand the vesical and rectal disturbances occurring in diseases of the nervous system, since our knowledge of the physiologic evacuation of the bladder and rectum is still quite incomplete and contains much that is hypothetic. In studying this subject we are confronted by a special difficulty, because the old teaching that the central mechanism for the vesical and rectal functions is situated in the spinal cord has been overthrown. The prevailing opinion at present, based upon the work of Goltz, Freusberg, Ewald,[1] and particularly upon the recent work of L. R. Müller,[2] inclines to the view that the central mechanism for the vesical and rectal functions is situated in the sympathetic system. Since this question has not been absolutely decided, but requires further investigation (which may show that both conceptions possibly contain a portion of the truth), the only thing to be done is to give, first, the old, and then the newer, teaching, and to conclude with a few remarks as to the possibility of combining the two theories (see p. 947).

1. The Old Conception of the Vesical and Rectal Functions, in which the Central Mechanism was Supposed to be in the Spinal Cord.—*The Physiologic Mechanism of the Vesical Functions.*—The following diagram of the innervation of the bladder (Fig. 373) should be of assistance in comprehending the clinical features of the question:

Bl represents the bladder. The detrusor muscle is represented by a thick line, the mucous membrane by a dotted line. The physiologic sphincter, which is composed of the different voluntary muscles surrounding the urethra[3] (Krause's musculus urethralis) is represented by two circular cross-sections at both sides of the bladder orifice, *b'*.

The points *a*, *b*, *c*, correspond to the so-called bladder centers, as they have been thus far usually conceived.

[1] *Pflüger's Arch.*, vols. viii., ix., lxiii.
[2] *Zeits. f. Nervenheilk.*, vol. xxi., parts 1 and 2.
[3] The so-called "smooth sphincter" probably plays no important physiologic function.

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a corresponds to a sensory cell, or, better expressed, a cell in connection with the sensory terminal arborizations of the tract *a* to *a'*; *b* represents a motor cell for the sphincter; *c* represents a motor cell for the detrusor. *a* should be considered as connected with the mucous membrane by means of the sensory tract *a* to *a'*; *b* with the sphincter by the motor tract *b* to *b'*; and *c* with the detrusor by the motor tract *c* to *c'*.

The normal bladder reflex takes place in this way: When the bladder fills, the mucous membrane stimulates the tract *a'* to *a*. From *a*

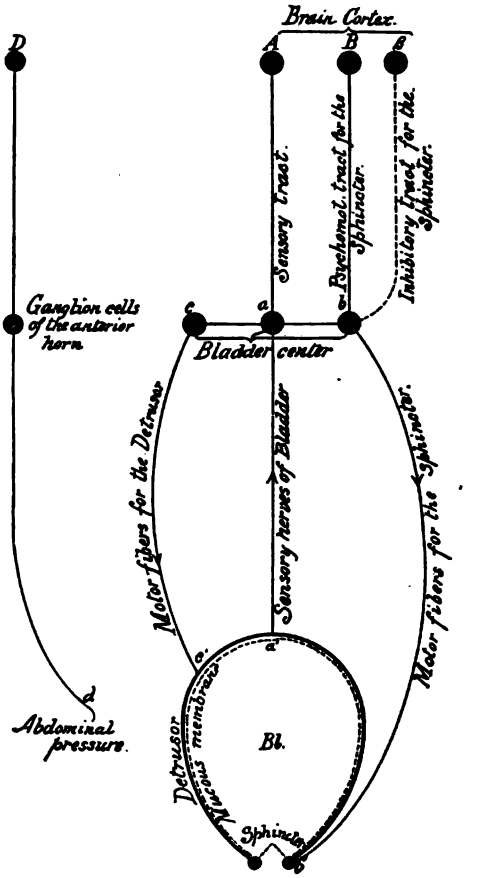

FIG. 373.—Scheme for the physiologic mechanism of the bladder functions.

the stimulation proceeds to the sphincter center, *b*, and from there, in the form of a tonic innervation, to the sphincter, *b'*, which then shuts the bladder more powerfully. When the filling increases still further, until a certain degree of distention is reached, a more intense stimulation *a'* to *a* proceeds from *a* even to *c*, and finally causes a contraction of the detrusor. The bladder will then be emptied. The normal blad-

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der reflex, therefore, consists of two acts: the closing reflex and the emptying reflex. We have included in our diagram the longitudinal tracts, which have an influence upon emptying the bladder; they have been demonstrated both physiologically and clinically, ascending and descending within the spinal cord. They are:

1. The tract *a* to *A*, a sensory tract leading to the brain, which informs us how full the bladder is.

2. The tract *B b b'*, subserving the voluntary innervation of the sphincter. As we cannot voluntarily innervate the detrusor, it probably does not possess an analogous tract.

3. The tract β to *b*, which inhibits the tonus of the sphincter. Since there is no voluntary detrusor tract, β *b* presides over the voluntary emptying of the bladder.

4. The tract *D* to *d*, inserted upon the left of the annexed diagram, comes from the brain and innervates the abdominal pressure, therefore assisting in forced voluntary emptying.

Voluntary emptying of the bladder takes place normally by means of these longitudinal tracts. For, after we have become cognizant of the bladder distention by means of the tract *a'* to *a*, we voluntarily relax the sphincter by an innervation along *B b b'*. This gives free scope to the reflex innervation of the detrusor by the path *a' a c c'*. Under some circumstances, when emptying the bladder is urgent, abdominal pressure is called in to assist by way of the tract *D d*.

It should, of course, be understood that the longitudinal tracts are bilateral[1] and that their radiations are in connection with both hemispheres of the brain. One hemisphere would, therefore, be sufficient to care for the voluntary function of the bladder (as in the case of the muscles, p. 829, Fig. 318).

Functions of the Bladder in Cerebral Affections.—Since only one hemisphere is involved ordinarily in diseases of the brain and, therefore, not all the fibers passing from the brain to the spinal cord are injured, and since the long tracts to the bladder are bilateral, lesions of the long tracts do not usually produce disturbances of the bladder in cerebral diseases, except from affections of the medulla and the pons. In the latter case, on account of the intimate proximity of the bilateral tracts, results are like those produced by lesions of the spinal cord (see below).

Bilateral cerebral lesions, on the contrary, may lead to disturbances in the bladder function, especially if they are diffused. This is because the bladder tracts are not arranged in the brain compactly in a bundle, but seem to spread out diffusely. These disturbances are ordinarily associated with disturbances of consciousness. An unconscious person allows his urine to escape because he has no will-power and no sensation. He is obliged to trust everything to the reflexes. The bladder reflex may be entirely unaffected, so that from time to time the bladder is emptied quite normally, even though unconsciously and involuntarily. Quite

[1] For the sake of simplicity our diagram shows only a unilateral arrangement of the tract.

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severe conditions of unconsciousness may lead to retention of the urine, with an eventual overflow of the overfilled bladder (paradoxic incontinence). These cases are most readily explained, as in spinal-cord affections (see below), by assuming loss of inhibition of the sphincter reflexes. The bladder may be similarly affected by brain injury in any serious illness, such as typhoid fever, etc.

Functions of the Bladder in Diseases of the Spinal Cord.—The following possibilities are conceivable:

1. The bladder center itself is injured. Both the sphincter and the detrusor reflexes are lost. The bladder behaves like a mere bag. The urine trickles continually, of course, only after the bladder has been more or less moderately distended, as a certain pressure of the urine is required to open the urethra. This is a condition of incontinence, with a more or less marked retention. Sounding by means of a catheter proves the existence of an actual bladder paralysis. The opposition of the sphincter is lost, and the contents of the bladder can be quite easily expressed by manual pressure upon the lower abdominal region.

2. The bladder center is intact, the lesion is located above the center, and only the long tracts which lead to the brain are injured. The influence of volition upon emptying the bladder will be more or less completely lost. If the injury to the long tracts is complete (and this is the usual effect of any pronounced focal lesion of the spinal cord, everything being so crowded together in a narrow cross-section), the patient will not be conscious of any distention of the bladder, nor possess any voluntary influence over it. One would naturally suppose that at least the reflex activity of the bladder would be normal; but this is only rarely true. To be sure, in exceptional cases of a cross-lesion of the spinal cord above the so-called bladder center, the patient will empty the full bladder, from time to time, in a perfectly normal fashion, but entirely involuntarily. Perhaps this type of disturbance occurs only in the less complete interruptions of conduction. But much oftener these patients suffer from urinary retention; the bladder becomes more and more distended; the sphincter finally gives way; and the urine trickles out. Such a condition of incontinence with an overfilled bladder is called *paradoxic incontinence*. The distinctive appearance of decided retention, which is always much more pronounced in these cases than in those under the first head, is to be explained by assuming that in severe cross-lesions of the spinal cord above the bladder center, the tract inhibiting the sphincter reflex (β *b*, Fig. 373, p. 940) is injured as well as the other long tracts. Analogous to the increase of tendon reflexes usually accompanying these cases, the tonus of the sphincter is naturally accentuated enough to overcome the power of the detrusor, and retention consequently ensues.

3. The lesion is situated below the bladder center. Since both sensory and motor fibers for the bladder run down from the bladder center within the spinal cord for a short distance before they pass out, such a focal lesion will partially interrupt the reflex arc of the bladder. The effect must, therefore, be similar to that of a lesion of the bladder

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center itself (see 1, p. 942). As we do not know just where the motor and sensory bladder nerves leave the spinal cord, it might be conceivable that, below this spot, an area in the spinal cord might exist injury of which would not affect the bladder function because the bladder nerves would already have left the spinal cord. Clinical experience, however, points to the improbability of such a supposition, and the diagram (Fig. 365, p. 925), representing a deep exit of the motor nerves of the bladder, should, therefore, be considered as accurate. The statements of physiologists in regard to position of the "bladder center" are so contradictory, and the physiologic methods of examining this question are so ambiguous, that we must provisionally depend upon clincal experience. The latter suggests that the human bladder innervation can be injured by a lesion even at the end of the spinal cord.

The Bladder Functions in Peripheral Affections of the Bladder Nerves.—The function of the bladder is rarely disturbed in these affections. In any given case the type of disturbance can be easily explained by consulting the diagrams.

Another Representation of the Bladder Functions.—The preceding diagram should be regarded as wholly theoretic. It depends upon the unproved assumption of a circumscribed bladder function in the spinal cord. (See p. 928 *et seq.* for arguments against such an assumption.) This assumption is based upon the fact that complete paralysis of the bladder (p. 942, No. 1) has been proved, both experimentally and clinically, to occur with disturbances in the territory of the lowest part of the spinal cord. This fact may, however, be perfectly explained in another way by assuming that the importance of the lumbar sacral cord for the bladder functions depends upon the compact arrangement of the sensory and motor fibers of the bladder at their exit there from the spinal cord; whereas, higher up, they perhaps spread out diffusely through a large portion of the longitudinal and cross-sections of the cord. Although, without doubt, the shortest reflex arc—*i. e.*, the shortest connection between the sensory and motor bladder nerves—is localized in the lowest portion of the spinal cord, this does not necessarily exclude the circuit of the reflex arc from reaching high up into the spinal cord (perhaps even to the brain).[1] The pathologic affections of the bladder functions can be explained as well, and perhaps more readily, by Fig. 374 than by Fig. 373, provided that we make such an assumption and consider the mechanism of the bladder functions analogous to the mechanism of the cutaneous reflexes (see p. 779 and Fig. 368, p. 928). In Fig. 337 we need to assume only that the reflex arcs for the detrusor reach high up in the spinal cord, or even as high as the brain; whereas, the sphincter reflex takes place principally by shorter tracts (in the lumbar enlargement). This conception readily explains the occurrence of retention of urine (paradoxic incontinence) in all complete cross-lesions situated above the lumbar enlargement, for the detrusor reflex would then be injured more than the sphincter reflex. This conception does not need to assume a definite center to explain the fact that severer bladder disturbances result from lesions of the lumbar enlargement; for, according to this assumption, a lesion here affects the entire compact centripetal and centrifugal bladder innervation. Again, it explains most readily the variations of the less typical bladder affections in which the bladder fibers for the different functions must be assumed to be variously affected; for, when we represent the reflex

[1] The theory that the reflex tracts of the bladder reach up into the brain agrees with the fact that the bladder functions are not necessarily destroyed by focal lesions of the brain, as the tracts in question must be situated bilaterally in the brain, while most cerebral lesions only affect one side. Moreover, the exceptional cases where cerebral lesions do affect the bladder functions are most easily explained by this assumption.

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apparatus of the bladder (as in Fig. 374) distributed through the entire spinal cord and even to a part of the brain, it becomes perfectly natural to conceive that the bladder mechanism can be injured in manifold ways—as is especially the case in system diseases like tabes.

So far as the voluntary control of the bladder is concerned, Fig. 374 should be regarded in the same way as Fig. 373, and should be completed by adding the voluntary tracts for the closure and relaxation of the sphincter, as well as those innervating the abdominal pressure.

We might add that the principle of the bladder innervation is not influenced by assuming in both Figs. 373 and 374 that parts of the reflex arc run in sympathetic tracts.

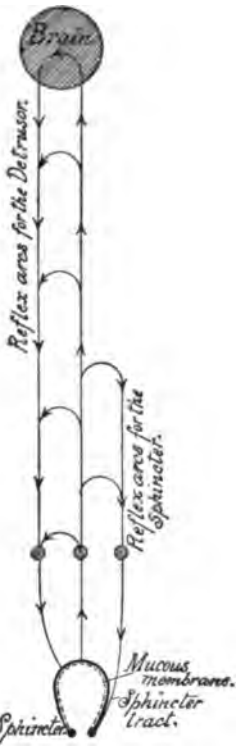

FIG. 374.—Scheme of bladder functions with superimposed reflex arcs.

Mechanism of Emptying the Rectum under Physiologic and Pathologic Conditions.—The emptying of the rectum is accomplished in the same way as the emptying of the bladder. With the proper changes, what has been said about the latter applies to the rectum. The old conception of a rectal center localized in the lumbar enlargement of the spinal cord (see Fig. 373) becomes less probable when we assume that the reflex arcs of the rectum (like those of the bladder in Fig. 374) run far up into the spinal cord, and perhaps even into the brain. Either theory will explain (as with the bladder) the fact that lesions of the lumbosacral cord produce an actual paralysis of the rectum; whereas lesions higher up cause for the most part a decided retention of feces. Clinically, however, the picture of fecal accumulation caused by paralysis of the detrusor reflex or by increase of the sphincter tonus can be altered if the peristalsis of the upper part of the gut, which is not under the direct control of the spinal cord, is sufficient to expel the fecal masses from the rectum in spite of the retention. On the other hand, the picture of sphincter paralysis in the rectum may be modified and much less noticeable if the fecal masses are hard enough to remain in the rectum, despite a weak or absent closure of the sphincter.

II. **The New Theory of Sympathetic Vesical, Rectal, and Ejaculatory Centers** (*Goltz, Freusberg, Ewald, L. R. Müller*).—The theory of the spinal localization of the vesical and rectal centers has long since been shaken by the experiments of Goltz, Freusberg, and Ewald.[1] These authors demonstrated that in dogs, after the removal of the lowermost portion of the spinal cord, which presumably contained the vesical and rectal centers, the primarily disturbed vesical and rectal functions became normal after a time. The theory of the spinal localization of the centers for the vesical, rectal, and ejaculatory functions had become so deeply rooted during the preceding fifty years, however, that it continued to dominate physiologic, as well as clinical, teaching. Since L. R. Müller's recent important experiments,[2] have completely confirmed those of Goltz's school and shown that the centers for the vesical and rectal functions and for the male sexual act are situated outside of the spinal cord in the sympathetic system, we must determine to what extent our clinical ideas should be revised.

The experimental results attained by Müller are essentially as follows: If the spinal cord of the dog be divided above the sacral segments, or if the sacral segments be extirpated, the result is practically the same. At first there is reten-

[1] *Pflüger's Archiv.*, vol. viii., ix., and lxiii.
[2] *Zeits. f. Nervenheilk.*, vol., xxi., parts 1 and 2.

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tion of the urine and of the feces. The bladder may be mechanically evacuated by pressure, and if this is not done regularly the bladder overflows (paradoxic incontinence). After a time, however, this urinary and fecal retention disappears and is replaced by periodic evacuations differing from the normal only in that they are involuntary. Since this re-establishment of periodic evacuations occurs even after complete extirpation of the lumbar and sacral cord, the central mechanism for these functions evidently must be situated outside of the spinal cord in the sympathetic system. Experiments in reference to the localization of the sexual functions in male dogs led to similar results. After the extirpation of the sacral, and of a great portion of the lumbar cord, the dogs are capable not only of erection, but also of ejaculation. The center for this function must, consequently, also lie outside of the spinal cord. In these experiments, however, the lesion of the spinal cord necessarily produces certain changes of the functions. Extirpation of the lowermost portion of the spinal cord, or simply transverse section, is followed by a complete loss of the influence of the will upon the evacuation of the bladder and rectum, and such evacuation becomes purely automatic, as may easily be recognized from the condition of the animal. The animals are, of course, anesthetic for the process of evacuation. Immediately after extirpation of the sacral cord the anus gapes from paralysis of the striated sphincter, but it gradually becomes closed, evidently as a result of the vicarious action of the involuntary musculature of the internal sphincter. The striated sphincter, however, remains paralyzed, and the anal reflex (p. 778) is permanently destroyed. If the spinal cord is simply divided above the sacral segment the tonus of the sphincter and the anal reflex are maintained; in fact, both may even be accentuated. The voluntary perineal muscles which assist in the voluntary closure of the bladder are possibly affected in the same manner as the external sphincter of the anus, although Müller makes no definite statements in this connection. They are paralyzed by destruction of the sacral cord, but the closure of the bladder is effected vicariously by the involuntary sphincter. The experimental results seem to indicate that reflex erection is associated with the maintenance of the lowermost portion of the sacral cord, but that psychic erection is dependent upon the maintenance of the lower dorsal cord. The lower sacral cord consequently seems to receive the sensory fibers from the penis, and to give rise to the genital reflex, while the centrifugal fibers from the brain to the sexual centers evidently leave the spinal cord in the lowermost dorsal segment. Semen may be discharged even after the destruction of the lowermost portion of the spinal cord, but a vigorous reflex ejaculation is dependent upon its integrity—*i. e.*, upon the innervation of the voluntary muscles (bulbocavernosus, etc.).

From these experimental data it is easy to assume: (1) That the actual centers for the evacuation of the bladder and rectum, as well as those for erection and ejaculation, are situated outside of the spinal cord in the sympathetic system; (2) that motor fibers reach this sympathetic apparatus through the spinal cord by means of the rami communicantes, from the lumbar and sacral segments; and (3) that the motor fibers for the striated muscles of the pelvic floor, which effect the voluntary closure of the bladder and rectum, arise directly from the spinal cord, and have nothing to do with the sympathetic. The sensory impulses to the brain are necessarily conducted by the spinal cord. According to this conception the only part played by the spinal cord in the vesical and rectal functions is the conduction of sensory impulses to the brain and of voluntary cerebral impulses for the innervation and relaxation of the striated vesical and rectal sphincters and for the innervation of the abdominal tension.

Supported by his own investigations, as well as by those of Rehfisch,[1] Müller completely repudiates the old theory of the spinal centers for the bladder, rectum, and ejaculation, and constructs the following diagram (Fig. 375) for the explanation of the clinical symptoms in patients with affections of the spinal cord, in which only the sympathetic plexus is to be regarded as a central mechanism.

According to this new theory the clinical symptoms in the human subject are

[1] *Virchow's Archiv*, vol. clxi.

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easily explained if we assume, with Müller, that in all destructive lesions of the spinal cord, whether they involve the sacral, the lumbar, or higher regions, the primary disturbance is always urinary and fecal retention, followed by paradoxic incontinence, that typical periodic evacuations are always established later, and that permanent disturbances in affections of the spinal cord, like the initial retention and paradoxic incontinence, are due simply to the absence of sensation as well as of the voluntary components of evacuation. Further investigations are necessary, however, to determine whether these suppositions are really in accord with clinical observations. The peculiar vesical disturbances in tabes dorsalis are certainly easy of explanation by Müller's theory, even without assuming, as does Müller, that in these cases the tabes is localized in the sympathetic. Also according to Müller's theory the sensory disturbances in tabes are sufficient to explain both the retention and the slight phenomena of incontinence in these

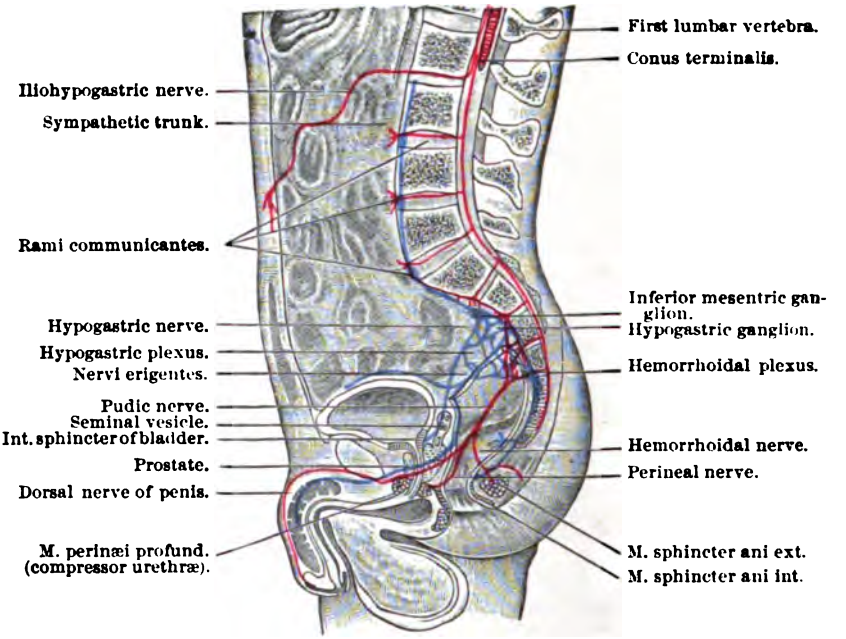

FIG. 375.—Diagram of the rectal and vesical innervation (according to Müller).

patients, since the regulating influence of the spinal cord and brain is more or less impaired as a result of the disturbed vesical sensibility. The sexual disturbances in patients with affections of the spinal cord may also be easily explained by Müller's diagram if we remember that the lesion hinders the transmission of the psychic excitations from the lower dorsal cord to the sympathetic sexual center, and that variations of the reflex irritability of the lowermost portions of the cord by spinal lesions may increase or diminish the reflex components of erection or ejaculation (see above). The fact that patients with destruction of the sacral cord and complete motor and sensory paralysis of the lower half of the body are still capable of procreation (examples of which are given by Müller) is best explained by the new theory.

It must be said that Müller's theory contains much that is plausible in reference to the non-striated musculature of the bladder and rectum, the spinal innervation of which, according to the old theory, always seemed rather dubious. His experiments also seem to be quite convincing, but the writer, nevertheless, believes that they are not entirely free from objection. For instance, these experiments, as well as those of Goltz and Ewald, have not certainly demonstrated that the

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vesical and rectal functions are carried on with the same vigor and precision after the destruction of the spinal cord as they were before, that the strength of the involuntary sphincters and detrusors does not also suffer, and that the degree of distention of the bladder and rectum prior to evacuation is the same without the influence of the spinal cord as it is with it. If we assume, according to the old theory (p. 943 *et seq.*), that the reflex mechanism of the bladder and of the rectum consists of a number of superimposed reflex arcs, it becomes quite plausible to conclude that the lowermost of these arcs is situated in the sympathetic, and that it is associated with spinal, and even cerebral, reflex arcs in such a manner that in case of necessity the sympathetic arc may carry on these functions, but that the finer and more vigorous functions are possible only with the co-operation of the spinal cord and brain. It is possibly a verbal quibble whether we regard the functions of the sympathetic, of the cord, and of the brain as occurring in reflex arcs, or whether we assume that the reflex function belongs only to the sympathetic, and that the higher functions of the cord and brain are excluded from this reflex. Since every nervous function proceeds upon the principle of a reflex, however, the former supposition seems to be fully justified. If we were called upon at present to decide between the old theory of spinal centers for the bladder, rectum, and erection, and Müller's theory of the localization of these functions in the sympathetic, we should be forced to give the new theory the preference, since it is founded upon the results of experiment. It would then be necessary to give a different explanation of clinical phenomena from that which has been offered in the past. By the conception of superimposed reflex arcs, however, it seems to the writer that the two theories may be made to harmonize. The recorded experiments are not sufficient to decide this question. The vesical and rectal functions remaining after destruction of the spinal cord must be more accurately tested, qualitatively and quantitatively, and this test should be clinical as well as experimental.

Examining the Vesical and Rectal Functions.

In examining the affected bladder and rectal functions the following points should be noted:

1. Fulness of the bladder and of the rectum—to be determined by palpation and percussion in the case of the bladder, and by digital examination in the case of the rectum.

2. Condition of the sphincter tonus—to be determined for the bladder by attempting to expel its contents mechanically or with the help of the catheter; for the rectum, by digital examination.

3. Sensibility of the bladder and rectal mucous membranes. The occurrence or lack of a desire for stool or urination will decide this point. The sensibility of the bladder can also be determined by a catheter; the rectum by a digital examination. Pain or pressure (tenesmus) accompanying evacuation should be heeded.

4. Characteristics of the evacuation: Absolute or partial retention of the urine and feces; overflow of the filled bladder and the rectum with pronounced retention and accentuated sphincter tonus (the real *paradoxic incontinence*); overflow of the filled bladder and of the filled rectum with moderate retention and absence of sphincter tonus (the real *bladder and rectal paralysis*); involuntary evacuation by means of the normal reflexes (*enuresis*); possibility or impossibilty of favoring the reflex involuntary evacuation (incontinence) or of inhibiting it. *Imperative incontinence* is the name applied to a special form of incontinence in which the only way to avoid an involuntary escape of urine or feces is immediately to accede to the very first demand for evacuation. The fact that constipation often occurs without any spinal cord affection makes it difficult to determine practically how much clinical value is to be assigned to the condition of the rectal evacuation. Therefore, before deciding that a constipation depends upon the spinal cord, it is especially important to examine points 1, 2, and 3. In any analysis of the character of evacuation we must also determine whether or not the patient still preserves the sensation of defecation, being careful to avoid confusing this sensation with that of being wet or soiled.

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APPENDIX.

The Utility of Routine Plans and Stamped or Printed Figures for Collecting and Tabulating the Results of Examination.

ROUTINE PLANS.

In tabulating the condition of a patient for certain special purposes it has been found very advantageous at the author's clinic to make use of definite routine plans. In this way nothing important in the examination can be forgotten, and the results will be entered in a careful and summarized fashion. These outlines have proved of so much value, and saved so much time, that it seems worth while to give examples of them.

They are of special value in examining the nervous system, both because the points upon which the complete examination depends are almost too numerous to keep constantly in mind, and because it is especially advantageous in this class of diseases to group the results of examination in an intelligent way (see p. 738). Of course they are also valuable in examining other affections. A few examples will illustrate this, although it scarcely need be mentioned that the outlines may be modified or enlarged at pleasure, according to the special purpose of the examiner. The following examples should in no way be considered as absolute standards, but merely as illustrations of what the author has found useful. Enough space upon the right side of the text should be reserved to enter the necessary notes. For the nervous system it is advisable to leave sufficient space for the data upon both sides, to correspond to the right and left sides of the body. Everything found to be "normal" should be marked so, and everything which has not been examined should be marked "not examined." Naturally, the entire status cannot be settled in every instance by filling out the outlines, because some conditions often require a more detailed description; but the outlines will serve to a certain extent as a nucleus for the entire clinical picture, about which everything else crystallizes. To prevent misunderstanding, the author wishes to call attention to the fact that such outlines do not in any way take the place of the ordinary extended descriptions of the symptoms of disease. Experience shows it to be entirely impractical to use them, except for certain special examination.

1. OUTLINE FOR THE EXAMINATION OF THE DIGESTION.

Name.
Date.
Appetite. Stool. Vomiting (time, character).
Retention in the morning. Pains (time, character).

CHARACTERISTICS OF THE GASTRIC CONTENTS: [1]

The Vomitus: Mucus, blood, leukocytes, meat fibers, starch, sarcinæ, bacteria.
The Expressed Contents of the Empty Stomach: Mucus, blood, leukocytes, meat fibers, starch, residue of other food, sarcinæ, bacteria.
The expressed contents of the empty stomach after the withdrawal of the gastric contents on the preceding evening.

BUTYROMETRIC GASTRIC EXAMINATION:

Amount of test-meal prepared . . c.c., containing . . gm. flour and . . gm. butter.
Amount of test-meal ingested . . c.c.

[1] What is not found should be crossed out.

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Expression after . . minutes. Amount expressed: . . c.c.

Acidity of the expressed meal: a . . c.c. $\frac{N}{10}$ NaOH to 10 c.c. contents = % HCl.

Free HCl { = + . . % HCl (acid excess).
= — . . % HCl (acid deficit).

Lactic acid: [Shaking with ether].

Residue estimation: 300 c.c. water added.

Acidity of the expressed dilution: b = . . c.c. $\frac{N}{10}$ NaOH = . . % HCl.

Residue $x = \frac{300 \cdot b}{a - b}$ = . . c.c.[3]

Total gastric contents (including residue estimation) at the time of the expression: To = . . c.c.

Fat percentage of the ingested flour soup (estimated butyrometically): F = . . %.

Fat percentage of the expressed flour soup (estimated butyrometically): f = . . %.

Residual amount of meal: $Su = \frac{f}{F} To$ = . . c.c. b ause $\frac{Su}{To} = \frac{f}{F}$).

Amount of gastric juice: $Ma = To - Su$ = . . c.c..

Acidity of the pure secretion: $A = \frac{a\ To}{Ma}$ = . . % (because $\frac{a}{A} = \frac{Ma}{To}$).

Secretion quotient: $\frac{Ma}{Su}$ =

Motility quotient: $\frac{\text{the soup which passes through the intestine}}{\text{soup ingested}}$ =

Summary of results: $\frac{To}{Su =}$ $\frac{}{Ma =}$ A =

EXAMINATION OF THE FILTRATE (of the test-breakfast,[1] of the vomitus).

Reaction to free HCl: . . . Phloroglucinvanillin . . . Methylviolet . . .
Rennin ferment . . . Tropäolin.
Starch digestion: Coloration of 5 c.c. of the gastric juice filtrate after addition of . . c.c. of normal iodid solution.
This color is blue,[1] violet,[1] red.[1]
Mett's test of digestion: Diameter of the capillary tubes . .

1. 1 part juice + 15 parts $\frac{N}{20}$ HCl : 2 × . . mm. (length of digested albumin).
2. 1 " + 31 " " " : 2 × . . mm. (" " "

IODOFORM GLUTOID TEST.

Control number of the capsules . .
Appearance of the first iodid reaction in the saliva after . . hours.
Duration of the reaction.
Results on a healthy individual.

2. OUTLINE FOR THE EXAMINATION OF CASES OF DIPHTHERIA AND OTHER ANGINAS.

Name.
Age.
Date.
day of illness.
Hoarseness.
Stenotic phenomena.
Membrane. No visible membrane. nasal cavity. buccal cavity.
tonsils. pillars of palate soft palate posterior pharyngeal wall.

[1] What is not found should be crossed out.

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Other Locations

Kind of membrane in the throat or vicinity { Punctiform. Patchy. Continuous. } Tenacious. Soft. Thick. Thin. Flimsy. Firmly adherent. Easily wiped off. Grayish. Yellowish.

Parts surrounding the membranes: Very red swollen not much changed.

Lymph glands: not little much swollen.

Angina lacunaris (punctiform membrane projecting from the depths of the crypts) catarrhalis.

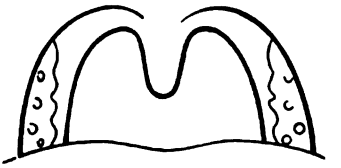

FIG. 376.—Diagram to represent the localization of the membrane.

Results of lung examination.

Albuminuria.

Bacteriologic findings.

Dry preparation (from where?).

Culture (from where?). Upon what medium?

Further notes.

3. OUTLINE FOR THE EXAMINATION OF THE BLOOD.

Name.

Date.

Hemoglobin.

Number of erythrocytes.

Quotient (hemoglobin value of the individual erythrocyte).

Number of leukocytes.

MORPHOLOGIC INVESTIGATION.

(*a*) *Fresh Specimen.*

Formation of rouleaux.

Fibrin.

Blood-platelets.

Poikilocytosis.

(*b*) *Stained and Fixed Specimen.*

Staining method.

Erythrocytes.

Shape.

Size.

Tinctorial peculiarities (diminished hemoglogin, polychromasia, basophilic granulation).

Nucleated reds. Normoblasts. Megaloblasts.

Differentiation of the leukocytes. (How many counted?).

Per cent. of the total number. Absolute number per c.mm.

Neutrophilic (polymorphonuclear).

Eosinophilic.

Large mononuclear without granulations.

(Transition forms).

Lymphocytes.

Mast cells.

Unusual forms.

Malarial parasites, bacteria.

Other observations.

(If there was an autopsy) condition of bone-marrow.

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4. OUTLINE FOR THE EXAMINATION OF THE NERVOUS SYSTEM.

(a) General Outline.

Date.
Name.
Sensorium.
Intelligence.
Disposition.
Memory.
Speech (for details, see special scheme).
Breathing.
Pulse.
Cranial nerves (for details, see special scheme).
Attitude.
Gait (with eyes open and shut).
Standing position (with eyes open and shut).

Voluntary Motility.

Neck:
- Sternocleidomastoid.
- Trapezius.
- Other muscles of the neck.

Upper Extremity (for details, see special scheme).[1]
- Power.
- Coördination.

Trunk:
- Intercostal muscles.
- Diaphragm.
- Abdominal muscles.
- Muscles of the back.

Irritative motor phenomena (clonic, tonic spasms, fibrillary twitchings, tremors, contractures, chorea, athetosis, etc.).

Muscular Atrophies:
- Electric reaction of the muscles (for details, see special scheme).

Sensibility.

(To be represented upon a chart of the body.[2] It should be mentioned here how the sensations of touch, pain, heat, cold, and pressure were examined).

Head and Neck:
- Sensation of touch.
- Sensation of pain.
- Sensation of heat.
- Sensation of cold.
- Localization of pain and touch.

Upper Extremity:
- Sensation of touch.
- Sensation of pain.
- Sensation of heat.
- Sensation of cold.
- Sensation of pressure.
- Localization of pain and touch.
- Innervation sense, "power sense" (how examined?).
- Appreciation and judgment of the active movements (how examined?).
- Appreciation of posture[3] (how examined?).
- Appreciation of an object by feeling it (how examined?).

[1] See (c) p. 954. [2] See p. 955. [3] See p. 767 *et seq.*

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Trunk:
- Sensation of touch.
- Sensation of pain.
- Sensation of heat.
- Sensation of cold.
- Sensation of pressure.
- Localization of pain and touch.

Lower Extremity:
- Sensation of touch.
- Sensation of pain.
- Sensation of heat.
- Sensation of cold.
- Sensation of pressure.
- Localization of pain and touch.
- Innervation sense, "power sense" (how examined?).
- Appreciation and judgment of the active movements (how examined?).
- Appreciation of posture[1] (how examined?).
- Appreciation of an object by feeling it (how examined?).

Spontaneous pains (neuralgias, parenchymatous pains).
Hyperalgesia of the skin.
Sensibility of the nerves and muscles to pressure.

Reflexes.

Cutaneous Reflexes (strength? alteration?).[2]
- Plantar reflex (to pricking).
- Plantar reflex (to tickling).
- Abdominal reflex (upper, lower, middle).
- Cremaster reflex (groin reflex).
- Anal reflex.
- Other reflexes.

Tendon Reflexes.
- Patellar reflex.
- Achilles reflex (foot clonus).
- Upper extremity.
- Periosteal reflexes.

Bladder Functions.

Fulness of the bladder.

Condition of the sphincter tonus (expression of the bladder, finally examining with a catheter).

Sensibility of the bladder mucous membrane. Desire for evacuation, urgency, tenesmus, pains.

Character of the evacuation: Absolute or partial retention; overflow of a filled bladder associated with a marked retention and an increased sphincter tonus (*paradoxic incontinence*); overflow of the bladder with a moderate filling and deficient sphincter tonus (*actual paralysis of the bladder*); the position of the fundus (palpation); evacuation through the normal reflex, but involuntarily (*enuresis*); possibility or impossibility of influencing the reflex evacuation, either favoring (incontinence, imperative incontinence, p. 947) or inhibiting it. Sensation produced by the evacuation to be distinguished from the sensation of the wetting.

Functions of the rectum: Character of the stool evacuation. For the rest we employ practically the same scheme as for the bladder functions.

Behavior of the nervous sexual apparatus. (Potency, etc.)

Trophic disturbances of the skin, hair, nails, decubitus, etc.

Vasomotor conditions.

Additional notes.

[1] See p. 767 *et seq.*

[2] See p. 786, Pathologic Reflexes.

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(*b*) **Outline for the Examination of the Cranial Nerve.**

Date.
Name.

I. *Olfactory.*
- Smell (how tested? cologne water, asafetida, ol. anisi).
- Result of rhinoscopic examination.

II. *Optic.*
- Central visual acuity (with corrected refraction).
- Visual field (hemiopia, limitations, fatigue).
- Result of ophthalmoscopic examination.
- Color blindness (how examined? Holmgren, Pflüger's flower contrast).

III., IV., VI. *Nerves of the Eye Muscles.*
- Direction of vision (conjugate deviation).
- Conjugate movements.
- Unilateral testing of the movements of the eyes.
- Double vision.
- Convergence (how tested?).
- Nystagmus:
  - Horizontal.
  - Vertical.
  - Rotatory.
- Pupils:
  - Size under moderate illumination.
  - Reaction to light (how tested?).
  - Direct.
  - Crossed.
  - Hemiopic rigidity to light (to be tested in hemiopia and double blindness, how tested?).
  - Narrowing in accommodation.
- Accommodation (how tested?).

V. *Trigeminus.*
- Sensory division.
  - Face.
  - Forehead.
  - Conjunctiva and cornea (corneal reflex)
  - Tongue.
  - Taste (how tested? salt, acetic acid).
  - Smell (how tested? acetic acid, ammonia).
- Motor division (palate muscles).
  - Raising the jaw.
  - Lateral movement of the jaw.
  - Atrophy of the chewing muscles.
  - Electrical examination (see special scheme).

VII. *Facial.*
- Upper branch.
- Lower branch.
- Palate (position, voluntary movement, speech, swallowing, reflex).
- Electrical examination (for details, see special scheme)
- Behavior of the facial associated movements.
- Behavior of the facial affected movements.
- Corneal reflex, optical facial reflex.
- Mechanical irritability (how tested?).
- Atrophy of the facial muscles.

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VIII. *Acoustic.*
Hearing in general.
Test of the air conduction.
Test of the bone conduction (v. Bezold's modification of Rinne's test).
Weber's test.
Schwabach's test.
Subjective noises, dizziness.
Results of otoscopic examination.

IX., X., XI. *Glossopharyngeal Vagus, Accessory.*
Taste (how tested? bitter, sweet?).
Act of swallowing.
Voice.
Breathing and pulse.
Results of laryngoscopic examination.
Trapezius, sternocleidomastoideus.
Atrophies.
Electrical examination (for details, see special scheme).

XII. *Hypoglossal.*
Extended movements.
Position of the tongue when protruded.
Atrophy of the tongue.
Fibrillary movements.
Electrical examination (for details, see special scheme).
Speech (see special scheme).
Other Remarks.

(*c*) Outlines for the Examination of Muscular Atrophies and Peripheral Motor Paralyses.

For the exact examination of the motility of the individual muscles and the peripheral motor nerves, the Bern Clinic uses the outline found upon pp. 910–914. Ample room should be left for the entries, and the outlines for the upper and the lower extremity should be printed upon separate sheets.

(*d*) Outlines for Electrical Examination.

Corresponding results of examining a healthy individual of equal stature and constitution, for comparison.

Date.
Name.
Name of the muscle examined.
Size in sq. c. of the stimulation electrode employed.

EXAMINATION OF THE MUSCLE.

Faradic Current.
Rapid interruption. Contraction measured upon the sliding apparatus.
Type of contraction. Single shocks.
Minimum contraction measured upon the sliding apparatus. Type of contraction. Signs of fatigue.

Galvanic Current.
Minimal CaCC in milliampères. Volt.
Type of contraction.
Minimal AnCC in milliampères. Volt.
Type of contraction.
Relation of CaCC to AnCC in milliampères. Volt.
Signs of fatigue.

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EXAMINATION OF THE NERVE.

Faradic Current.

Rapid interruption. Contraction measured upon the sliding apparatus. Type of contraction. Single shocks.

Minimum contraction measured upon the sliding apparatus. Type of contraction. Signs of fatigue.

Galvanic Current.

Minimal CaCC in milliampères. Volt.
Type of contraction.

Minimal AnCC in milliampères. Volt.
Type of contraction.

Relation of CaCC to AnCC in milliampères. Volt.

Signs of fatigue.

(*e*) Outline for the Examination of the Speech.

The summary upon p. 900 may be utilized as an outline.

ILLUSTRATIONS.

Charts are especially useful for recording the results of physical diagnosis. The fundamental rules recommended for their use are mentioned upon pp. 159 *et seq.*, 164 *et seq.*, 249, 260, 268–274, 279, and condensed upon pp. 321 and 348 *et seq.* They are employed in all the histories in Bern. Upon pp. 168–211 and 321–353 a large number of pathologic signs are to be found. The charts on pp. 159–161—better double the size—are good models. Exact representations are difficult to make upon the ordinary small chart. For routine office practice, however, rubber stamps are very serviceable.

Charts are quite as essential for representing disturbances of sensibility. The linear reproductions on pp. 915–920, Figs. 353–358, may be used as charts representing circumscribed disturbances of sensibility. Those upon pp. 772 and 773 are better for representing disturbances which affect a large part of the entire body (hemiplegias, paraplegias, hysteric disturbances of sensibility). The variations of sensibility can be best expressed upon these charts by different colors or shading.

Charts are also useful for representing laryngoscopic, rhinoscopic, and otoscopic lesions (Fig. 252, p. 695, and Fig. 267, p. 702).

The results of ophthalmoscopic examination may be very conveniently represented graphically in the sketch-book prepared by Prof. Haab (sold by Hofer Burger, in Zurich). Both plates in this book have been made by means of this sketch-book. The leaves are thickly colored to represent the red ground-tone of the opthalmoscopic picture. White or yellow shades can be produced by rubbing this ground-color more or less vigorously with an eraser or a knife, and the dark-red and black tones can be added with colored pencils or water-colors. The technic is explicitly described in the sketch-book, and any one can easily acquire facility enough to make very useful pictures.

SUPPLEMENT.

1. ANALYSIS OF THE IRREGULAR PULSE.

K. F. Wenkebach's[1] interesting research attempts to utilize Engelmann's[2] recent observations upon the properties of the heart muscle for the clinical analysis of the different kinds of irregular pulse. To understand what follows, we

[1] *Zeitschr. f. klin. Med.*, 1899, vol. xxxvi., 1899, vol. xxxvii., and 1900, vol. xxxix. Also, Wenkebach, *Die Arrhythmie als Ausdruck bestimmter Functionsstöringen des Herzens*, Leipzig, 1903.

[2] *Pflüger's Archiv*, vol. lxii. and lxv.

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must take for granted Engelmann's supposition that the physiologic rhythmic stimulation for cardiac activity proceeds from the opening of the great veins into the heart, the auricle and the ventricle. The normal heart action is the physiologic response of the heart muscle to these physiologic rhythmic stimulations. In addition we must accept as proved that (again according to the experiments of Engelmann) there must be differentiated as special properties of the heart-muscle fibers a power to produce stimulation, an irritability or stimulability, a power of conducting stimulation, and a capacity of contraction. These peculiarities, Engelmann maintains, are inherent in the muscle fibers themselves, but may be altered positively or negatively by many influences, which proceed partly from the nervous system. Engelmann differentiates *positive or negative chronotrophic* influences, by which the number of the physiologic automatic stimulations is increased or diminished in the unit of time; *positive or negative bathmotrophic influences*, which increase or diminish the irritability; *positive or negative dromotrophic* influences, which increase or diminish the conduction power of the heart-muscle fibers; and, finally, *positive and negative inotrophic* influences, which affect the contracting power of the heart-muscle fibers.

"PARARHYTHMIC" PULSE FROM EXTRASYSTOLE.

(The Ordinary Intermittent Pulse Caused by Extrasystole.)

Wenkebach, in the work alluded to above, analyzes first what we ordinarily designate clinically as simple isolated *pulse intermission*. According to auscultation and to sphygmographic tracings, this very frequent anomaly of the pulse is almost always to be attributed to the appearance of extrasystoles. These systoles are always excited by unphysiologic (according to Hering [1] myogenous) irritation, and mostly from the ventricular musculature. In the intermittent pulse they are not potent enough to produce a plainly palpable pulse in the radial, but can be discovered by auscultating the heart. Although sometimes indefinite, they are for the most part exhibited in some fashion or other in the sphygmographic tracings. Such extrasystoles are observed experimentally in digitalis poisoning and also in increased resistance to the cardiac contractions.

The peculiarities of the arhythmias dependent upon an extrasystole are to be explained from our physiologic knowledge as follows: As Bowditch and Marey have already shown, there occurs in the course of the cardiac cycle a so-called refractory phase (the shaded part in Fig. 377, *I*) in which the ventricle is non-irritable to the invading stimulus. This phase begins shortly before and persists for a short time after systole. The further diastole advances the more irritable the heart muscle becomes, and the slighter the stimulation needed to produce a so-called extrasystole—*i. e.*, an especial contraction independent of the physiologic rhythmic irritation. As a result of the course of the refractory phases the extrasystole is smaller the sooner it follows the normal heart contraction, or the refractory phase. On the other hand, however, an extrasystole produces a refractory condition of the ventricle, which usually persists until after the next physiologic stimulation has reached the ventricle. This next physiologic stimulation has therefore ordinarily no effect, but the following one functionates properly. Therefore a pulse generally fails after an extrasystole.

However, since the rhythm of the physiologic stimulation proceeding from the openings of the veins is unaffected by these conditions, the ventricle again contracts itself after the expiration of the extrasystole, exactly at the same instant at which it would have contracted if the pause in the following cardiac revolution had not occurred. Therefore, after an extrasystole (until the following normal systole), a pause ensues which exceeds in length the normal interval between the two pulses. It is called a compensatory resting pause, because, as a result of this longer interval, despite the extrasystole, the pulse-count in the unit of time remains constant. Engelmann called this phenomenon the law of the preserva-

[1] *Pflüger's Archiv*, vol. lxxxii., and *Prager med. Wochenschr.*, vol. xxvi., parts 1 and 2, 1901.

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tion of the physiologic stimulation period. Intermissions which depend upon such extrasystoles present the following peculiarities: Let curve *I* (Fig. 377) represent the radial pulse curve corresponding to a normal contraction of the heart. Then Curves *II–V* represent radial curves affected by the introduction of an extrasystole, and the compensatory failure of the following normal systole. As a result of the law mentioned above (the retention of the physiologic stimulation period) the normal systole which follows the extrasystole occurs at exactly the same time as it would have otherwise, as can be seen in the figure by comparing the apices. Another result of this law is that the pause between the beginning of the systole which precedes the extrasystole and the beginning of the one which follows the extrasystole (included in the figure by a bracket) is exactly twice as long as the normal interval. Pulse intermissions caused by extrasystoles can be recognized by this peculiarity in the sphygmogram even if the extra-

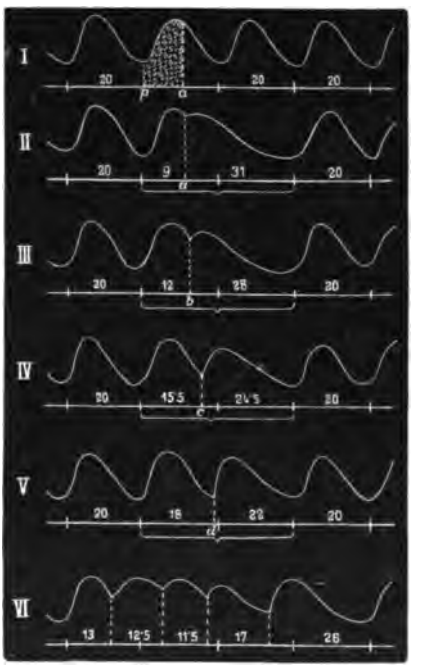

FIG. 377.—Diagram of extrasystoles and compensatory pauses by stimulation of the ventricle at different times during diastole (after Engelmann).

systole itself is hidden beneath the secondary apex of the pulse curve. To summarize, we may say that the ordinary intermittent pulse is characterized by the occurrence of an extrasystole which cannot be palpated in the radial, and that the intermission measures exactly twice as much as the preceding normal cardiac revolution.

The sphygmogram may present entirely different characters, depending upon the point of time at which the extrasystole appears; the later in the diastole it appears, the plainer the extrasystole becomes, both in the pulse curve and in auscultation. It may then present the appearance of a normal but slightly anticipated pulse (Fig. 377, *V*). Quite vigorous extrasystoles produce two tones audible over the heart; weaker ones, merely one first tone. Figure 378 represents all transitions between an apparently pure intermission with an extrasystole, which cannot be plainly recognized by the sphygmogram, through the doubled apex curves, which are sometimes called a pulsus bigeminus, up to a

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pulse-beat which apparently merely occurs a little too early. Wenkebach assumes that the so-called "futile" heart contractions—*i. e.*, those not palpable at the radial—must necessarily be extrasystoles. The "futility" of an extrasystole depends essentially upon the phase at which it appears, and many of the palpable and auscultatory peculiarities which Quincke and Hochhaus have assumed in defining "futile" contractions (p. 297 *et seq.*) must depend principally upon the occurrence of the extrasystole at a definite phase of the cardiac cycle. These points are not, therefore, sufficient to differentiate the "futile" contractions as a certain specific sort of contraction from other rudimentary systoles—*i. e.*, extrasystoles.

The first systole after the compensatory pause (post-compensatory systole) is ordinarily more vigorous and produces more effect upon the pulse curve than a

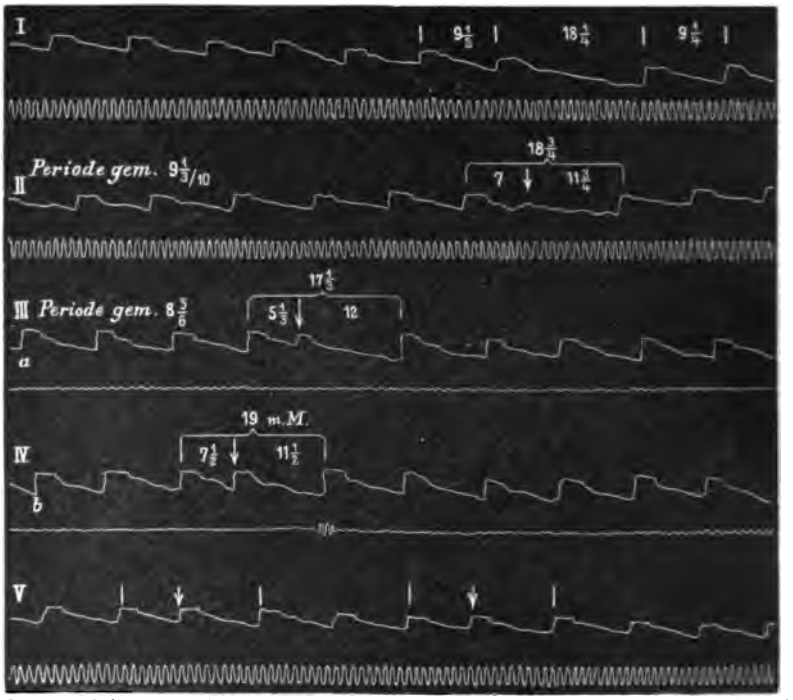

FIG. 378.—Sphygmograph with extrasystoles: In the curves I–V, which are all taken from the same patient, the extrasystole occurs later and later. In I it does not appear, but is recognizable only by auscultation and by the occurrence of a pause twice as long as normal. In V the extrasystoles in size and time of occurrence might be confused with the normal systoles, especially as they alternate regularly with the latter. In V *s* represents the normal systole; *l*, the extrasystole (after Wenkebach).

systole which follows after the normal interval. This is not represented in Fig. 377, but it can be detected in Fig. 378, *I* to *V*. It depends upon the longer rest the musculature obtains and upon the more complete diastolic filling of the ventricle.

Extrasystoles do not, however, always lead to an intermission of the pulse. The intermission, and with it the compensatory pause (Fig. 379), may be lacking. This may be the case when the pulse-frequency is diminished, and as a result the "refractory" phase is short in proportion to the length of the pulse. The pulse following extrasystole then occurs at the normal moment of time, so that the extrasystole seems to be simply inserted, and the law of the retention of the physiologic stimulation is preserved.

Characteristic pulse curves will occur if the normal cardiac action is altered

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by several extrasystoles following one after the other. Several pulses appearing at abnormal intervals then seem to be inserted into a normal pulse curve. That even this irregularity depends upon extrasystoles may be seen from the fact that, on account of the law of the retention of the physiologic stimulation period, the part of the pulse curve which contains the irregular pulse—*i. e.*, the extrasystoles—can be divided up into an integral number of normal pulse periods. (See the diagram, Fig. 377, *VI.*)

When the intermissions in a pulse curve occur in different kinds of groups, and thereby the extrasystoles produce sometimes more, and sometimes less, distinct pulses, and when the post-compensatory pulse is abnormally long, and the pulse following this abnormally small, then we may lose entirely the regularity of the rhythm in the ventricle and in the arterial pulse on account of the extrasystole, even despite the regular physiologic stimulation interval at the venous orifices. Such an irregular, unequal, and intermittent pulse (which depends upon extrasystoles) is, nevertheless, quite different from the so-called true irregular pulses which depend upon a disturbance of the rhythm originating in the stimulation in the venous orifices. Wenkebach emphasizes this by saying that it makes a great difference whether a person makes false steps here and there and limps because he has a lame leg or because he is walking upon a path with holes or stones. In difficult cases only an exact measure of time (which the Jacquet sphygmochronograph (p. 101) can furnish) will enable us to decide whether the arhythmia depends upon extrasystoles or upon an irregularity of the physiologic stimulation periods themselves. Wenkebach insists that the latter should be called *true arhyth-*

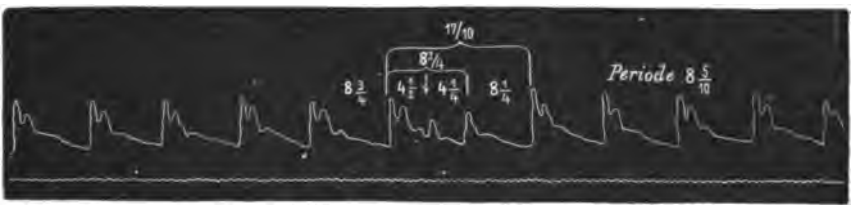

FIG. 379.—Extrasystole without compensatory pause (after Wenkebach).

mias; the former, *pararhythmias.* According to this we should speak of arhythmias from extrasystoles as presenting a pararhythmic, irregular, unequal, and intermittent pulse.

Wenkebach sometimes observed a characteristic deviation from the ordinary time relations in the pararhythmic intermittent pulse, in that the compensatory rest was too short—*i. e.*, the intermission was shorter than double the normal period. This type is observed in valvular lesions, and shows that such extra systoles do not proceed from the ventricles, but either from the venous orifices or from the auricles. In explanation Wenkebach reminds us of the following experimentally confirmed facts. In an extrasystole arising from stimulation of the venous openings the compensatory slowing of the pause after the extrasystole entirely fails, because when the extrasystole is produced, the stimulability is exhausted so that the normal amount of time is needed before another stimulation can be produced. In this case, if the extrasystole causes an intermission of the peripheral pulse, the length of such an intermission is less than twice the normal period.

In the extrasystole proceeding from the auricle the pulse following the extrasystole may resemble that of a ventricular extrasystole—*i. e.*, conforming to the law of maintenance of the physiologic period of irritation, it may occur exactly at the time at which the second normal systole would have commenced had the extrasystole been absent. In case the extrasystole leads to an intermission of the pulse, this intermission is also twice as long as the normal period of rest. In other cases, however, even when the extrasystole proceeds from the auricle,

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the time between two successive normal systoles or the duration of the intermission may be shorter than twice the normal period. According to Wenkebach, this is the case when the extra-irritation originating in the auricle also produces a recurrent contraction extending to the site of the origin of the physiologic irritation at the venous orifices. In this case the intermission is shortened, because, at the moment when this recurrent contraction reaches the venous orifices, all of the irritation here present is nullified, so that the return of the physiologic series of irritation is hastened. Hering,[1] who has studied this question especially by the help of animal experiments, reaches the diagnostic result that in case the extrasystole is associated with normal compensatory pauses, the starting-point of the stimulation causing the extrasystoles is either the ventricle or the auricle, but never one of the venous openings into the auricle; and that the shortening of the compensatory pause proves that the stimulation does not precede from the ventricle, but either from the auricle or from the great veins. According to Hering, the extrasystoles, as has been already mentioned, are always to be attributed to the direct stimulation of the cardiac musculature, and the resulting irregularity is to be designated as *myerethic irregularity*.

ALLORHYTHMIAS.

The regularly intermitting pulse is so called by Wenkebach. A regularly intermittent pulse—*i. e.*, one occurring after a certain number of cardiac beats—may in exceptional cases depend upon the fact that the extrasystoles leading to the intermission occur at regular intervals. Such a regularity, naturally, generally persists but a short time. As contrasted with this, Muskens had already noted that the most probable explanation for the occurrence of regularly repeated intermissions is furnished by assuming a definite amount of diminution of the conduction power of the heart muscle. Wenkebach has now demonstrated, by a careful analysis of pulse curves, that this is the ordinary etiology of the regularly intermitting pulses. To understand what follows, a few words are necessary. Engelmann represented the amount of the conduction power within a certain section of the heart as $\triangle$. The conduction power in the venous sinus is represented as $\triangle s$, that in the auricle as $\triangle a$, and that in the ventricle as $\triangle v$. $\triangle a/v$ would then represent the power of conduction between the auricle and ventricle, etc. The time in which the contraction stimulation travels from one place to another depends upon the conduction power. The interval $As-Vs$ (between the systole of the auricle and that of the ventricle) is of especial interest. During systole, not only the stimulability, but also the power of conduction $\triangle$, is gradually recuperated. With this explanation it is comprehensible that if the power of conduction—*e. g.*, at the boundary between the auricle and ventricle—is injured it may, after several heart-beats, become so much depressed that finally the stimulation no longer reaches the ventricular musculature in sufficient force to produce a contraction; but that after such a rest, in a certain way forced upon the ventricle, new beats will reappear. Given a certain degree of conduction weakness, this process may be repeated in such a way that one ventricular contraction will regularly be lost after a certain number of heart-beats, and so groups of regular pulses will be interrupted by an intermission. Such groups have for a long time been recognized in physiologic experiments, and called Lucian's periods. If we examined the sphygmogram in patients with such a regularly intermitting pulse more exactly, we should find that the intervals between the pulse beats within a group vary quite decidedly from each other; and that in each group this variation is repeated in a perfectly typical fashion. From a more accurate analysis of these variations in the intervals of the pulse within a group, Wenkebach now shows that the regular loss of the pulse (which we are discussing) depends upon a negative dromotropic influence (see p. 956)—*i. e.*, upon a disturbance of the conduction power. After the establishment of the pulse the first interval within a group

[1] *Pflüger's Archiv*, vol. lxxxii., and *Prager med. Wochenschr.*, 1901, vol. xxvi, parts 1 and 2, 1901.

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is the largest, and the following intervals are decidedly smaller; but for the most part they again increase somewhat in size, up to a new intermission. This is explained by assuming from what follows (and, moreover, it has been experimentally proved by Engelmann) that the conduction power △ is injured most by the first contraction after pause and is less markedly affected by the following contractions. The annexed diagram (Fig. 380), taken from Wenkebach, illustrates the relations very clearly.

The diagram is based upon the above assumption: that △ is diminished by cardiac activity itself (by fatigue). The horizontal lines in the figure are to be considered as time abscissæ. The rhythmic physiologic stimulations which arise at the venous orifice are marked at regular intervals upon the time abscissa ρ, 20 units apart. The straight lines drawn obliquely downward to the right are the graphic representations of the course of the stimulation from the venous orifices to the auricle and ventricle. The part of the figure included in the brace *A* represents the area of the auricle, that included within the brace *V* the area of the ventricle. The points of intersection of the oblique and horizontal lines represent the moments of time at which the physiologic stimulation, passing from the root of the heart to its apex, first, crosses the auricular border; second, stimulates the ventricular contraction. Wenkebach's own words furnish the best explanation of the rest of the diagram: "The rate of the stimulation contraction at the

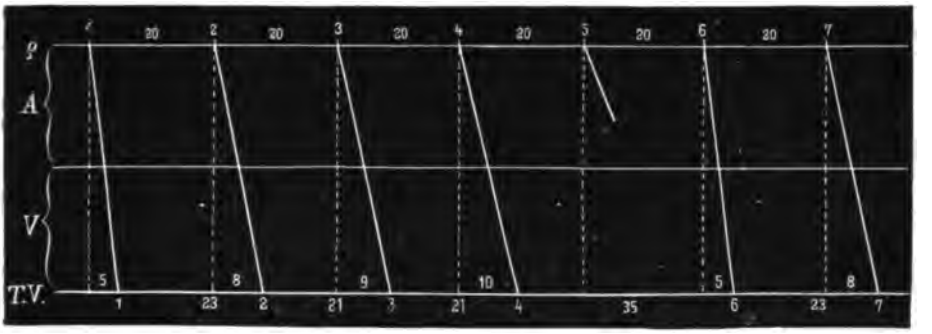

FIG. 380.—Diagram to represent a regularly intermittent pulse, caused by disturbance of the conductivity of the heart muscle: One pulse is lost after every four (after Wenkebach).

heart root $T\rho = 20$; and the interval ρ to *Vs*, that is the time between the stimulation ρ and the ventricular systole *Vs*, in a favorable case (after a pause = 5); but at the extreme case, where the stimulation is still transmitted just vigorously enough to produce a ventricular contraction = 10. During the contractions the interval ρ to *Vs* is prolonged from 5 to 10. Following the experiment, we may assume that after the first systole of the group, ρ to *Vs* is increased 3; after the following systoles, ρ to *Vs* is increased 1 each time.

"Should △ in each contraction remain equal, as is normally the case, the tempo of the ventricular systole *TVs* would always equal ρ, even if △ is very slight. A diminution of △ would occasion the proportionate increase of *TVs*. But since each systole has another influence upon △, the case will be quite different; and in the instance given here, the first interval (represented in the figure) of ventricular systole, $T_1 Vs = 23$, being increased by 3, on account of the increase of the interval ρ to *Vs* from 5 to 8. The following intervals of the ventricular systole, $T_2 Vs$ and $T_3 Vs = 21$, on account of a further increase by 1. Then, however, the limit is reached, and the fifth stimulation is either not transmitted at all or is no longer sufficiently transmitted. The ventricular contraction fails, and an intermission results. The interval ρ to *Vs* in this pause returns to 5, and the duration of the intermission (*T* int.) amounts to 5 less than the double of $T\rho$; that is *T* int. $= 2T\rho - 5 = 35$. The same process is repeated after the intermission. The tempo of the chamber contraction then depends entirely upon the rapidity of dim-

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inution of $\triangle$ in regular rhythm of the contraction stimulation, and with it the increase of ρ to Vs."[1]

Wenkebach found that the time relations in the accompanying sphygmogram (Fig. 381) were in complete conformity with this diagram (Fig. 380), and with the explanation given for the intermission. We observe in Fig. 381 that the numbers representing the intervals confirm the view that the periods after the intervals are the longest, the following much shorter, and those still later somewhat longer again. Although it cannot be recognized in the figure, a gradual diminution in the power of conduction occurs toward the end of a group in the pulse curve, observed as a diminished celerity in the ascending of the pulse wave.

FIG. 381.—Regularly intermittent pulse with groups of four pulses (after Wenkebach).

Wenkebach considers that the exact measuring of the pulse curve shows the size of the interval of the automatic stimulation at the venous orifices $T\rho$, as well as the amount of the diminution of conduction power in the course of a group. The original article should be consulted in this connection.

Gaskel and Engelmann have called attention to the practical point that vagus irritation diminishes the size of $\triangle$. In this way they explain the allorhythmia frequently occurring after vagus stimulation, as well as the appearance of regular intermissions (especially the bigeminal and trigeminal pulse) after employing digitalis, as this drug stimulates the vagus. At the same time if there is a regularly intermittent pulse, digitalis increases the disturbance of rhythm, because it diminishes $\triangle$ by vagus stimulation. This also explains how atropin improves certain regular intermissions, as this poison depresses the vagus tonus, and thereby improves the conduction power of the heart muscle.

Still other kinds of allorhythmias may arise from more pronounced diminution of $\triangle$. Corresponding to the conception of allorhythmia, these are all characterized by a certain amount of regularity, or even by perfectly regular repetition of the intermissions—*i. e.*, some grouping of the pulses. However, if a longer pulse curve is taken, the regularity of the grouping need not be absolutely even, because, during the observation, varied influences may affect the power of conduction. Any degree of gradation may occur between the ordinary regular

FIG. 382.—So-called "regularly double intermitting pulse" (pulse-groups separated by two longer cardiac revolutions) (after Wenkebach).

pulse-rhythm and the regular pulse with half-frequency (bradycardia) depending upon the amount of injury to the conduction power—*i. e.*, the negative dromotropic influence.

Such transitional forms are composed of groups of 6 to 2 pulse strokes. The regular intermitting pulse in groups of 2 strokes is nothing but a transformed pulsus bigeminus alternans. The real pulsus alternans (p. 963) does not belong here; but, according to Wenkebach, to a disturbance, not of the rhythm, but of contractility, In another group of cases the diminished power of conduction is expressed by the appearance, after the intermission, of a single systole, then

[1] *Zeits. f. klin. Med.*, vol. xxxvii., p. 482.

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another intermission, and then a group of several pulse beats. This Wenkebach[1] has called the "regularly double intermitting pulse," a term, however, which might lead to misunderstanding. Figs. 382 and 383 illustrate and explain the type. Still another serious disturbance of △ will produce regular group formations, in which the groups are separated by 3, 4, 5, or n cardiac revolutions prolonged through the loss of a systole. Finally, if $n = \infty$, regular bradycardia with half-frequency occurs, as then only the second stimulation is permanently active.

In all the cases thus far described the diminution in conduction power goes only so far as to occasion the loss of a systole more or less often, but we may conceive of a case in which two or even several systoles, one immediately after the other, are lost; then longer pauses—*i. e.*, still more pronounced bradycardia—would result. Such an excessive bradycardia may be responsible for the phenomenon observed in Stokes-Adams disease. His's observations (p. 297) upon the heart blocking noticed in this disease argue that a diminished conduction stimulation is the salient feature.

Any allorhythmia characterized by no particular regularity of the grouping may be attributed to a defect of the conduction power, provided that auscultation confirms the lack of extrasystoles. Further assistance will be furnished by

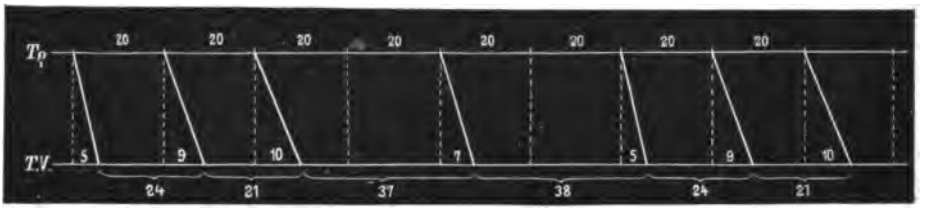

FIG. 383.—Diagram to explain Fig. 382 (after Wenkebach).

noting that the intermissions are always shorter than twice the normal periods of cardiac action (lack of compensatory rest which occurs with extrasystoles) and that within each group the first revolution after the intermission is the longest, the following is smaller, and the still later ones increase gradually. This can easily be understood by comparing the scheme, and it is very important for the diagnosis of difficult cases.

TRUE PRIMARY ARHYTHMIAS.

We have thus far described the disturbances in pulse rhythm called pararhythmias and allorhythmias, which depend, upon the one hand, upon extra systoles, and, on the other hand, upon diminution in the power of conduction. Now we may turn to what Wenkebach calls the true arhythmias, those disturbances of the pulse rhythm which depend upon a disturbance of the primary stimulation rhythm or of the primary stimulation strength at the venous orifices. They can be diagnosed only by excluding the pararhythmias and allorhythmias.

DISTURBANCES OF THE RHYTHM FROM DISTURBANCES OF CONTRACTILITY.

The Pulsus Alternans.

The ventricular contraction may fail, if the contractility of the heart muscle has been affected, even though the stimulation rhythm remains normal and the

[1] Wenkebach is justified in calling attention to the fact that the terms pulsus bigeminus and trigeminus had better be discarded, since he shows in his particularly instructive book (p. 171) that sphygmograms requiring these designations are brought about in a number of different ways—by extrasystoles, by disturbed conductive power, and by true arhythmias—and that, consequently, they should not be classed under one common name.

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conduction power good. The resulting arhythmia will be appreciable, both to palpation of the pulse, and in the sphygmogram. In this case it depends merely upon negative inotropic influences. A grouping of more or less distinct regularity may occur from the summation of fatigue phenomena. These arhythmias, which the author would describe as "intropic arhythmias," can generally be diagnosed from the differences in the size of the pulses, although without any marked crowding of the periods. The pulses which fail, and to which, as in pararhythmia, no extrasystoles correspond, are, of course, excepted.

The pulsus alternans must be discussed here among the intropic arhythmias, because it arises from a disturbance of contractility. A more careful examination and measurement of its pulse curve shows that it presents a very characteristic deviation of rhythm from the normal. *Pulsus alternans* is a pulse series with large and small pulse waves regularly alternating with each other at almost equal intervals (see Fig. 43, p. 125). Wenkebach[1] has quite recently analyzed this pulse most accurately, and decided that it depends upon a disturbance of the contractility for the following reasons: It certainly cannot depend upon a variation in the intensity of the contraction stimulation, because, as is well known, the heart always reacts maximally, even to a diminished stimulation, provided that the latter is sufficient to cause any contraction at all. For the same reason it cannot be due to a diminished power to conduct stimulation. It is incorrect to suppose that it could depend upon a bigeminal pulse with an extrasystole occurring practically halfway between two normal systoles; because, in the first place, it is quite exceptional for extrasystoles to persist so long a time, and because, besides, extrasystoles interposed almost in the place of the normal systoles would produce practically a normal pulse. Wenkebach's explanation is better. He considers that it depends upon a disturbance in the contractile power. After a vigorous contraction the contractile power is not sufficiently recuperated to respond with an equally vigorous systole to the next stimulation. Wenkebach appreciates a difficulty in this explanation, for every contraction is in one sense maximal, and whatever contractile power is at its command is utilized, and therefore the heart must be quite as fatigued after a weaker contraction as after a stronger one. At the same time we have no right to assume, without anything further, that the heart is recuperated more quickly after a weak contraction than after a strong one. Wenkebach believes that this difficulty can be accounted for by assuming that the stronger and weaker contractions affect the contractility differently by not following equally rapidly. B. F. Hoffmann,[1] in fact, did demonstrate that the contractions of the heart muscles occur more rapidly in a hypodynamic condition—*i. e.*, with less contractile power—than with normal contractility. Now, assuming that the smaller beats of the pulsus alternans occur more rapidly than the larger, the pause from the end of the smaller to the beginning of the greater pulse will be slightly lengthened; whereas, the pause from the end of the greater to the beginning of the smaller will be shortened. This can be easily demonstrated in curves of the pulsus alternans. As a result the contractile power recovers better after the smaller than after the greater, and by such an action the regular variation between the greater and smaller pulses will be retained. Even the beginning of the small pulse appears anticipated in the radial curve, as contrasted with the beginning of the great pulse, and Wenkebach explains this peculiarity by assuming that the small pulse waves are more rapidly transmitted to the periphery than the large ones.

The alternans is then a result of diminished contractile power of the heart muscles, and is again and again automatically reproduced. Wenkebach explains the primary cause of the entire phenomenon as follows: With a hypodynamic condition of the heart—*i. e.*, diminished contractile power—if a single physiologic irritation happens to occur somewhat too early it finds the heart muscle in a more hypodynamic condition than the previous stimulation did. The resulting

[1] *Zeits. f. klin. Med.*, 1902, vol. xliv., parts 3, 4, p. 218.
[2] *Pflüger's Archiv*, 1901, vol. lxxxiv

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contraction will, therefore, not only be smaller, but also, according to Hoffmann, will be performed more rapidly. Therefore, if the irritation of the next stimulus does not occur too late, the longer pause will permit the contractile power to recover perfectly, and so the following contraction will be more vigorous. This, in turn, lasting longer, the succeeding pause will, as a result, be shortened, and, consequently, the succeeding contraction smaller. This play can be repeated indefinitely, as long as no new factors enter the field. Hence one single irregularity of the rhythm is sufficient to produce a pulsus alternans; and, provided that the following stimulations are repeated at regular intervals, it may persist for a long time, until a medium-sized contraction occurs and the pulsus alternans is equalized.

2. ACIDIMETRIC TITRATION OF FLUIDS WHICH CONTAIN ALKALINE EARTHS AND AMMONIA SALTS IN ADDITION TO SALTS OF PHOSPHORIC ACID.

(**Gastric Juice, Urine,** pp. 378 and 541.)

Moritz[1] has recently suggested a method for the acidimetric titration of fluids, such as the gastric juice and the urine, containing alkaline earths and ammonium salts as well as phosphates, which is entirely free from objection, and which eliminates those inaccuracies of titration the existence of which was demonstrated by Nägeli in his investigations upon the acidimetric titration of the urine (p. 541).

Moritz states that the difficulties associated with the acidimetric titration of urine and gastric juice with phenolphthalein are as follows: 1. The monalkaline and dialkaline phosphates in phosphatic fluids react with phenolphthalein as they do with litmus, counterbalancing each other during a certain period of the titration, and consequently render the end reaction indefinite. 2. The contained ammonium salts interfere with titration to a certain extent, since ammonia acts so feebly upon the phenolphthalein that more alkali must always be added than corresponds to the actual neutralization. 3. As a result of the weak alkaline reaction of the monalkaline carbonates ($NaHCO_3$ and $KaHCO_3$), the carbonic acid titration is not exact. 4. In the phenolphthalein titration with alkali, the fluid seems to be too acid from the precipitation of indefinite quantities of basic calcium salts. Magnesium salts do not produce similar disturbances.

Moritz has shown that the difficulties dependent upon the presence of ammonium salts, phosphates, and carbonates may be completely overcome and exact end reactions obtained by diluting the fluid to be titrated with an equal volume of concentrated sodium chlorid solution which has been prepared with boiled water freed of its carbonic acid. This effect is doubtless due to an inhibition of dissociation. After this addition, a solution of an alkaline bicarbonate reacts neutrally with phenolphthalein. After the addition of the saline solution, according to Moritz, the carbonic acid is titrated to the limit between the primary and secondary salts, and the phosphoric acid to the limit between the secondary and tertiary salts. Moritz also eliminates the difficulty dependent upon the presence of calcium salts by precipitating them by the addition of sodium oxalate.

[1] *Arch. f. klin. Med.*, vol. lxxx., pp. 5 and 6.

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According to Moritz's directions, the entire procedure (both for the gastric juice and for the urine) is carried out as follows:

The titration is best performed in small Erlenmeyer flasks holding about 150 c.c. About 10 c.c. of fluid are employed, to which are added about 4 c.c. of an approximately semi-normal sodium oxalate solution (neutralized $\frac{N}{10}$ sodium oxalate solution, see p. 378) and 15 c.c. of a concentrated sodium chlorid solution. After the addition of the oxalate, the experimenter should wait a short time until the calcium becomes precipitated. This occurs almost instantaneously, and when it is completed the saline solution is added. Two such mixtures are made in two similar flasks, and one is employed for color comparison. By this method the change of color is apparent even with dark gastric juices and urines. A special procedure is necessary for urines containing a phosphatic precipitate. If the urine contain no carbonate, the phosphatic precipitate may be dissolved with $\frac{N}{10}$ acid; the amount of added acid must be subsequently deducted from the result obtained by titration. Should carbonates be present this procedure cannot be followed, since the acid would liberate CO_2, and the urinary acidity would consequently be decreased. In this case the precipitate from a definite quantity of well-stirred urine is collected upon a filter, well washed, and titrated (with methyl orange as an indicator) in the smallest volume of $\frac{N}{10}$ acid that will hold it in solution; the sodium oxalate and sodium chloride are then added as usual, and the solution is titrated back with $\frac{N}{10}$ KOH and phenolphthalein. The alkali equivalent found in the precipitate must then be deducted from the acidity found in the filtered urine.

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